

CALIFORNIA LEGISLATURE

JOINT INFORMATIONAL HEARING

SENATE FOOD AND AGRICULTURE COMMITTEE

SENATOR DEAN FLOREZ, CHAIR

AND

SENATE HEALTH COMMITTEE

SENATOR ELAINE KONTOMINAS ALQUIST, CHAIR

**FOOD AND BEVERAGES:
STRATEGIES TO RECOUP THE HEALTH COSTS OF
EXCESSIVE SUGAR CONSUMPTION**



April 20, 2010

State Capitol • Sacramento, California

COMMITTEE MEMBERS:

Senator Dean Florez, Chair

Senator Abel Maldonado, Vice Chair

Senator Loni Hancock

Senator Dennis Hollingsworth

Senator Lois Wolk

CALIFORNIA LEGISLATURE

Senate

STATE CAPITOL
SACRAMENTO, CALIFORNIA
95814

Joint Hearing of the Senate Food and Agriculture Committee
and Senate Health Committee

Food and Beverages: Strategies to Recoup the Health Costs of Excessive Sugar Consumption

April 20, 2010 – 1:00 p.m. – State Capitol, Room 2040

REVISED AGENDA

Opening Comments – Senator Dean Florez

1. Food and Beverages: Link Between Over Consumption of Sugar and Obesity

- Gail Woodward Lopez, MPH, RD, Associate Director, the Dr. Robert C. and Veronica Atkins Center for Weight and Health, University of California, Berkeley
- Harold Goldstein, DrPH, Executive Director, California Center for Public Health Advocacy

2. Industry and Economic Perspective

- Danielle Greenberg, PhD, FACN, Director of Nutrition and Scientific Affairs at PepsiCo. Former Associate Research Professor of Psychology in Psychiatry at Cornell University Medical College
- Robert Achermann, Director of Government Relations, California Nevada Soft Drink Association
- Julian Canete, Executive Director, California Hispanic Chamber of Commerce
- Samantha Dabish, Vice President, Neighborhood Market Association

3. Biological Consequences of Sugar-Sweetened Beverage Consumption

- Michael I. Goran, PhD, Professor of Preventive Medicine, Physiology & Biophysics and Pediatrics, The Dr. Robert C. and Veronica Atkins Endowed Chair in Childhood Obesity and Diabetes, Keck School of Medicine, University of Southern California, Director of USC Childhood Obesity Research Center
- Lisa Katic, Registered Dietician, Principle, K Consulting

4. Marketing of Sugar-Sweetened Beverages and its Impact on Children

- Katie Woodruff, MPH, Deputy Director, Berkeley Media Studies Group

5. Tackling Obesity – Local Perspectives

- Genoveva Islas-Hooker, MPH, Regional Coordinator, Central California Regional Obesity Prevention Program
- Dana Richardson, Representative of the Healthy Eating Active Communities Initiative

6. Public Comment

Joint Informational Hearing

SENATE FOOD and AGRICULTURE COMMITTEE

Senator Dean Florez, Chair

and

SENATE HEALTH COMMITTEE

Senator Elaine Kontominas Alquist, Chair

***Food and Beverages: Strategies to Recoup the Health Costs of
Excessive Sugar Consumption***

Background Material

**April 20, 2010
Sacramento, California**

*Sweetened Beverage
Link to Obesity*

September 2009

Bubbling Over: Soda Consumption and Its Link to Obesity in California

Susan H. Babey, Malia Jones, Hongjian Yu and Harold Goldstein

In California, 62% of adolescents ages 12-17 and 41% of children ages 2-11 drink at least one soda or other sweetened beverage every day. In addition, 24% of adults drink at least one soda or other sweetened beverage on an average day. Adults who drink soda occasionally (not every day) are 15% more likely to be overweight or obese, and adults who drink one or more sodas per day are 27% more likely to be overweight or obese than adults who do not drink soda, even when adjusting for poverty status and race/ethnicity.

This policy brief, produced collaboratively by the California Center for Public Health Advocacy and the UCLA Center for Health Policy Research, examines soda consumption in California by cities and counties using data from the 2005 California Health Interview Survey (CHIS 2005). In addition, the brief investigates whether there is an association between soda consumption and the prevalence of overweight and obesity.

There are major differences in soda consumption rates by geographic area in California, suggesting that social and environmental factors affect the consumption of soda. Also, the prevalence of overweight and obesity is higher among those who drink one or more sodas or other sweetened beverages every day than among those who do not consume these soft drinks. Establishing public policies that focus on reducing soda consumption could contribute to reversing California's increasing overweight and obesity problem.

Background

The prevalence of overweight and obesity has increased dramatically in both adults and children in the last three decades in the

United States. In the 1970s, about 15% of adults were obese and by 2004 the rate had climbed to 32%.¹ Although the prevalence of overweight among children is lower than among adults, the rates among children and adolescents have increased considerably more. The prevalence of overweight and obesity nearly tripled among 12-19 year olds and more than quadrupled among 6-11 year olds in the last three decades.

In California, 21% of adults are currently obese and an additional 35% are overweight. Among adolescents, 14% are obese and another 16% are overweight.² Similar to national trends, the trend in California is toward increasing weight in both adults and adolescents.³ Each year in California, overweight and obesity cost families, employers, the health care industry and the government \$21 billion.⁴ California spends more public and private money on the health consequences of obesity than any other state.⁵

Overweight and obesity are associated with serious health risks. In children and adolescents, overweight and obesity are associated with increased risk for cardiovascular disease indicators including



This policy brief was developed in collaboration with the California Center for Public Health Advocacy

high total cholesterol, high blood pressure, and high fasting insulin, an early indicator of diabetes risk.⁶ In addition, overweight children and adolescents are more likely to be overweight or obese as adults.⁷ In adults, overweight and obesity are associated with increased risk for diabetes, heart disease, stroke, some types of cancer and premature death.^{1, 8, 9}

Drinking sweetened beverages such as soda and fruit drinks that have added caloric sweeteners (e.g., sucrose, high fructose corn syrup) is one marker of a poor diet, and is associated with overweight and obesity in people of all ages.¹⁰⁻¹³ A number of studies have found that greater consumption of sweetened beverages is associated with overweight and obesity among both adults and children.¹²⁻¹⁹ In addition, randomized controlled trials that examine the impact of reducing intake of sweetened beverages on weight indicate that reducing consumption of soda and other sweetened drinks leads to reductions in overweight and obesity.^{20, 21} Among adults, drinking soda is also associated with increased risk for type 2 diabetes.¹³

Moreover, drinking sweetened beverages has increased, and it is now more common than ever, particularly among adolescents.²² Between 1977 and 2002 Americans increased their calorie intake from soft drinks by 228%.²³ Portion sizes have also increased from an average serving size of 6.5 fl oz (88 calories) in the 1950s, to 12 fl oz (150 calories), 20 fl oz (266 calories), and even larger portion sizes common today.²⁴⁻²⁶ The average serving size of soft drinks in fast food restaurants in 2002 was 23 fl oz (299 calories), with some chains now commonly selling soft drinks in 32 to 64 fl oz portions (416 to 832 calories, respectively).²⁷ Sweetened beverages are a significant contributor to total caloric intake, especially for children and adolescents, and they lack the nutrients our bodies need.^{24, 26, 28} Additionally, eating habits established in childhood are important determinants of eating habits as adults.^{29, 30}

Soda Consumption in California

Drinking sweetened beverages is common among California adults, adolescents and children. Data from CHIS 2005 show that nearly one out of four adults (24%) drink at least one soda every day—6.4 million California adults—and 36% drink soda occasionally, but not every day. Forty percent of adults report not drinking soda at all. In addition, 41% of children ages 2-11 drink at least one soda every day, nearly 2.2 million children in all. The rates of soda consumption among adolescents are much higher than among adults or children. More than 62% of adolescents ages 12-17—over two million teens—drink soda every day, including 13% (over 400,000) who drink three or more sodas every day. California adolescents drink 1.2 sodas per day on average. Conservatively assuming one soda is a 12-ounce can which contains 10 teaspoons of sugar, the average California adolescent consumes the equivalent of 39 pounds of sugar each year from soda and other sweetened beverages.

Soda Consumption Associated with Higher Prevalence of Overweight and Obesity

In California, 56% of adults and 30% of adolescents are either overweight or obese. The prevalence of overweight and obesity is higher among adults and adolescents who drink soda than among those who don't.

For both adults and adolescents, rates of overweight and obesity are 18% higher among those who drink one or more sodas every day compared to those who do not drink soda. Among adults, 62% of those who drink one or more sodas daily are either overweight or obese compared to 52% of adults who do not drink soda. Among adolescents, 32% of those who consume at least one soda per day are either overweight or obese, while 27% of those who consume no sodas on a typical day are either overweight or obese.

Soda consumption is associated with poverty and race/ethnicity; lower income people and people of color tend to drink more soda.³¹ These same groups also tend to be at higher risk for overweight and obesity. However, in our analysis of California adults, the association between soda consumption and overweight or obesity was independent of poverty status and race/ethnicity. Adults who drink soda occasionally (not every day) are 15% more likely to be overweight or obese, and adults who drink one or more sodas per day are 27% more likely to be overweight or obese than adults who do not drink soda, even when adjusting for poverty status and race/ethnicity (Exhibit 1).

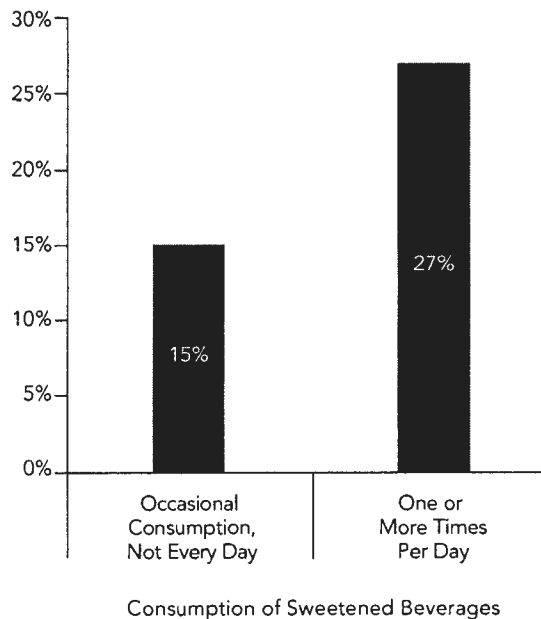
Among adolescents, the association between soda consumption and overweight is not independent of poverty status and race/ethnicity. This may be partially due to the relatively small sample size for adolescents compared to adults. Compared to white adolescents, African-American and Latino adolescents are more likely to consume soda daily, while Asian adolescents are less likely. Adolescents from lower-income families are more likely to drink soda every day compared to adolescents from higher-income families.

Soda Consumption Varies from Place to Place in California

Trends in soda consumption and obesity may be influenced by social and environmental factors. For example, the food environment, including the presence of fast-food outlets, convenience stores, grocery stores and other food vendors, has an impact on health and dietary choices of the local population.³²⁻³⁴ A recent study by the California Center for Public Health Advocacy showed that California has more than four times as many fast-food restaurants and convenience stores as grocery stores and produce vendors—suggesting that Californians have greater access to foods with lower nutritional values than to healthier foods.³⁵ Moreover, this food environment has been linked to the prevalence of obesity and diabetes among California adults.³²

Increased Likelihood of Being Overweight or Obese for Those Who Drink Sodas Compared to Those Who Do Not, Adjusted for Race/Ethnicity and Income, Adults Age 18 and Over, California, 2005

Exhibit 1



Source: 2005 California Health Interview Survey

At the same time, soda consumption is associated with the use of fast-food restaurants among adolescents, and there is wide variation in the relative availability of fast-food restaurants in California communities.^{36, 37}

Findings from CHIS 2005 show that there are major geographic differences in soda consumption in California (Exhibit 2). The percent of children drinking at least one soda each day ranges from 18% in Marin County to 61% in Imperial County. Among adolescents, the percent drinking one or more sodas each day ranges from 39% in Mendocino County to 78% in San Joaquin County. Among adults, the percent drinking one or more sodas each day ranges from just 11% in Marin County to 39% in Kings County.

Soda consumption also varies considerably among cities and census designated places (Exhibit 3). Among children and adolescents ages 2-17, the percent drinking at least one

Exhibit 2

Percent Drinking One or More Sodas per Day by County or County Group, Children, Adolescents and Adults, California, 2005

	Children Ages 2-11	Adolescents Ages 12-17	Adults Age 18 and Over
	One or More Sodas %	One or More Sodas %	One or More Sodas %
California	41.2	62.2	24.3
Alameda	31.0	58.9	17.4
Butte	30.4	61.8	20.3
Contra Costa	40.7	47.2	21.2
Del Norte, Siskiyou, Lassen, Trinity, Modoc, Plumas, Sierra	24.5	63.0	20.8
El Dorado	31.8	55.3	21.6
Fresno	53.1	68.7	35.0
Humboldt	33.2	50.3	16.4
Imperial	60.7	61.2	36.4
Kern	55.0	67.2	36.6
Kings	57.2	57.7	39.1
Lake	31.6	62.8	30.1
Los Angeles	44.3	64.9	25.5
Madera	39.9	75.3	37.4
Marin	18.4	41.3	10.6
Mendocino	38.1	39.0	18.8
Merced	55.4	*	32.7
Monterey	32.8	58.1	27.1
Napa	41.5	56.8	27.3
Nevada	25.6	40.9	17.5
Orange	36.9	56.4	23.4
Placer	31.5	66.2	18.4
Riverside	40.6	69.5	29.5
Sacramento	35.4	55.5	23.6
San Benito	26.4	58.9	25.6
San Bernardino	49.6	68.5	29.6
San Diego	34.8	63.1	21.1
San Francisco	21.5	42.1	10.9
San Joaquin	44.2	77.8	26.6
San Luis Obispo	41.7	66.8	18.3
San Mateo	32.5	50.1	14.4
Santa Barbara	39.8	53.8	19.0
Santa Clara	40.9	48.2	21.1
Santa Cruz	41.4	56.0	15.5
Shasta	32.0	60.0	27.5
Solano	45.2	58.7	26.1
Sonoma	42.0	60.7	20.7
Stanislaus	47.5	*	34.3
Sutter	44.5	*	29.2
Tehama, Glenn, Colusa	36.8	*	30.1
Tuolumne, Calaveras, Amador, Inyo, Mariposa, Mono, Alpine	35.0	*	17.3
Tulare	44.2	71.0	36.1
Ventura	39.0	60.4	24.8
Yolo	37.3	62.4	13.9
Yuba	50.5	62.9	30.9

Note:

* Indicates the estimate was not statistically reliable. Not all differences between rates are statistically significant. The 95% confidence intervals are available at: http://www.healthpolicy.ucla.edu/soda_consumption.html

Source: 2005 California Health Interview Survey

Percent Drinking One or More Sodas per Day by Cities and Census Designated Places,
California, 2005

Exhibit 3

	Children and Adolescents Ages 2-17 %	Adults Age 18 and Over %		Children and Adolescents Ages 2-17 %	Adults Age 18 and Over %
California	49.4	24.3	Mission Viejo	43.3	18.0
Anaheim	45.4	26.5	Modesto	57.0	31.8
Antioch	44.8	21.9	Moreno Valley	55.4	33.7
Bakersfield	60.1	33.9	Murrieta	49.7	26.5
Baldwin Park	52.2	29.0	Norwalk	51.5	31.0
Bellflower	51.3	30.9	Oakland	44.1	20.6
Buena Park	44.0	24.5	Oceanside	47.7	20.8
Burbank	48.3	19.6	Ontario	57.7	32.9
Carlsbad	43.5	16.3	Orange	46.0	22.6
Carson	52.7	25.0	Oxnard	50.6	30.0
Chino	56.3	31.2	Palmdale	54.9	32.1
Chino Hills	52.4	22.2	Pasadena	54.2	22.9
Chula Vista	46.2	23.1	Pomona	56.6	29.5
Citrus Heights	39.4	21.9	Rancho Cucamonga	54.6	26.0
Clovis	53.8	27.0	Redding	44.2	25.3
Compton	54.7	33.2	Rialto	59.4	32.8
Concord	44.2	21.5	Richmond	46.1	28.4
Corona	50.7	29.6	Riverside	49.8	31.7
Costa Mesa	43.5	25.0	Roseville	43.6	16.4
Daly City	38.3	13.7	Sacramento	44.3	25.4
Downey	51.4	29.6	Salinas	46.9	28.9
East Los Angeles *	53.3	38.4	San Bernardino	58.6	32.7
El Cajon	47.6	22.2	San Buenaventura (Ventura)	46.6	22.3
El Monte	51.8	29.2	San Diego	46.2	22.8
Elk Grove *	43.3	21.2	San Francisco	36.9	11.5
Escondido	48.1	22.6	San Jose	42.8	21.7
Fairfield	47.0	26.5	Santa Ana	47.3	33.2
Florence-Graham *	54.2	36.5	Santa Clara	40.6	19.2
Fontana	57.5	31.9	Santa Clarita	49.9	20.6
Fremont	38.0	14.1	Santa Maria	48.3	24.1
Fresno	57.4	33.5	Santa Rosa	45.4	19.7
Fullerton	44.0	23.6	Simi Valley	44.0	20.5
Garden Grove	43.9	24.0	Southgate	52.9	36.8
Glendale	47.6	19.6	Stockton	57.3	28.1
Hawthorne	53.2	31.4	Sunnyvale	39.8	18.7
Hayward	41.3	18.4	Temecula	47.8	28.2
Hesperia	55.5	27.2	Thousand Oaks	43.8	19.8
Huntington Beach	40.7	20.7	Torrance	46.0	18.9
Indio	55.6	37.5	Tracy	56.9	24.9
Inglewood	55.0	32.6	Vacaville	45.4	25.4
Irvine	43.6	19.5	Vallejo	48.8	25.7
Lancaster	54.8	30.7	Victorville	57.0	29.2
Livermore	41.1	15.1	Visalia	56.3	30.8
Long Beach	51.5	27.2	Vista	48.8	23.8
Los Angeles	51.9	24.8	West Covina	50.4	21.6
Lynwood	53.5	33.3	Westminster	42.8	22.4
Merced	61.9	33.3			

Note:

* Indicates a Census Designated Place. Census designated places are communities that lack separate governments but otherwise resemble incorporated places such as cities. This table includes only cities in which the combined population of children and adolescents ages 2-17 was at least 20,000. Not all differences between rates are statistically significant. The 95% confidence intervals are available at: http://www.healthpolicy.ucla.edu/soda_consumption.html

Source: 2005 California Health Interview Survey

soda per day ranged from 37% in San Francisco to 62% in Merced. Among adults, the percent drinking at least one soda per day ranged from 12% in San Francisco to 38% in East Los Angeles.

Conclusions

In California, 62% of adolescents ages 12-17 and 41% of children ages 2-11 drink at least one soda or other sweetened beverage every day. In addition, nearly one out of four adults (24%) drink soda every day and 36% drink soda occasionally. This amounts to 10.7 million Californians over the age of one who drink at least one soda each day. This soda consumption greatly increases the amount of added sugar and other caloric sweeteners in the diet of Californians without contributing substantially to the nutritional needs of the population.

For both adults and adolescents, the prevalence of overweight and obesity is higher among those who drink one or more sodas or other sweetened beverages every day than among those who do not. Among adults, even after adjusting for race and household income, those who drink one or more sodas each day are 27% more likely to be overweight or obese than adults who do not drink soda. These findings are consistent with other research.³⁸ Additionally, childhood eating habits and weight status are important determinants of health as adults.^{7, 29, 30} Taken together, these findings suggest a number of potential benefits from reducing soft drink consumption including reduced risk of obesity, improved dietary intake and reduced risk of diabetes.

Data Source and Methods

This policy brief examines geographical variation in soda consumption among children, adolescents and adults in California as well as its association with overweight and obesity among adults and adolescents using data from the 2005 California Health Interview Survey (CHIS 2005). All statements in this report that compare rates for one group with another group reflect statistically significant differences ($p < 0.05$) unless otherwise noted. CHIS 2005 completed interviews with over 4,000 adolescents and over 43,000 adults, drawn from every county in the state, in English, Spanish, Chinese (both Mandarin and Cantonese),

Vietnamese and Korean. The California Health Interview Survey is a collaboration of the UCLA Center for Health Policy Research, California Department of Public Health, the California Department of Health Care Services and the Public Health Institute. Funding for the CHIS 2005 statewide survey was provided by the California Department of Health Care Services, The California Endowment, the National Cancer Institute, The Robert Wood Johnson Foundation, the California Children and Families Commission, the California Office of the Patient Advocate, the California Department of Mental Health, the Centers for Disease Control and Prevention (CDC) and Kaiser Permanente. For local funders and other information on CHIS, visit www.chis.ucla.edu.

In adults, overweight is defined as a Body Mass Index (BMI) between 25 and 30, while obesity is defined as BMI of 30 or greater. Among adolescents, overweight is defined as having a BMI between the 85th and 95th percentile on the Centers for Disease Control and Prevention's BMI-for-age growth charts, while obesity is defined as having a BMI above the 95th percentile.³⁹

Adults and adolescents self-reported their consumption of soda and other sweetened beverages. Adults were asked the following two questions: "During the past month, how many times (per day, per week or per month) did you drink soda such as Coke or 7-Up? Do not include diet soda." and "How many times did you drink fruit-flavored drinks such as lemonade or Sunny Delight? Do not include diet drinks." Responses to these questions were combined and converted to a common metric to estimate daily consumption of soda and other sweetened beverages. Adolescents were asked: "Yesterday, how many glasses or cans of soda such as Coke, or other sweetened drinks such as fruit punch or Sunny Delight did you drink? Do not count diet drinks." For children ages 2-11, the most knowledgeable parent or guardian responded to the following question: "Yesterday, how many glasses or cans of soda such as Coke or other sweetened drinks such as fruit punch or Sunny Delight did (he/she) drink? Do not count diet drinks." For all respondents, consumption of 100% fruit juice was reported in a previous question and is not included in our estimates of sweetened beverage consumption.

We used small-area estimation to generate model-based estimates of the proportion of adults and children who consume one or more sodas per day for each city.^{40, 41} Small-area estimation uses modeling to produce estimates for small geographic areas, such as cities, for which there is not sufficient sample to produce direct estimates. The models are based on

individual-level demographic and health outcome data from CHIS 2005 as well as demographic data at the census block group level from the Census and Claritas Inc. To maximize the reliability and validity of the estimates, we present only estimates for cities with a population of at least 20,000 for the age group being modeled. For more information about small-area estimation methodology, see: Yu H, Meng YY, Mendez-Luck CA, Jhavar M, Wallace SP. *Small-Area Estimation of Health Insurance Coverage for California Legislative Districts*.

Author Information

Susan H. Babey, PhD, is a senior research scientist at the UCLA Center for Health Policy Research. Malia Jones, MPH, is a graduate student researcher at the UCLA Center for Health Policy Research. Hongjian Yu, PhD, is the director for statistical support at the UCLA Center for Health Policy Research. Harold Goldstein, DrPH, is the Executive Director of the California Center for Public Health Advocacy.

Acknowledgements

The authors wish to thank Theresa Hastert, MPP, Jenny Chia, PhD, Lijie Di, MS, Gwen Driscoll and Celeste Maglan for their assistance. The authors would also like to thank the following individuals for their helpful comments: Stefan Harvey, Assistant Director, the California Center for Public Health Advocacy; Lisa Hershey, MPH, Chief, Coordinating Office for Obesity Prevention, California Department of Public Health; Leslie Mikkelsen, MPH, Managing Director, Prevention Institute; Gail Woodward-Lopez, MPH, RD, Associate Director, Center for Weight and Health, University of California, Berkeley.

Funding

Support for this policy brief was provided by a grant from The California Endowment to the California Center for Public Health Advocacy.

Suggested Citation

Babey SH, Jones M, Yu H, Goldstein H. *Bubbling Over: Soda Consumption and Its Link to Obesity in California*. UCLA Center for Health Policy Research and California Center for Public Health Advocacy, 2009.

Endnotes

- 1 Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999-2004. *JAMA*. Apr 5 2006;295(13):1549-1555.
- 2 2005 California Health Interview Survey.
- 3 Babey SH, Grant D, Brown ER. *Adult Smoking Rate Declines, While Asthma, Diabetes and Obesity Rates Rise*. Los Angeles: UCLA Center for Health Policy Research; Nov 2006.

- 4 *The Economic Costs of Overweight, Obesity, and Physical Inactivity Among California Adults – 2006* (July 2009). The California Center for Public Health Advocacy.
- 5 Finkelstein EA, Fiebelkorn IC, Wang G. State-level estimates of annual medical expenditures attributable to obesity. *Obesity Research*. Jan 2004;12(1):18-24.
- 6 Freedman DS, Mei Z, Srinivasan SR, Berenson GS, Dietz WH. Cardiovascular risk factors and excess adiposity among overweight children and adolescents: the Bogalusa Heart Study. *Journal of Pediatrics*. Jan 2007;150(1):12-17 e12.
- 7 Guo SS, Wu W, Chumlea WC, Roche AF. Predicting overweight and obesity in adulthood from body mass index values in childhood and adolescence. *American Journal of Clinical Nutrition*. Sep 2002;76(3):653-658.
- 8 Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The disease burden associated with overweight and obesity. *JAMA*. Oct 27 1999;282(16):1523-1529.
- 9 Ogden CL, Yanovski SZ, Carroll MD, Flegal KM. The epidemiology of obesity. *Gastroenterology*. May 2007;132(6):2087-2102.
- 10 French SA, Lin B-H, Guthrie JF. National trends in soft drink consumption among children and adolescents age 6 to 17 years: Prevalence, amounts, and sources, 1977/1978 to 1994/1998. *Journal of the American Dietetic Association*. 2003;103(10):1326-1331.
- 11 Harnack L, Stang J, Story M. Soft Drink Consumption Among U.S. Children and Adolescents: Nutritional Consequences. *Journal of the American Dietetic Association*. 1999;99(4):436-441.
- 12 Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systematic review. *American Journal of Clinical Nutrition*. Aug 2006;84(2):274-288.
- 13 Schulze MB, Manson JE, Ludwig DS, et al. Sugar-sweetened beverages, weight gain, and incidence of type 2 diabetes in young and middle-aged women. *JAMA*. Aug 25 2004;292(8):927-934.
- 14 Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. *The Lancet*. 2001;357(9255):505-508.
- 15 Giammattei J, Blix G, Marshak HH, Wollitzer AO, Pettitt DJ. Television watching and soft drink consumption: associations with obesity in 11- to 13-year-old schoolchildren. *Archives of Pediatric and Adolescent Medicine*. Sep 2003;157(9):882-886.
- 16 Phillips SM, Bandini LG, Naumova EN, et al. Energy-dense snack food intake in adolescence: longitudinal relationship to weight and fatness. *Obesity Research*. Mar 2004;12(3):461-472.
- 17 LaRowe TL, Moeller SM, Adams AK. Beverage patterns, diet quality, and body mass index of U.S. preschool and school-aged children. *Journal of the American Dietetic Association*. Jul 2007;107(7):1124-1133.
- 18 Welsh JA, Cogswell ME, Rogers S, Rockett H, Mei Z, Grummer-Strawn LM. Overweight among low-income preschool children associated with the consumption of sweet drinks: Missouri, 1999-2002. *Pediatrics*. Feb 2005;115(2):e223-229.
- 19 Dubois L, Farmer A, Girard M, Peterson K. Regular sugar-sweetened beverage consumption between meals increases risk of overweight among preschool-aged children. *Journal of the American Dietetic Association*. Jun 2007;107(6):924-934: discussion 934-925.
- 20 Ebbeling CB, Feldman HA, Osganian SK, Chomitz VR, Ellenbogen SJ, Ludwig DS. Effects of decreasing sugar-sweetened beverage consumption on body weight in adolescents: a randomized, controlled pilot study. *Pediatrics*. Mar 2006;117(3):673-680.

UCLA CENTER FOR HEALTH POLICY RESEARCH



The UCLA Center
for Health Policy Research
is affiliated with the
UCLA School of Public Health
and the UCLA School of Public Affairs.

The views expressed in this policy brief
are those of the authors and do not
necessarily represent the UCLA Center for
Health Policy Research, the Regents of the
University of California, or collaborating
organizations or funders.

PB2009-5

Copyright © 2009 by the Regents of the
University of California and the California
Center for Public Health Advocacy

Editor-in-Chief: E. Richard Brown, PhD

Phone: 310-794-0909

Fax: 310-794-2686

Email: chpr@ucla.edu

Web Site: www.healthpolicy.ucla.edu

- 21 James J, Thomas P, Cavan D, Kerr D. Preventing childhood obesity by reducing consumption of carbonated drinks: cluster randomised controlled trial. *British Medical Journal*. May 22 2004;328(7450):1237.
- 22 Wang YC, Bleich SN, Gortmaker SL. Increasing caloric contribution from sugar-sweetened beverages and 100% fruit juices among U.S. children and adolescents, 1988-2004. *Pediatrics*. Jun 2008;121(6):e1604-1614.
- 23 Duffey KJ, Popkin BM. Shifts in patterns and consumption of beverages between 1965 and 2002. *Obesity (Silver Spring)*. Nov 2007;15(11):2739-2747.
- 24 Jacobsen M. Liquid candy: How soft drinks are harming Americans' health. <http://www.cspinet.org/liquidcandy/>. Accessed December 22, 2008.
- 25 Nielsen SJ, Popkin BM. Patterns and trends in food portion sizes, 1977-1998. *JAMA*. Jan 22-29 2003;289(4):450-453.
- 26 Nielsen SJ, Popkin BM. Changes in beverage intake between 1977 and 2001. *American Journal of Preventive Medicine*. Oct 2004;27(3):205-210.
- 27 Young LR, Nestle M. Expanding portion sizes in the U.S. marketplace: implications for nutrition counseling. *Journal of the American Dietetic Association*. Feb 2003;103(2):231-234.
- 28 Block G. Foods contributing to energy intake in the U.S.: data from NHANES III and NHANES 1999-2000. *Journal of Food Composition and Analysis*. 2004;17(3-4):439-447.
- 29 Mikkila V, Rasanen L, Raitakari OT, Pietinen P, Viikari J. Consistent dietary patterns identified from childhood to adulthood: the cardiovascular risk in Young Finns Study. *British Journal of Nutrition*. Jun 2005;93(6):923-931.
- 30 Lake AA, Mathers JC, Rugg-Gunn AJ, Adamson AJ. Longitudinal change in food habits between adolescence (11-12 years) and adulthood (32-33 years): the ASH30 Study. *Journal of Public Health*. Mar 2006;28(1):10-16.
- 31 Rehm CD, Matte TD, Van Wye G, Young C, Frieden TR. Demographic and behavioral factors associated with daily sugar-sweetened soda consumption in New York City adults. *Journal of Urban Health*. May 2008;85(3):375-385.
- 32 *Designed for Disease: the Link Between Local Food Environments and Obesity and Diabetes*: California Center for Public Health Advocacy, PolicyLink, and the UCLA Center for Health Policy Research.; April 2008.
- 33 Morland K, Diez Roux A, Wing S. Supermarkets, other food stores, and obesity: the atherosclerosis risk in communities study. *American Journal of Preventive Medicine*. Apr 2006;30(4):333-339.
- 34 Morland K, Wing S, Diez Roux A. The contextual effect of the local food environment on residents' diets: the atherosclerosis risk in communities study. *American Journal of Public Health*. Nov 2002;92(11):1761-1767.
- 35 *Searching for healthy food: The food landscape in California cities and counties*: California Center for Public Health Advocacy; 2007.
- 36 Taveras EM, Berkey CS, Rifas-Shiman SL, et al. Association of consumption of fried food away from home with body mass index and diet quality in older children and adolescents. *Pediatrics*. Oct 2005;116(4):e518-524.
- 37 Wiecha JL, Finkelstein D, Troped PJ, Fragala M, Peterson KE. School vending machine use and fast-food restaurant use are associated with sugar-sweetened beverage intake in youth. *Journal of the American Dietetic Association*. Oct 2006;106(10):1624-1630.
- 38 Vartanian LR, Schwartz MB, Brownell KD. Effects of Soft Drink Consumption on Nutrition and Health: A Systematic Review and Meta-Analysis. *American Journal of Public Health*. April 1, 2007 2007;97(4):667-675.
- 39 Centers for Disease Control and Prevention. Defining overweight and obesity. <http://www.cdc.gov/nccdphp/dnpa/obesity/defining.htm>. Accessed November 14, 2008.
- 40 Yu H, Meng YY, Mendez-Luck CA, Jhavar M, Wallace SP. Small-area estimation of health insurance coverage for California legislative districts. *American Journal of Public Health*. Apr 2007;97(4):731-737.
- 41 Mendez-Luck CA, Yu H, Meng YY, Jhavar M, Wallace SP. Estimating health conditions for small areas: asthma symptom prevalence for state legislative districts. *Health Services Research*. Dec 2007;42(6 Pt 2):2389-2409.



Prepared for
The California Center for Public Health Advocacy

by

The Dr. Robert C. and Veronica Atkins
Center for Weight and Health
University of California, Berkeley
January 2009

Authors:
Gail Woodward-Lopez
Janice Kao
Lorrene Ritchie

UNIVERSITY OF CALIFORNIA, BERKELEY

BERKELEY • DAVIS • IRVINE • LOS ANGELES • MERCED • RIVERSIDE • SAN DIEGO • SAN FRANCISCO



SANTA BARBARA • SANTA CRUZ

COLLEGE OF NATURAL RESOURCES
DEPARTMENT OF NUTRITIONAL SCIENCES AND TOXICOLOGY

BERKELEY, CALIFORNIA 94720-3104
Tel: (510) 642-6490 Fax: (510) 642-0535

THE DR. ROBERT C. AND VERONICA ATKINS

Center for Weight and Health

COLLEGE OF NATURAL RESOURCES & SCHOOL OF PUBLIC HEALTH
BERKELEY, CALIFORNIA 94720-3100
101 GIANNINI HALL #3100
9 Morgan Hall #3104

To What Extent Have Sweetened Beverages Contributed to the Obesity Epidemic?

Executive Summary

A comprehensive literature review and analysis was conducted by the University of California, Berkeley, Dr. Robert C. and Veronica Atkins Center for Weight and Health to examine the extent to which sweetened beverages have contributed to obesity. Multiple lines of evidence were examined to give a complete picture of if, and how, sweetened beverages lead to excess weight gain.

The preponderance of evidence supports the conclusion that consumption of sweetened beverages is a substantial contributor to the obesity epidemic. Data were examined from a variety of sources and inclusive of a variety of study designs, ranging from national intake trends, mechanistic studies examining response to liquid calorie challenges, observational studies involving cross-sectional and longitudinal associations between sweetened beverage intake, calories and adiposity, as well as intervention trials manipulating the intake of sweetened beverage intake and measuring resultant change in body weight.

All lines of evidence consistently support the conclusion that consumption of sweetened beverages increases the risk of overweight and has contributed to the obesity epidemic experienced over the last several decades. Reducing the consumption of sweetened beverages is an important strategy to reverse obesity trends in the U.S.

The Center for Weight and Health gratefully acknowledges a national panel of experts for their review of and input into this analysis (see Acknowledgements section at the end of this Summary).

Obesity rates have risen dramatically since the late 1970s:

- In the last 30 years, obesity has become epidemic in the U.S. – rising by nearly 50% in adults and tripling among children.
- Although no group has escaped the epidemic, ethnic minorities and the poor are most affected.

Increases in calorie intake parallel the rise in obesity:

- Changes in recent decades in physical activity appear limited compared to the dramatic increases in energy intakes.
- Between the late 1970s and 2000, the amount of energy **available** for human consumption increased by more than 500 calories per day for each person in the U.S.
- National surveys of food intake suggest a smaller, but nevertheless substantial, increase over the same time period of nearly **300 calories** per day by each person.

Sweetened beverage consumption also parallels the rise in obesity:

- Sweetened beverage intake and the prevalence of obesity have risen in tandem over the last several decades.
- Increased intake of sweetened beverages has been noted among all age and gender groups, but higher intakes have been observed among groups experiencing the highest obesity rates.
- Of 23 food groups soft drinks were the largest contributor of energy to the diet and the top source of liquid calories in the U.S. Soda accounts for the majority of the sweetened beverages consumed.

Sweetened beverages account for a large portion of the increase in calorie intake and therefore are a likely contributor to excess weight gained in recent decades:

- According to national survey data, the increase in calorie consumption from sweetened beverages is equivalent to 43% of the total increase in calorie consumption between 1977 and 2001.

Multiple studies explain how sweetened beverages lead to the consumption of excess calories:

- Numerous studies consistently show an association between the intake of sweetened beverages and higher calorie intake.
- Calories in liquid form are not well compensated for by reductions in the intake of other sources of energy; therefore calories from sweetened beverages tend to be “extra” calories that lead to higher total energy intake.
- Lack of compensation for calories from sweetened beverages is likely due to the low satiety value of calories in liquid form (i.e. liquid calories are not as filling as calories in solid form).
- Increases in portion sizes, marketing and accessibility are likely contributors to excessive consumption of sweetened beverages.

Studies consistently find that consuming more sweetened beverages is associated with higher weights:

- The majority of observational studies on diet and weight found a significant association between sweetened beverage intake and adiposity.

Intervention trials have demonstrated that reducing sweetened beverage intake can help prevent excess weight gain and that increasing sweetened beverage intake can lead to weight gain:

- Randomized, controlled trials in children successfully reduced sweetened beverage intake and observed trends toward lower adiposity among some groups.
- Randomized, controlled or crossover trials in adults showed that increasing sweetened beverage intake resulted in weight gain.

ACKNOWLEDGEMENTS

The UC Berkeley Center for Weight and Health gratefully acknowledges the following national panel of experts for their review of and input into this analysis:

- George Bray, MD, Boyd Professor, Pennington Center, Louisiana State University
- Patricia Crawford, DrPH, Director, University of California, Berkeley, Dr. Robert C. and Veronica Atkins Center for Weight and Health
- Steven Hooker PhD, Director, Prevention Research Center, University of South Carolina
- Richard Mattes, MPH, PhD, RD, Professor, Foods and Nutrition, Purdue University
- Harry Popkin, PhD, Professor, Department of Nutrition, Gillings School of Public Health, and Director, Interdisciplinary Obesity Center, University of North Carolina
- Jini Sallis, PhD, Professor Department of Psychology, San Diego State University and Director of The Robert Wood Johnson Foundation Active Living Research
- Marlene Schwartz, PhD, Deputy Director, Rudd Center for Food Policy and Obesity, Yale University
- Mary Story, PhD, Professor, Division of Epidemiology, School of Public Health, University of Minnesota and Director of The Robert Wood Johnson Foundation Active Living Research
- Y. Claire Wang, MD, Assistant Professor of Health Policy & Management, Mailman School of Public Health, Columbia University

To What Extent Have Sweetened Beverages Contributed to the Obesity Epidemic?

To answer this question, the UC Berkeley Dr. Robert C. and Veronica Atkins Center for Weight and Health conducted a comprehensive literature review to examine four lines of evidence:

Secular trends in the intake of sweetened beverages¹ to determine the extent to which changes in the intake of these beverages correspond to recent rises in the prevalence of obesity.

Plausible mechanisms that might explain the relationship between sweetened beverage intake and obesity, including metabolic, short-term experimental and observational studies that examine the relationship between sweetened beverage intake and calorie intake.

Observational studies that examine the relationship between adiposity² and intake of sweetened beverages in free-living populations.

Intervention trials that examine the impact on adiposity of programs designed to improve health or prevent weight gain by reducing the consumption of sweetened beverages (often in conjunction with other dietary and/or physical activity changes) or that study the impact on weight of prescribed increases in sweetened beverage intake. Because of inherent differences between obesity prevention and treatment, weight loss interventions were excluded.

The literature search included peer-reviewed articles with publication dates from January 1992 through January 2008. A more detailed discussion of the search methodology and inclusion criteria is discussed elsewhere (Woodward-Lopez, 2006).

I. Obesity Trends

Obesity has risen dramatically in the U.S. since the late 1970s

During the last 30 years, there has been a substantial increase in the prevalence of overweight and obesity³ among adults in the United States. From the 1960s to the late 1970s, rates were relatively constant with about 31% of adults overweight and an additional 15% classified as obese (Flegal, 1998). After the 1970s these rates began to climb. By 2004, 34% of adults were overweight and an additional 32% were further classified as obese (Ogden, 2006) (Figure 1).

¹ For the purposes of this paper when speaking in general terms, “sweetened beverages” refers to any combination of beverages that contains added caloric sweetener. However, individual studies and data sets referred to in this paper used variable definitions, most commonly as follows: “sweetened beverages” - any beverage with added caloric sweetener, most commonly includes fruit-flavored drinks and sodas and sometimes includes low-calorie drinks, sweetened teas and coffees; “soft drinks or sodas” - calorically sweetened carbonated beverages (sometimes includes diet drinks); “fruit drinks” - non-carbonated fruit-flavored drinks with added caloric sweetener including “ades” and punches. Please see appendices for definitions that were available for individual studies. In many cases no definition was provided.

² For the purposes of this paper, adiposity refers to any measure of body fat, including Body Mass Index (BMI) as well as more direct measures such as skinfold measures, bioelectrical impedance and dual-energy x-ray absorptiometry (DXA).

³ Overweight in adults is defined as a BMI ≥ 25 and < 30 ; obesity in adults is defined as BMI of ≥ 30 .

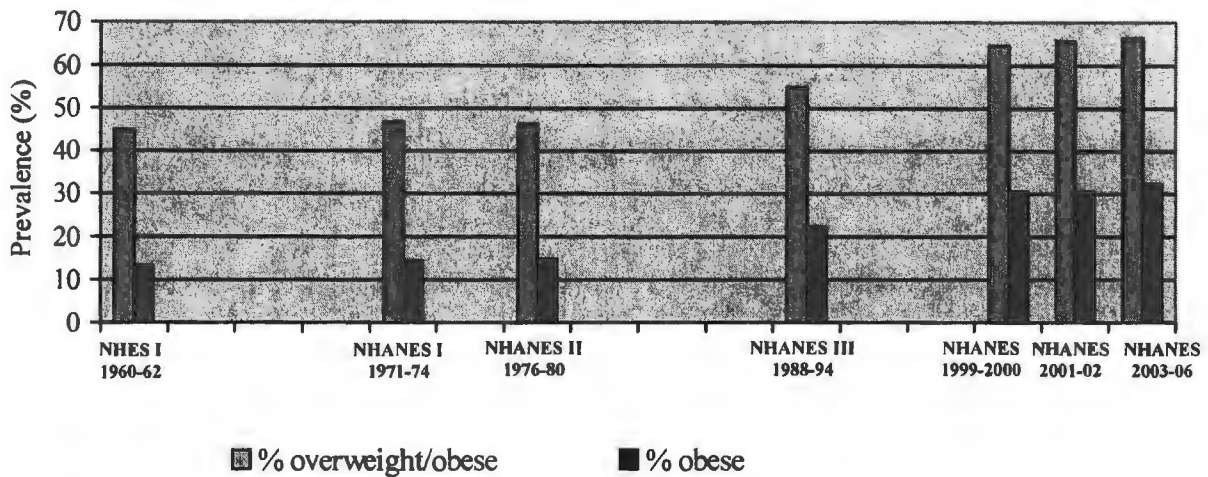


Figure 1. Trends in Overweight and Obesity in U.S. Adults, Age ≥20 years (NHANES⁴).
 (Sources: Flegal et al., 1998; Ogden et al., 2006.)

In the U.S. rising rates of obesity are most dramatic among children

Although the prevalence of overweight⁵ among children is lower than among adults, the rates among children have increased considerably more in recent decades. As with adults the prevalence of overweight was relatively constant from the 1960s to the 1970s, but since the early seventies, the prevalence of overweight has more than quadrupled among 6-11 year olds and nearly tripled among 12-19 year olds (Ogden, 2002; Ogden, 2008) (Figure 2). Even very young children in the U.S. have been affected: 12% of 2-5 year old children were overweight in 2006 (Ogden, 2008).

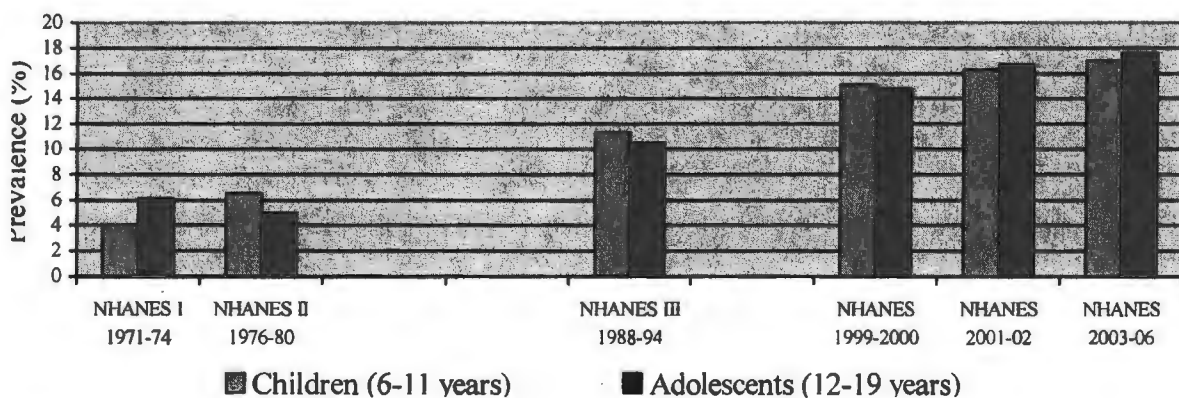


Figure 2. Trends in Overweight in U.S. Children and Adolescents (NHANES).
 (Sources: Ogden et al., 2002; Ogden et al., 2006 ; Ogden et al., 2008.)

⁴ National Health and Nutrition Examination Survey

⁵ Overweight in children defined as a BMI > 95th percentile for age and gender

Ethnic minorities and the poor are the most affected by obesity

Obesity crosses all ethnic and socioeconomic lines in the U.S. No group—young or old, rich or poor—has escaped this epidemic. However, African Americans, Hispanics and Native Americans are disproportionately affected (Flegal, 1998; Hedley, 2004; Ogden, 2006; Crawford, 2001) (Figure 3). Asian-Americans tend to have the lowest prevalence but have also experienced rapid increases in overweight and obesity in recent decades (Bates, 2008). Among immigrant populations, subsequent generations of Americans tend to be heavier, suggesting that the environment in the U.S. and the westernized lifestyle are contributing to obesity (Bates, 2008) (Figure 4).

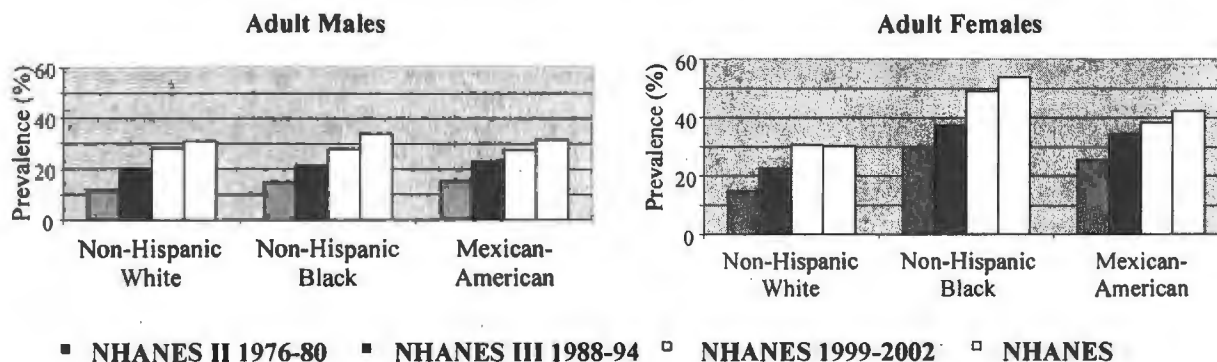


Figure 3. Trends in Obesity in U.S. Adults by Ethnicity (NHANES). (Sources: Flegal et al., 1998; Hedley et al., 2004; Ogden et al., 2006.)

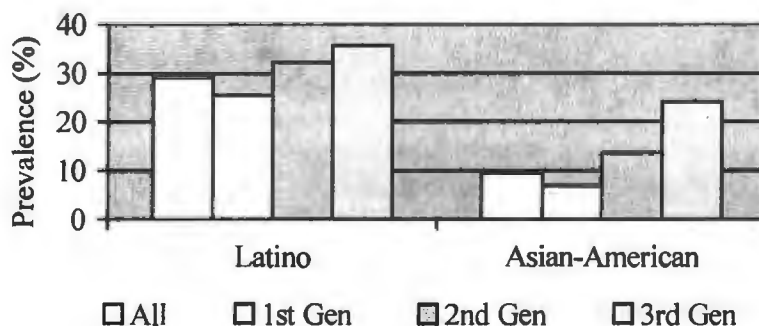


Figure 4. Prevalence of Obesity in U.S. Adults by Generational Status (National Latino and Asian-American Survey, 2003). (Source: Bates et al., 2008)

As with adults, all ethnic groups of children have experienced rapid rises in the prevalence of overweight over the past few decades. Some groups, such as Non-Hispanic White and African-American boys, which had relatively low prevalence in 1976-80, have experienced the most substantial increases (Baskin, 2005; Ogden, 2006; Ogden, 2008) (Figures 5 and 6). Currently African American girls and Mexican American boys have the highest prevalence of overweight among children (Figures 5 and 6).

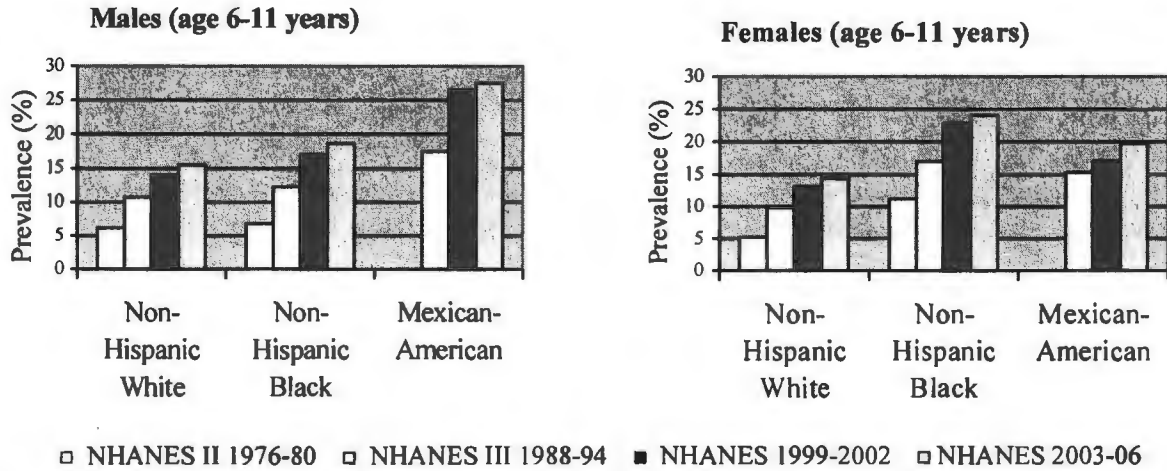


Figure 5. Trends in Overweight in U.S. Children by Ethnicity (NHANES). (Sources: Baskin et al., 2005; Ogden et al., 2006; Ogden et al., 2008.)

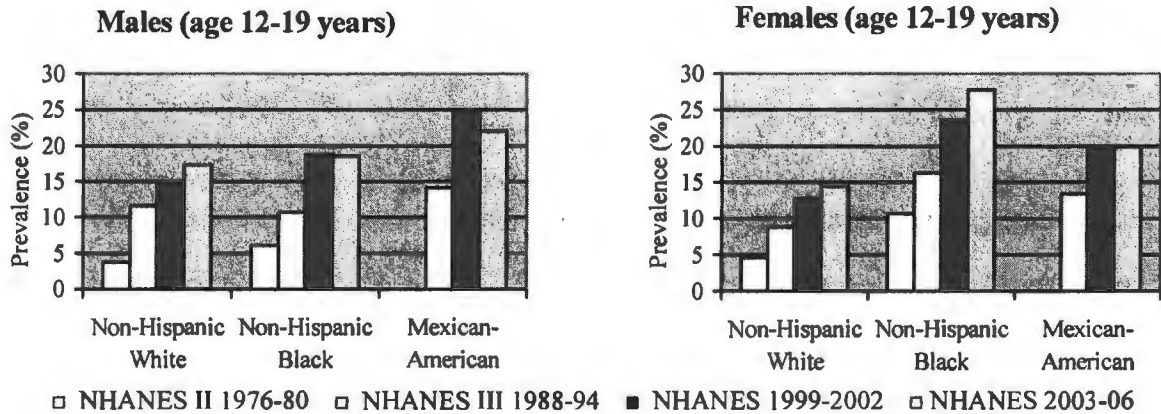


Figure 6. Trends in Overweight in U.S. Adolescents by Ethnicity (NHANES). (Sources: Baskin et al., 2005; Ogden et al., 2006 ; Ogden et al., 2008.)

Obesity rates tend to be higher among the less well educated and lower income individuals in the U.S. (Zhang, 2004; Patterson, 1997; Troiano, 1998) (Figures 7 and 8). The complex relationship between socioeconomic status and obesity suggests that factors such as decreased access to healthy, lower calorie foods and decreased purchasing power, combined with unsafe neighborhoods and less access to opportunities to be physically active, may contribute to obesity among low-income populations. Targeted marketing of “empty calorie” foods to low-income and minority populations has also been implicated as a factor (IOM, 2006). Education may also play a role. For example, adults with more education have higher intakes of lower calorie beverages, such as diet sodas and lower fat milks, and lower intakes of sweetened beverages, than individuals with lower educational attainments (Shimakawa, 1994; Binkley, 2007).

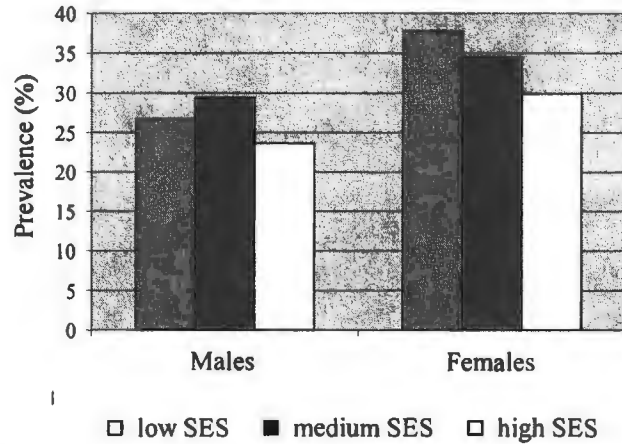


Figure 7. Prevalence of Obesity in U.S. Adults by Socioeconomic Status (NHANES 1999 - 2000). (Source: Zhang and Wang, 2004.)

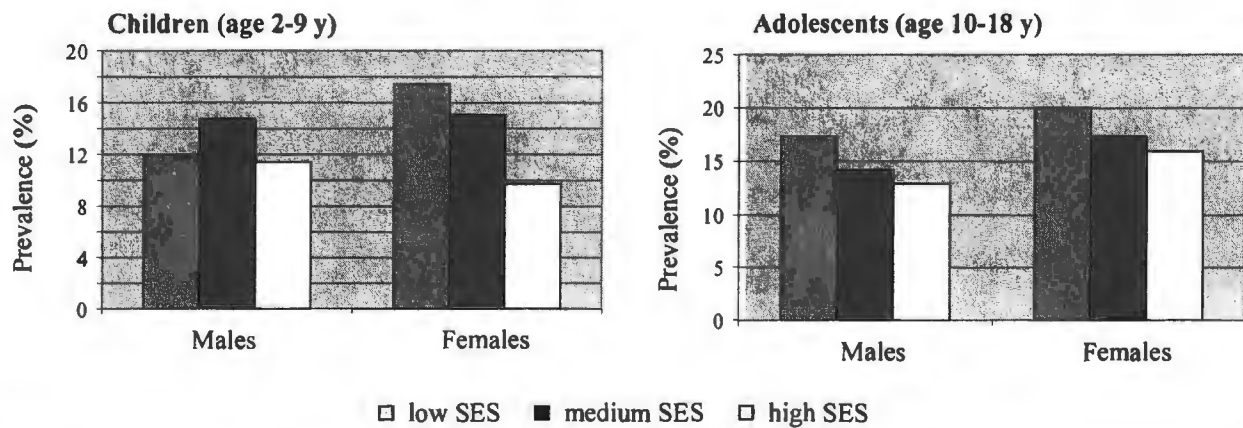


Figure 8. Prevalence of Overweight in U.S. Children and Adolescents by Socioeconomic Status (NHANES 1999 – 2002). (Source: Wang and Zhang, 2006.)

California is the most ethnically diverse state in the nation with 36% Latino and 43% Non-Hispanic Whites, and the remaining 21% African-Americans, Asians, mixed race and a variety of other ethnic groups (U.S. Census, 2008). Regardless of race, rates of obesity among California preschoolers (aged 2-5 years) from low-income families are considerably higher (17.4%) than for comparable US children (13.9%) according to Pediatric Nutrition Surveillance System PedNSS) (CMS, 2008). Because communities of color are disproportionately affected by obesity, California is particularly vulnerable to the consequences of the obesity epidemic, presently spending more public and private money on the health consequences of obesity than any other state (CDHS, 2005).

Obesity increases the risk of developing many diseases and health problems

Obesity is an indicator of poor nutrition and inadequate physical activity, the consequences of which are predicted to overturn historical gains in the health of Americans. Obesity increases the risk of diabetes, heart disease, certain types of cancer, arthritis, asthma and breathing problems (Must, 1999; IOM, 2005). Depending on their level of obesity, from 60% to over 80% of obese adults have type 2 diabetes, high blood cholesterol, high blood pressure, or other related conditions (Must, 1999). It has been reported that up to 60% of obese children aged 5-10 years have early signs of heart disease (Freedman, 1999). Type 2 diabetes, previously only seen among adults, is now increasing among children. If the current obesity trends are not reversed, it is predicted that one in three children—and nearly half of Latino and African American children—born in the year 2000 will develop type 2 diabetes in their lifetime (Narayan, 2003). Obesity also increases the risk of social discrimination and psychological problems such as depression and low self-esteem (French, 1995).

II. Trends in Physical Activity and Caloric Intake

Limited data suggest there have only been small changes in physical activity levels in recent decades

Compared to dietary data, relatively little data are available on changes over time in physical activity levels, making it difficult to determine if reductions in physical activity have corresponded with the rise in obesity rates that have been observed since the late 1970s. No single survey or data set has examined physical activity levels over the entire time period from the 1970s to the 2000s, so data must be compared from several studies that examine various smaller time periods, different subsets of the population, and different types of physical activity. This is complicated by the fact that it is difficult to compare and interpret findings across studies because of methodological differences in assessing physical activity.

Surveys conducted in California between 1985 and 1995 suggest that there have been small increases in the percentage of adults who engage in vigorous physical activity 3 times per week and little change in those who participate in moderate physical activity over the same time period (Governor's Council, 1995). According to the same surveys, there has been a decrease in the percent of adults who report no leisure-time physical activity. National data also indicate slight improvements over the time period from 1990 to 2000 in the percentage of adults (men and women) meeting recommended activity levels (Brownson, 2005). More recent data suggests that leisure-time physical activity among adults continues to increase (Kruger, 2007). Similar trends are observed for youth. Surveys of children and adolescents suggest that there was no significant change or improvement in vigorous or moderate physical activity between 1993 and 2005 (Stanford Prevention Research Center, 2007).

Livingstone (2003), however, points out that most studies with good trend data have looked only at leisure-time physical activity. While limitations exist in the data available, indicators are that work-related physical activity has decreased (even in manual occupations) in recent decades.

Livingstone suggests that the observed increases in leisure-time physical activity may not be enough to offset the decline in work-related physical activity.

Other indirect indicators of physical activity levels suggest that physical activity levels have declined in recent decades. These include: time participating in PE classes, sports teams and outdoor recreation; changes in active transport (walking and biking) to school or work; employment in high vs. low physical activity occupations; average daily vehicle miles traveled per person; proportion of trips to work by walking; and changes in the walkability of neighborhoods (Brownson, 2005; Stanford Prevention Research Center, 2007). For example, the National Personal Transportation Survey data showed that in 1969 walking or biking was the most common means of transport to school for 5-18 year olds (used by 41% of children). By 2001 only 13% of children used active means of transport – most children traveling to school by car or bus (McDonald, 2007). Therefore, although trend data for overall physical activity levels suggest minimal change, specific types of physical activity have declined as obesity rates have risen. Conversely, time spent in sedentary activities such as TV viewing, video gaming, and computer use have increased dramatically since the 1960s (Gortmaker, 1990; Rideout, 2005) and may contribute to obesity through a variety of mechanisms (Hamilton, 2008).

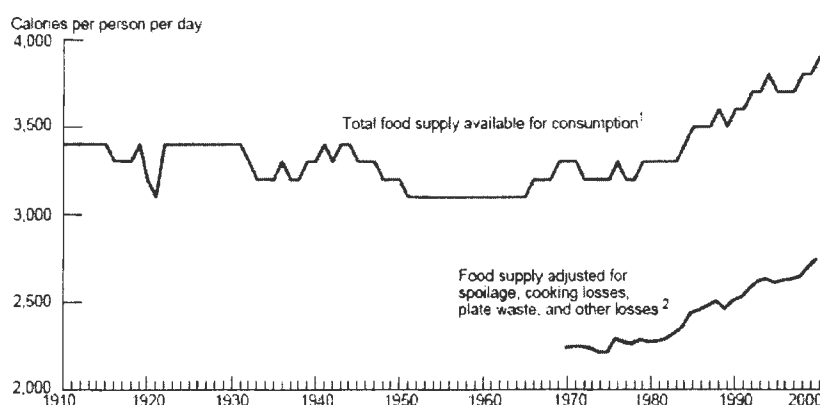
A recently released report found that adults (ages 18-50) in the U.S. and Europe burn about the same number of calories on physical activity as they did 20 years ago (Westerterp, 2008). This meta-analysis of 13 studies involving nearly 400 subjects included only investigations that used doubly labeled water, an accurate technique that objectively measures individuals' energy expenditure as they go about their daily lives. This analysis showed that adults have not reduced their energy expenditure over the same time period during which obesity rates have increased dramatically. However, the subjects used in these studies may not have been representative of the general population in terms of activity habits or weight changes. Further, it is possible that very small changes in daily physical activity expenditure – that over time could lead to measurable changes in weight – have occurred that were not detectable even by this objective method.

Although the numbers vary according to the recommendation used, self-reported physical activity levels for a majority of Americans are below what is recommended (CDC, 2008). When physical activity is measured by objective means, the picture is even more dismal. In a recent analysis of nationally representative data (NHANES 2003-2004) in which physical activity was measured by accelerometer, only 42% of children aged 6-11 years old and 8% of adolescents obtained the recommended 60 minutes/day of physical activity (Troiano, 2008). Among adults, less than 5% obtained a minimum of 30 minutes/day of physical activity. In contrast, approximately 55% of waking time was spent in sedentary activities (e.g., sitting) (Matthews, 2008).

Given the limited data available, it is not possible to arrive at a firm conclusion regarding changes in physical activity levels since the 1970s. However, inadequate levels of physical activity when combined with dramatic increases in the energy supply (described below) have likely left the population susceptible to the development of obesity. This susceptibility is due in part because an individual's energy homeostasis relies upon physiological signals for hunger that are very efficient compared to the signals for satiety that are relatively inefficient (Prentice, 2004).

Increases in calorie intake parallel the rise in obesity

In contrast to the lack of data regarding changes in physical activity, information from three national data sets suggest that there have been substantial changes in caloric intake. Data about trends in the national food supply (USDA) and data from two nationally representative surveys of food intake show that calorie intake has increased steadily since the late 1970s (Figures 9 and 10). The increases in calorie availability and intake correspond remarkably to the rise in obesity over the same time period. Between the late 1970s and the year 2000 the amount of energy available for human consumption in the U.S. increased by more than 500 calories per day per person, even after adjusting for estimated spoilage, cooking, plate waste and other losses (USDA/ERS, 2003).⁶ The two national surveys of food intake suggest a more modest, but nevertheless substantial, increase over the same time period of nearly 300 calories per person (all ages combined) per day (Nielsen, 2004).



¹ Rounded to the nearest hundred. ² Not calculated for years before 1970.

Figure 9. Calories from the U.S. Per Capita Food Supply, Adjusted for Losses. (Source: USDA/ERS(a), 2003.)

⁶ This food supply data includes all food that enters the domestic marketing system intended for distribution or sale for human consumption in the U.S. It includes imported foods and excludes exports and inventories.

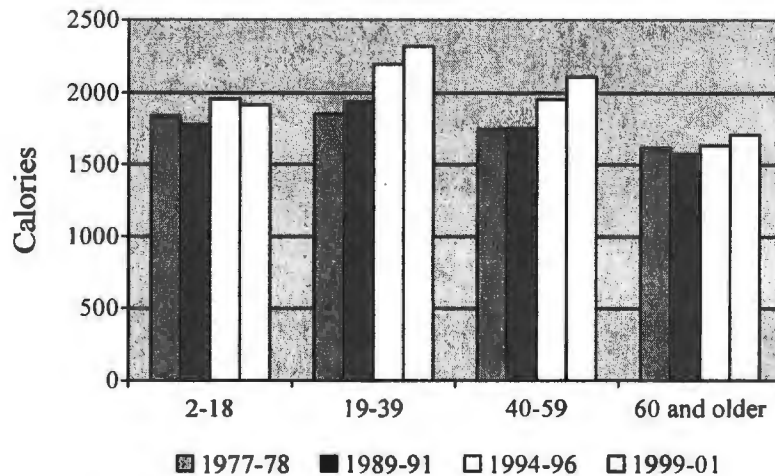


Figure 10. Trends in Total Energy Intake for Different Age Groups (NFCS⁷ 1977-78; CSFII⁸ 1989-91, 1996; NHANES 1999-01). (Source: Nielsen and Popkin, 2004.)

Similar trends in energy intake have been observed in other countries. Bleich et al. (2008) used data derived from developed countries on trends in obesity, food supply, and levels of physical and sedentary activity and two different analytical approaches (one relying on country-level data and a second on individual-level data) to estimate the relative contribution of increased caloric intake and reduced physical activity to obesity. In all countries, increases in caloric supply paralleled obesity trends. Regardless of approach, increased energy intake was found to be primarily responsible for the observed gains in weight.

III. Trends in Sweetened Beverage Consumption

Sweetened beverage intake and the prevalence of obesity have risen in parallel over the same time period

Between 1970 and 2000 the weight of added sugars in the food supply increased by about 20% per capita (Figure 11). In 1994-1996 Americans obtained 46% of their added sugars from sweetened beverages, one-third from soft drinks alone (Johnson, 2001; Guthrie, 2000). Between 1977 and 2002 Americans on average (all ages) increased their calorie intake from soft drinks by 228% (i.e., more than doubled) and their calorie intake from fruit drinks by 171% (i.e., nearly doubled) (Duffey, 2007). According to national survey data collected in 1999-2001, Americans on average (all ages) consumed 9% of their daily calories from sweetened beverages (Jacobson, 2005). Children aged 2-18 years obtained 10% of their calories from sweetened beverages and

⁷ Nationwide Food Consumption Survey

⁸ Continuing Survey of Food Intake by Individuals

teenagers obtained 13% of their calories from these drinks. Sweetened beverage intakes have continued to rise in recent years. For example, by 1999-2004, adults (ages 20-44) consumed 12% of their total daily intake from sweetened beverages, up from 9% just a few years earlier (Bleich, 2009). Figures 12-14 illustrate the increased in soft drink production and intake in the U.S. over the time period that obesity rates have most dramatically risen.

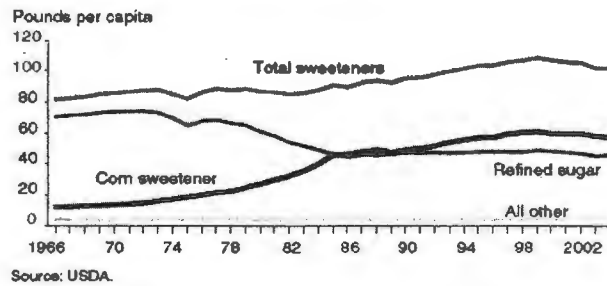


Figure 11. Estimated Per Capita Sweetener Consumption, Total and By Type of Sweetener, 1966-2004. (Source: USDA, 2005.)

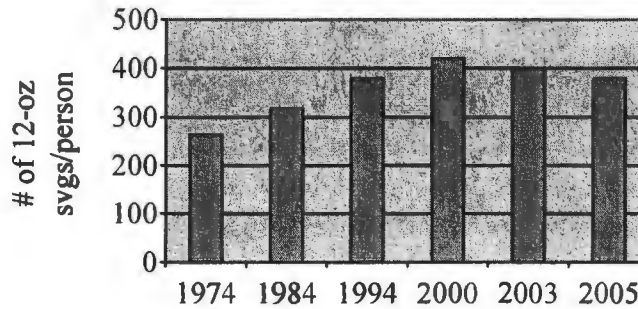


Figure 12. Trends in Annual Soft Drink Production in the U.S. (Sources: USDA/ERS(b), 2003 and USDA/ERS, 2008).

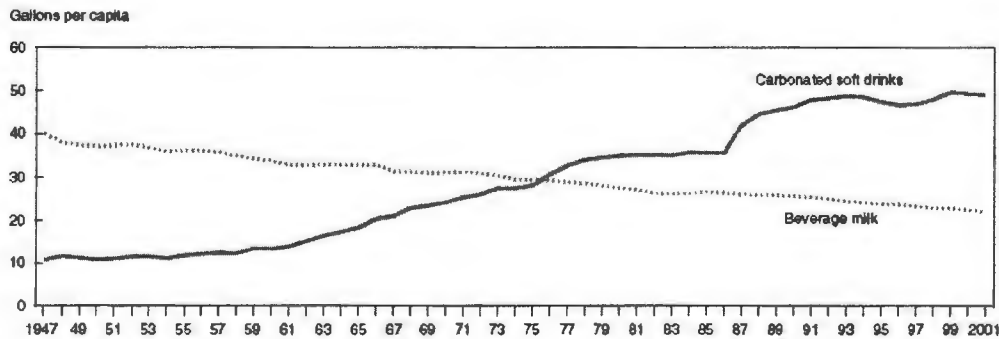


Figure 13. Per Capita Carbonated Soft Drink and Milk Consumption in the U.S. (Source: USDA/ERS, 2004.)

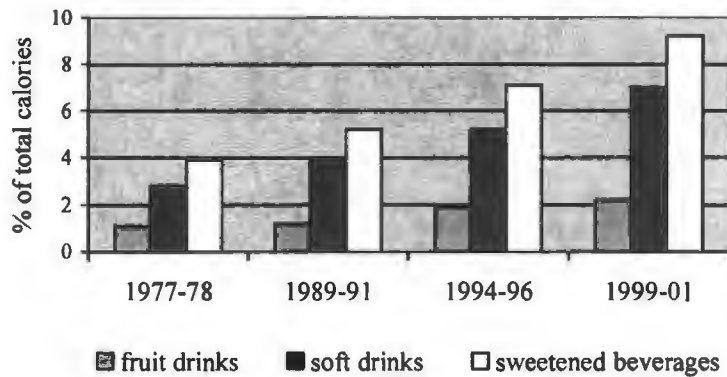


Figure 14. Trends in U.S. Beverage Consumption as Percent of Total Daily Calorie Intake (NFCS 1977-78; CSFII 1989-91, 1996; NHANES 1999-01). (Source: Nielsen and Popkin, 2004.)

Portion sizes have also increased from an average serving size of 6.5 fl oz (88 calories) in the 1950s, to 12 fl oz (160 calories) and 20 fl oz (266 calories), and even larger portion sizes common today (Jacobson, 2005; Nielsen, 2003). The average serving of soft drink in fast food restaurants in 2002 was 23 fl oz (299 calories), with some chains now commonly selling soft drinks in 32 (416 calories) to 64 fl oz containers (832 calories) (Young, 2003). According to national survey data, in 1988-94 the average portion size reported by adults who consumed sweetened beverages on one or more occasion was 11 fl oz; average portion size increased to 17 fl oz by 1999-2004 (Bleich, 2009).

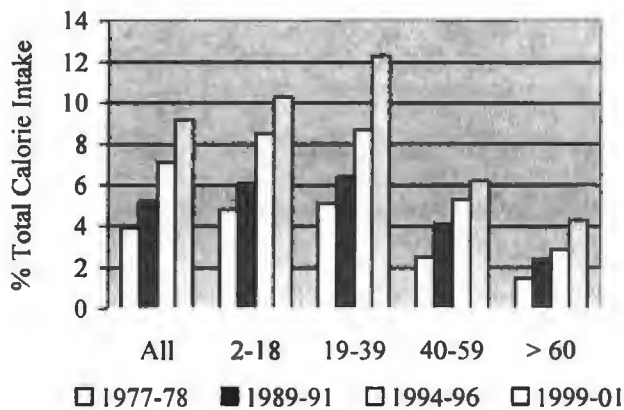


Figure 15. Trends in U.S. Sweetened Beverage Consumption as Percent of Total Daily Calorie Intake (NFCS 1977-78; CSFII 1989-91, 1996; NHANES 1999-01). (Source: Nielsen and Popkin, 2004.)

All age and gender groups have increased their intake of sweetened beverages.

Among children aged 2-18, the percent of calories consumed from sweetened beverages more than doubled between 1977 and 2001 (Nielsen, 2004) (Figure 15). Increases were even greater among young adults. Adults, age 19-39 consumed most calories from sweetened beverages (12% of total calories) followed by 2-18 year olds (10% of total calories) and adults 40-59 years old (6% of total calories) (Nielsen, 2004).

Lytle et al. (2000) reported that, as students moved from elementary to middle school, the proportion of beverages consumed as soft drinks more than tripled, while milk and 100% fruit juice consumption declined substantially. Data collected between 1999 and 2004 revealed that boys 13-18 years of age consumed an average of 357 calories per day in the form of sweetened beverages. Excluding those who consumed no sweetened beverages, the mean intake was 409 calories per day. Girls of the same age consumed somewhat less: an average of 242 calories per day from sweetened beverages among all girls and 300 calories per day, excluding those who consumed no sweetened beverages (Jacobson, 2005).

According to the USDA, a moderately active 16-year-old boy (or girl) who eats the recommended servings from all foods groups would have only 425 (or 265 calories for a girl) discretionary fat and sugar calories (USDA, 2008). Therefore when an “average amount” of sweetened beverages are consumed in addition to the foods recommended by the dietary guidelines, no other discretionary snacks or sweets or high fat foods can be consumed without the daily calories exceeding current recommendations. In about half of the population, youth are consuming substantially more than the mean intake recommended; in those cases daily calories will potentially greatly exceed recommendations in the absence of any other snacks or sweets. These findings suggest a critical role of sweetened beverages in contributing to excess energy intake and/or displacing foods with essential nutrients.

Ethnic minorities and low-income individuals consume more sweetened beverages than non-Hispanic Whites and those with higher incomes

Findings from a nationally representative survey conducted in 1988-94 (NHANES III) indicate that among adolescents, Black youth consume the most soda (regular, not-diet), followed by Hispanics; non-Hispanic Whites consume the least (Woodward-Lopez, 2006) (Figure 16). Adolescent non-Hispanic Whites, however were the largest consumers of diet soda. Between 1988-1994 and 1999-2004, there were significant increases in per capita consumption of sweetened beverages among black and Mexican American children, further widening the gap between ethnic groups (Wang, 2008).

Among the middle-school aged students studied by Giammattei et al. (2003), Latinos consumed more soft drinks than non-Hispanic Whites (1.6 vs. 1.1 soft drinks per day), while Asian students consumed the least (0.7 soft drinks per day). In this study, soft drinks included both diet and regular varieties. According to an analysis of NHANES data from 1988-94 and 1999-2002, teens from poor families got more of their calories from sweetened beverages and experienced a greater increase in the intake of sweetened beverages than those not living in poverty (Miech,

2006). More recent data (Wang, 2008) suggest that the differences in intake among youth by socioeconomic status have narrowed due to increases in intake among all socioeconomic groups.

Among adults, data from NHANES (1999-2004) revealed that on the surveyed day more blacks (76%) and Mexican Americans (70%) consumed sweetened beverages than whites (60%) and per capita calories consumed as sweetened beverage followed similar trends (calories/day were 234, 192 and 205 for blacks, Mexican Americans and whites) (Bleich, 2009). Higher intakes were also observed for individuals with lower incomes and less education. In summary, ethnic minorities and the poor, who have the highest risk of obesity, also consume the most sweetened beverages.

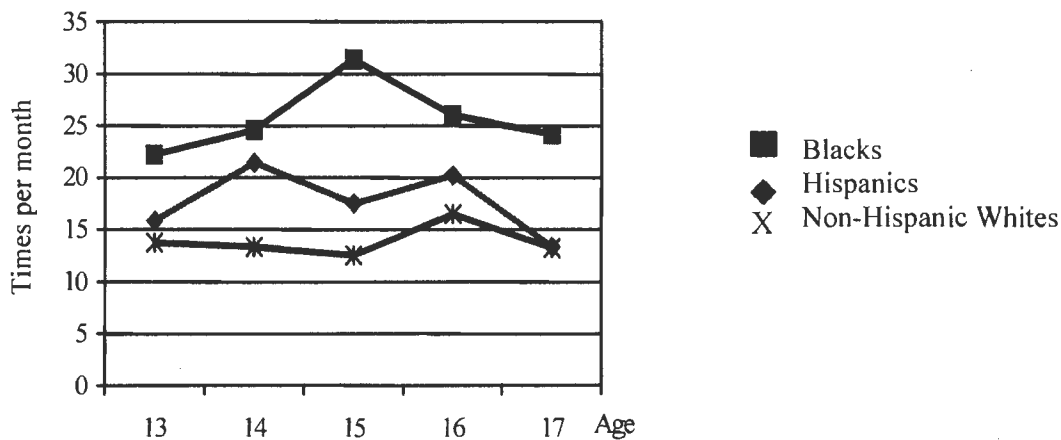


Figure 16. Consumption of Regular (Non-Diet) Soda by Black, Hispanic and Non-Hispanic White Adolescents, 13-17 Years Old (NHANES III).

Even preschoolers are increasing their intake of sweetened beverages at the expense of healthier options

Very young children also consume sweetened beverages. The 1994-1996 CSFII ⁹ reported that 53% of 2-5 year olds consumed soft drinks at least once in two days. The 1999-2004 NHANES reported that 70% of 2-5 year olds consumed at least one sweetened beverage on the survey day (Wang, 2008). In one study as many as one-third of 15-18 month old children consumed sweetened beverages on the day prior to the survey (Fox, 2004). Average portion size for 2-5 year old children is 7.2 fl oz per serving (Smiciklas-Wright, 2002). In a 4-year longitudinal study of 2-6 year olds, intake of carbonated and other sweetened beverages increased significantly while intake of 100% fruit juice declined (Skinner, 2001).

⁹ Continuing Survey of Food Intake by Individuals

Although sodas account for the greatest proportion of sweetened beverage intake, the intake of other sweetened beverages is on the rise

According to industry reports the U.S. liquid refreshment beverage market grew by only 2.9 % in 2006 (Beverage World, 2007). Although carbonated soft drinks still have the largest share of the market, with the average consumer drinking 50.4 gallons in 2006, per capita consumption has been on the decline in recent years (Beverage World, 2007) (Figures 12 and 17). At the same time, however, there has been a marked increase in the per capita consumption of other sweetened beverages. According to Beverage World, sports drinks are fourth in terms of market share; per capita consumption has continued to increase steadily since 1993 with the average consumer drinking 4.1 gallons in 2005. Energy drinks led the way in terms of growth, with 49.1% growth in 2006. Several other categories experienced double-digit growth in 2006 such as sports drinks (11.7%), ready to drink coffee (10.4 %) and ready-to-drink tea (26.2%). Per capita ready-to-drink tea consumption was 2.54 gallons in 2003. Fruit drink consumption has also been on the decline from 2004-2006, but holds onto third place in terms of market share. Bottled water was second in terms of market share in 2006 (Beverage Digest, 2008). Intake data also indicate that Nevertheless, recent NHANES data comparing reported intakes between 1988-94 and 1999-2004, has shown that the relative contribution of soda and fruit drinks to total sweetened beverage intakes has continued to increase across all age groups of adults (Bleich, 2009). For example, soda contributed 56% of all sweetened beverage calories in 1988-94 and increased to 61% by 1999-2004. The picture is a bit different among youth who are consuming increasing proportions of other sweetened beverages such as sports drinks and fruits drinks. Although soda is still the primary source of sweetened beverage calories among adolescents, younger children get the majority of these calories from fruits drinks (Wang, 2008).

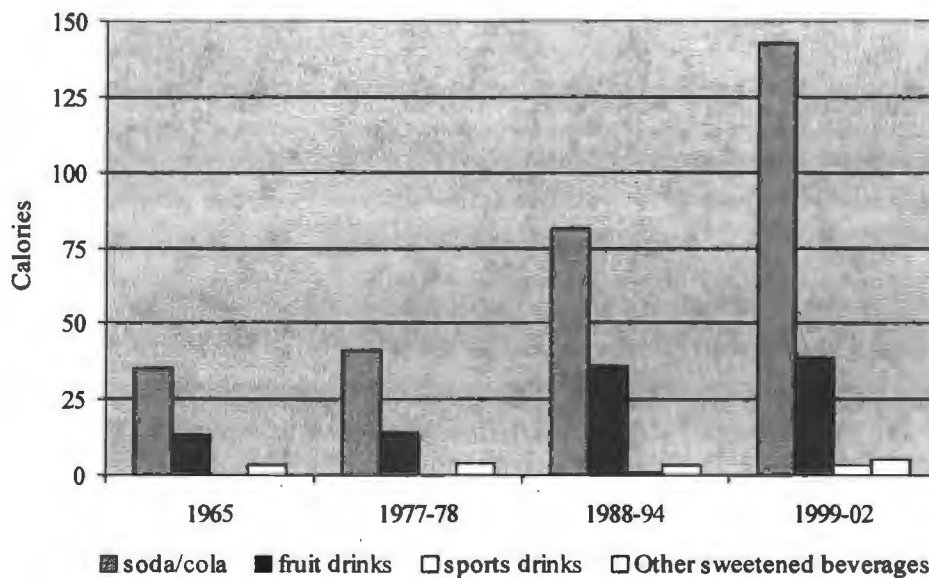


Figure 17. Per Capita Calorie Consumption by U.S. Adults of Selected Sweetened Beverages (NFCS 1965, 1977-78; NHANES 1988-94, 1999-02). (Source: Duffey and Popkin, 2007.)

Most added sugar is consumed at home and most sweetened beverages are purchased at grocery stores, followed by those consumed at restaurants and fast food establishments

According to a large national survey (NFCS¹⁰ and CSFII), by far the largest proportion of calories from sugar are consumed at home (Popkin, 2003) (Figure 18). According to national data on purchasing trends (1994-1995 CSFII), among females 12-19 years of age, 55% of sodas consumed were purchased from grocery stores, 25% from fast food and other restaurants, and 9% from school cafeterias and vending machines (Bowman, 2002). Data from the 1999-2004 NHANES revealed that for children (2-19 years old) on a weekday, 60% to 80% of calories from sweetened beverages and fruit juice were consumed at home (Wang, 2008). The home environment was also the major site of sweetened beverage consumption by adults, ranging from approximately half of all sweetened beverage calories for young adults to three-fourths for adults 65 years and above (Bleich, 2009).

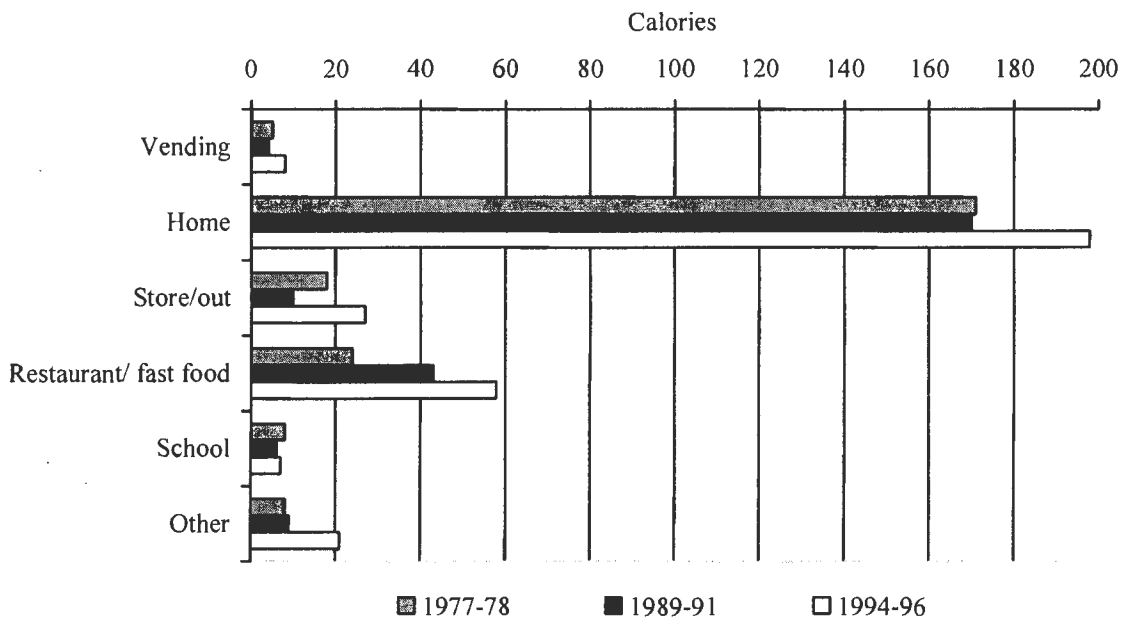


Figure 18. Trends in Calories from Added Sugars by Location for Americans ≥ 2 Years Old (NFCS 1977-78, CSFII 1989-91, 1994-96). (Source: Popkin and Nielsen, 2003.)

Conclusion: Trends in the availability and consumption of sweetened beverages suggest they have contributed to increases in calorie intake and obesity.

Although parallel trends cannot establish causation, the correspondence between the rapid rise in obesity and a similarly rapid increase in sweetened beverage intake and the increase in calorie intake over the same time period point to sweetened beverages as a likely contributor to the obesity epidemic. All ethnic groups and age groups including the very young have experienced

¹⁰ Nationwide Food Consumption Survey

increasing levels of sweetened beverage consumption. Of particular concern is the higher intake of sweetened beverages by those experiencing the highest rates of obesity. Although high intakes of added sugar in all forms is a nutritional concern, intake of sugar in liquid form accounts for almost half the total added sugar intake. Further, sweetened beverages displace healthier, more nutrient-dense beverages like milk, 100% fruit juice, and water.

IV. Mechanisms Which Explain the Relationship between Sweetened Beverages and Excess Weight Gain

Calories in liquid form are not well regulated and therefore may lead to weight gain

Our bodies can adjust for day-to-day fluctuations in energy intake and energy expenditure. For example, if we eat an unusually large meal one day, at subsequent meals we typically feel less hungry and don't eat as much. However, over time, this compensation may not be 100% complete, as suggested by unintentional fluctuations in body weight. Although evidence is not unanimous (Almiron-Roig, 2003), a large body of research suggests that we may not be able to self-regulate "liquid calories"—energy consumed in beverage forms—as well as calories from solid foods. Mattes (1996) estimated from over 40 studies primarily involving adults that, on average, 64% of an energy challenge—an additional source of food energy—as a solid is offset by subsequent dietary adjustment, while little of a liquid challenge (~9%) is compensated for by later changes in energy intake. Poor compensation for energy provided in fluid form has been documented testing a variety of caloric beverages (e.g., coffee, alcohol, soda, fruit juice, milk), with carbohydrate, fat, protein or energy density of solids and liquids matched. Compensation appears to vary by viscosity, with clear liquid sources of calories having a lower satiety value than more viscous fluids (Mattes, 2006). Incomplete compensation for calories from beverages have been documented primarily in short-term studies (ranging from several hours to days in the studies involving beverages in the Mattes (1996) analysis), but also during longer, more nutritionally relevant time periods (Tournier, 1991; Mourao, 2007).

Adding sweetened drinks to diets in studies of adults ranging from 3-10 weeks has been shown to result in increased ad libitum (i.e., self-selected) daily calorie consumption (Tordoff, 1990; DiMeglio, 2000; Raben, 2002b; Van Wymelbeke, 2004; Reid, 2007). For example, providing approximately 500 calories/day in the form of soda to normal weight adults who were otherwise free to consume foods and beverages of their choice, resulted in a 13% overall increase in total energy intake over a 3-week period (Tordoff, 1990). In a randomized, controlled 10-week trial involving overweight adults, those provided 800 calories/day, primarily in the form of soft drinks and fruit drinks, consumed three times more total calories than those given diet beverages and foods (Raben, 2002b). In a 4-week trial, women provided 430 calories/day in the form of sucrose sweetened drinks consumed an extra 190 total calories daily (Reid, 2007). A maximum compensation of ~50% was measured for the additional calories consumed as a sweetened beverage (see discussion on page 28 of randomized, controlled and crossover trials to increase sweetened beverage intake). In summary, in both short-term and longer-term trials, individuals appear to adjust their intakes of other foods only modestly to compensate for energy consumed in liquid form.

Multiple mechanisms explain why the intake of sugar in liquids is not well regulated

Although the mechanisms responsible for this incomplete compensation for liquid sources of calories have not been identified, several have been proposed (Mattes, 1996; DiMeglio, 2000). Beverages, because they typically are quickly consumed and rapidly absorbed by the gastrointestinal tract, may not stimulate satiety signals to the same extent as solids. Further there is evidence that fluids may not be as effective in stimulating insulin release, insulin being one of the physiologic signals for energy balance (Teff, 1994).

Poor regulation of calories from sweetened beverages has also been attributed to their high glycemic index (GI). GI is a measure of the degree to which blood glucose increases after intake of sugar or other carbohydrate (Bachman, 2006). However, a comprehensive review of the relationship between GI and energy intake, appetite, and weight found little support for GI as a factor in the etiology of obesity (Raben, 2002a).

The metabolic rate for burning calories may be acutely higher after ingestion of solid compared to liquid calories (Habas, 1998). Having evolved on a diet where water was the primary beverage, thirst and hunger mechanisms in humans may not be fully integrated; beverages may be consumed to satisfy thirst, even if in so doing more energy is consumed than required for energy balance. Finally, because sweetened beverages are rated as tasting good, they may be more likely to be consumed in excess than unsweetened beverages. For example, in one study preschoolers drank more sweetened chocolate milk than plain milk, resulting in increased energy intake during meals when sweetened milk was served (Wilson, 2000).

Human inability to fully compensate for calories from beverages may also have a cognitive basis; because beverages are typically not as calorie dense as solids, through experience we may not “learn” to compensate fully. Although sweetened beverages are classified according to the food guide pyramid as “discretionary calories,” it is not clear whether beverages such as soft drinks are treated as such by consumers. In the Forbidden Food Study, adding sugar to foods or beverages was reported to be a “forbidden” practice by nearly 30% of children. However, consumption of soft drinks was reported as “never or almost never allowed” by less than 10% of children (O’Dea, 1999). In national survey data of teens, soft drinks were as likely to be consumed at meals as at snack times (Guenther, 1986).

Evidence does not suggest that high fructose corn syrup plays a greater role than sucrose

The nature of the sweetener used in beverages may also be influential in weight gain. Sucrose, commonly known as table sugar, is composed of equal parts fructose and glucose. High fructose corn syrup (HFCS), the sweetener most commonly used in sweetened beverages, is also composed of fructose and glucose, but with a slightly higher percentage (55%) of fructose.

The consumption of HFCS has increased by 1000% in the past three decades, accounting for over 40% of the total added sugars and 132 calories per day in the typical American diet (Bray, 2004). Fructose is handled differently by the body than other simple sugars. For example, fructose stimulates less insulin secretion and leptin production (Teff, 2001), and increases fat synthesis (Tittelbach, 2000; Van Gaal, 1999). However, fructose is only one component of HFCS and the amount of fructose typically used in metabolic studies exceeds the amount normally ingested from products containing HFCS.

Despite the increase in use of HFCS, the overall fructose to glucose ratio in the U.S. diet has not changed appreciably (Malik, 2006). Two experimental studies in adults found that there was no difference between beverages sweetened with high fructose corn syrup or sucrose in terms of their short-term impact on satiety, appetite, or food intake (Akhavan, 2007; Soenen, 2007). A panel recently convened by the Center for Food Nutrition and Agriculture Policy, which is funded by the sugar and beverage industry, conducted a comprehensive review of the literature on HFCS and concluded that HFCS does not appear to contribute to obesity any differently than other sources of calories (Forshee, 2007). The relatively limited available evidence therefore suggests that sucrose and high fructose corn syrup (HFCS) play a similar physiologic role in the etiology of obesity. However other factors may be at play in the trend toward increased fructose intakes. For example, the relatively lower cost of HFCS and profitability of beverages made with HFCS may lead to more production and marketing of these beverages and therefore higher consumption on the part of consumers (Drewnowski, 2007).

Ever-increasing portion sizes, low prices and increased availability and marketing contribute to the increased consumption of sweetened beverages

The differential obesity-promoting capacity of sweetened beverages may also be attributable to their low price in addition to their liquid sugar or calorie contents, with soda and sweetened fruit drinks among the least expensive sources of calories in the marketplace (Drewnowski, 2007). For example 100% fruit and vegetable juices cost anywhere from 2-10 times more than soft drinks and fruit drinks (Drewnowski, 2007). Although the consumer price index for food rose from 100 in the early 1980s to 180 by 2002, not all foods increased by the same amount. For example, over this same time period the price index for fresh fruits and vegetables increased to 258, while it only increased to 126 for soft drinks, making them among the cheapest sources of calories (Sturm, 2005). Several studies have show that price reductions and subsidies influence consumer food and beverage purchases (French, 1997; French, 2003; French, 2005; Anderson, 2001; Herman, 2008; Johnson, 2004). Furthermore the lower cost beverages are most consistently associated with weight gain (Drewnowski, 2007).

The growing portion size of sweetened beverages available also contributes to increased consumption. Between 1977 and 1996, the portion size for the average soda nearly doubled: from 13 fl oz to nearly 20 fl oz, an increase of about 50 calories per serving (Nielsen, 2003). In one study, a 50% increase in the beverage portion size resulted in a 10-25% increase in caloric intake by adults (Flood, 2006).

Sweetened beverages are heavily promoted on television, websites, games and product placement agreements, and such marketing has been increasing over the time period in which consumption and obesity have increased. For example in the year 2000 the soft drink industry spent over \$700 million on advertising alone, up from \$381 million in 1986 (Beverage Digest, 1998; Jacobson 2005; Gallo, 1999). These figures do not include other types of marketing and promotional activities such as couponing or sponsorship of events and professional organizations. According to TNS Media Intelligence, in 2007 the top three soft drink companies actually reduced their spending on domestic advertising, down \$185 million to \$608.5 million (Abramson, 2008). However since other types of marketing such as promotions, email, handing

out samples, etc. are not included in this figure, it is not clear whether total marketing expenditures are down.

Sweetened beverages displace foods that may protect against excess weight gain

It is possible that consumption of soda and other sweetened beverages may affect weight status by displacing nutrients critical for proper regulation of energy balance. Specifically, dietary calcium has been related to adiposity; individuals with lower calcium and dairy intakes tend to have a greater risk of overweight (Woodward-Lopez, 2006). As soft drink consumption has increased population-wide, milk intake has decreased (Figure 13). The odds of an individual child (Harnack, 1999) or adult (Guthrie, 1996) consuming adequate milk and calcium decreases significantly as his/her soda consumption increases (Harnack, 1999). A recent meta-analysis combining the results of over 20 studies of adults and children confirmed that individuals who drink large amounts of sweetened beverages (including soda and others) tend to have low intakes of milk and calcium (Vartanian, 2007).

Sweetened beverage intake is consistently associated with higher energy intake

Of 23 food groups soft drinks are the largest contributor of energy to the diet and the top source of liquid calories in the U.S. (USDA, 1997; Troiano, 2000; Block, 2004). High consumption of sweetened beverages is consistently associated with higher total energy consumption (Guenther, 1986; Harnack, 1999; Cullen, 2002; Vartanian, 2007).

Adolescents drinking an average of 8 fl oz or more of soda (non-diet) daily consumed almost 200 calories more total energy every day than those drinking other types of beverages (Harnack, 1999). Children who consumed the largest amounts of sweetened drinks took in 330 daily calories more than children with the lowest intake of sweetened beverages (Cullen, 2002). Finally, in a recent meta-analysis including 21 studies (cross-sectional, longitudinal and experimental), all but two cross-sectional studies found that increased sweetened beverage intake was associated with increased daily energy consumption (Vartanian, 2007). As discussed previously, short-term trials involving supplementation with sweetened beverages to subjects otherwise free to choose the foods and beverages they consume have also demonstrated increases in total energy intake. Therefore studies using a variety of designs consistently show that consumption of sweetened beverages is related to higher overall energy consumption. These increases in energy intake without an increase in energy expenditure will inevitably result in weight gain.

Conclusion: Evidence suggests multiple mechanisms by which sweetened beverages can lead to excess calorie intake

In summary, several lines of evidence suggest mechanisms whereby sweetened beverages can lead to excess calorie intake. One of the most compelling is that by which calories consumed in the form of sweetened beverages are not completely compensated for by subsequent decreases in intake (Bachman, 2006). In the absence of dietary compensation or an increase in energy expenditure, an increase in energy intake inevitably results in weight gain.

V. Observational Studies that Examine the Relationship between Sweetened Beverage Intake and Adiposity

(see Appendix A for a more detailed discussion of the findings)

Observational studies examine the relationship between sweetened beverage intake and body adiposity among free living populations in the absence of an intervention. Longitudinal studies are considered more conclusive because they follow people over time and therefore can determine whether the sweetened beverage intake preceded the change in body weight. Nationally representative studies of the U.S. population are given more weight than other cross-sectional studies because their findings are generalizable. Further, these studies employ rigorous, standardized data collection methodologies.

Numerous, well-designed observational studies have found statistically significant positive relationships between sweetened beverage intake and adiposity

Thirty-seven observational studies were identified that examined the association between some type of sweetened beverage¹¹ intake and adiposity¹² (Appendix B, Tables 2-4). Sixteen of these were longitudinal (Appendix B, Table 2), that is, they followed the same group of people over time to see if those with higher sweetened beverage intake gained more weight. Over half (56%) of the longitudinal studies reported a statistically significant association between one or more categories of sweetened beverage intake and a measure of adiposity (Appendix B, Table 2). Those studies that found a significant positive association were more likely to receive higher quality ratings¹³ than those that did not. Six of the nine longitudinal studies that found such an association received the highest quality rating compared to only one of the six studies that did not detect a significant association.

Twenty-one cross-sectional studies (Appendix B, Tables 3-4) were identified that examined the association between some type of sweetened beverage intake and adiposity at one point in time. Seven of the 21 cross-sectional studies included subjects from nationally representative samples

¹¹ For the purposes of this paper when speaking in general terms “sweetened beverages” refers to any combination of beverages that contains an added caloric sweetener. However individual studies and data sets referred to in this paper used variable definitions, most commonly as follows: “sweetened beverages” - any beverage with added caloric sweetener and most commonly includes fruit-flavored drinks and sodas and sometimes includes sweetened teas and coffees; “soft drinks or sodas” - calorically sweetened carbonated beverages (sometimes includes diet drinks); “fruit drinks” - non-carbonated fruit-flavored drinks with added caloric sweetener including “ades” and punches. Please see appendices for definitions that were available for individual studies. In many cases no definition was provided.

¹² Adiposity outcomes variables were defined in different ways in these studies. Some studies conducted several analyses using different outcome variables. BMI was most commonly used as either a continuous or categorical variable. DXA, BIA, or skinfold measures were used in some studies. The measure used and definition of the outcome variable for each study are described in Tables 2-4.

¹³ Each article was abstracted and given a quality score of plus, neutral or minus using the American Dietetic Association’s Quality Criteria Checklist Abstraction. This abstraction process is based on the Agency for Healthcare Research and Quality domains for research studies (West, 2002). The checklist includes four questions about relevance of the results to the central question of the review and ten questions about the validity of the results using information about the design of the study, methods of data collection and analysis.

(Appendix B, Table 3). Two of the five nationally representative studies of children found statistically significant associations between sweetened beverage intake and adiposity. Both of those studies received the highest quality rating, whereas only one of the three that did not find a significant association received the highest quality rating. Two of the studies that did not detect a significant association were conducted by the same author and were funded by the beverage industry (Forshee, 2003; Forshee, 2004). Only two of the nationally representative studies included adults. One found a significant positive association between sweetened beverage intake and the other did not. Both received a neutral quality rating. Therefore although slightly less than half of the nationally representative cross-sectional studies detected a significant positive association, these studies were more likely to have higher quality ratings.

Twelve of the other cross-sectional observational studies examined the relationship between some type of sweetened beverage intake and adiposity among children (Appendix B, Table 4). Eight of the 12 found a statistically significant positive association between sweetened beverage intake and some measure of adiposity (Nicklas, 2003; Ochoa, 2007; Giammattei, 2003; Gillis, 2003; Berkey, 2004; Rockett, 2001; Sanigorski, 2007; Ariza, 2004). Four of the seven studies that found a significant positive association between adiposity and sweetened beverage intake among children, received the highest quality rating, whereas only one of the five that did not detect a significant positive association received a plus rating. Thus, more studies found a significant association and these were of higher quality than those that did not.

Two additional cross sectional studies included adults (Liebman, 2003; French 1994). Both found positive associations between sweetened beverage intake and adiposity. Both studies received a neutral quality rating.

It is notable that none of the observational studies (longitudinal or cross sectional) found a significant *negative* association between adiposity and any type of sweetened beverage intake suggesting that the significant positive associations that were observed were not due to random effects.

Conclusion: The majority of the observational studies found a significant association between sweetened beverage intake and adiposity.

Taken as a whole, the majority of the observational studies, including the more conclusive longitudinal studies, and those of more rigorous design in every category (cross sectional and longitudinal), found significant positive associations between sweetened beverage intake and adiposity. None of the studies found a significant negative association between any type of sweetened beverage intake and adiposity among any age or ethnic group. These findings therefore provide consistent evidence that sweetened beverage intake contributes to excess weight gain. The evidence supporting this association was stronger for children than for adults, due in part to the fact that there were fewer observational studies involving adults.

Limited evidence supports an association between sweetened beverage intake and adiposity among all age, ethnic and gender groups that were studied, however evidence was strongest for females and older compared to younger children.

Data were limited with regard to gender, age and ethnic group differences, but findings from the studies available include:

- Evidence supporting a link between sweetened beverage intake and adiposity among racially and ethnically diverse populations in the U.S. (Striegel-Moore, 2006; Warner, 2006; Ludwig, 2001; Giammattei, 2003);
- Stronger evidence for the association between sweetened beverage intake and adiposity among females than among males (Striegel-Moore, 2006; Phillips, 2004; Schulze, 2004; Rockett, 2001; French, 1994);
- Stronger evidence for the association between sweetened beverage intake and adiposity among school-age than preschool-age children, due in part to the relatively smaller number of studies among the preschool age group (Welsh, 2005; Newby, 2004; Warner, 2006; Sugimori, 2004; Dubois, 2007; LaRowe, 2007; O'Connor, 2006).

Numerous well-designed studies have found significant positive associations between adiposity and both soft drink and total sweetened beverage intake. Findings are more limited with regard to fruit drinks.

Data were limited with regard to the association between different types of sweetened drinks¹⁴ and adiposity. The evidence was strongest for the association of higher body weights with total sweetened beverage intake (Welsh, 2005; Ludwig, 2001; Dubois, 2007; Lin, 2004; Nicklas, 2003; Ochoa, 2007; Gillis, 2003; Berkey, 2004; Rockett, 2001; Gibson, 2007; Ariza, 2004; Liebman, 2003) and soft drink intake (Striegel-Moore, 2006; Warner, 2006; Phillips, 2004; Tam, 2006; Schulze, 2004; Bes-Rastrollo, 2006; Troiano, 2000; LaRowe, 2007; Giammattei, 2003; Sanigorski, 2007; Liebman, 2003; French, 1994), primarily because these were the most commonly studied categories of beverages. Among adults (Schulze, 2004; Liebman, 2003) and children (Welsh, 2005; LaRowe, 2007; Gillis, 2003; Sanigorski, 2007) a few studies also supported an association with fruit drink intake, but studies were fewer and the results were more mixed for this category of beverage. Few studies addressed other types of sweetened beverages.

VI. Intervention Trials That Examine the Impact of Changes in Sweetened Beverage on Body Weight

Four controlled trials in children (two randomized) successfully reduced sweetened beverage intake and observed trends toward lower adiposity among some groups

Four controlled trials (**Appendix C**) were identified that evaluated the relationship between sweetened beverage consumption and body weight (Beech, 2003; Teufel, 1998; Ebbeling 2006; James, 2004). In all studies the subjects were children between 7-18 years of age. All were successful in reducing sweetened beverage intake compared to the control groups and the two randomized, controlled trials found significant decreases in at least one measure of body weight

¹⁴ "Sweetened beverages" refers to any beverage with added caloric sweetener and most commonly includes fruit-flavored drinks and sodas and sometimes includes sweetened teas and coffees. "Soft drinks" refer to calorically sweetened carbonated beverages and "fruit drinks" refer to non-carbonated fruit-flavored drinks with added caloric sweetener including "ades" and punches.

among subjects compared to controls (Ebbeling, 2006; James, 2004). The two other studies observed non-significant trends in the same direction.

Two studies with the stronger designs found statistically significant reductions in adiposity or BMI whereas the two studies of weaker design (non-randomized) found that the trends toward lower adiposity were not significant. Two comprehensive multi-component studies, the Zuni Diabetes Prevention Program and the Girls' Health Enrichment Multi-site Study (GEMS), included beverage consumption as one component of their intervention. Neither study found a relation between their intervention and BMI change, but in neither case did they examine the independent effect of the change in consumption of sweetened beverages. Furthermore the Zuni study lacked a true control group and the findings from Native American adolescents are not generalizable to other population groups. In the case of the GEMS study, limitations in sample size and length of intervention may have contributed to the lack of significant findings. Therefore the lack of significant findings from these two studies is neither surprising nor definitive.

The two studies that found significant decreases in body weight were stronger in design. One (Ebbeling, 2006) was a home-based intervention in Massachusetts directed toward 13 to 18 year old girls and boys that involved counseling and weekly deliveries of non-caloric beverages. Overall, subjects experienced lower increases in BMI relative to controls, but this difference was not significant. However, among the heaviest subjects (upper tertile of baseline BMI) there was a statistically significant reduction in BMI. The subjects reduced their BMI by 0.63 points whereas the control group increased their BMI by 0.12. The Christchurch Obesity Prevention Project in Schools (CHOPPS) was a school-based education program that focused exclusively on discouraging the consumption of carbonated beverages (James, 2004). Four educational sessions were delivered over a 1-year period to students ages 7-11 years. Findings showed a statistically significant reduction in the percent of children that stayed overweight in the intervention group as compared to an increase in the percent of children that became overweight in the control group.

Four randomized, controlled or crossover trials in adults showed that increasing sweetened beverage intake resulted in weight gain

In one cross-over study, 30 normal weight adults were successively provided, over successive 3-week periods, either artificially sweetened soda, regular soda (sweetened with high-fructose corn syrup) or no soda supplementation (Tordoff, 1990). The regular soda provided 530 calories/day (the equivalent of 3-4 12-fl-oz cans each day). Subjects were not told which type of soda they were given. When consuming regular soda, subjects increased their calorie intake by 13% and gained an average of 2.6 pounds, gains that were significant compared to the no soda or artificial soda conditions. Another cross-over trial tested adjustments in intake over 4-week periods of supplementation with 449 calories/day in either a solid (jelly beans) or liquid (soda) form (DiMeglio, 2000). Weight significantly increased when energy was provided in the liquid compared to the solid form. Compensation for the additional calories provided as a liquid averaged only 17%.

Several randomized, controlled trials have compared the impact of sucrose versus artificially sweetened drinks on body weight. One such trial of 41 overweight adults followed for 10 weeks

provided either sucrose (800 calories/day) or artificial sweeteners primarily in the form of soft drinks and fruit drinks (70-80% by weight) (Raben, 2002b). The sucrose group consumed 3 times more total calories than those given diet beverages and after 10 weeks gained 3.5 pounds while the artificial sweetener group lost 2.2 pounds – a significant difference between the groups of over half a pound a week. In a 4-week trial of 133 women involving sucrose vs. artificially sweetened beverages, women provided 430 calories/day in the form of sucrose sweetened drinks consumed an extra 190 total calories daily (Reid, 2007). In both of these studies, ad libitum compensation for the energy supplement averaged 56%; in other words, subjects decreased their usual intake by an amount equal to only half of the liquid calories they were given.

Conclusion: Intervention trials support the hypothesis that consumption of sweetened beverages can contribute to excess weight gain, and reducing sweetened beverages can help prevent excess weight gain.

Randomized, controlled intervention trials are generally required to establish a causal relationship; in this case, between sweetened beverage consumption and weight. All four of the randomized, controlled trials to decrease sweetened beverage intake that were reviewed, showed decreases in adiposity when sweetened beverage intake was reduced. The two studies with the strongest designs found statistically significant reductions in percent of overweight children or in BMI among the heaviest group of children. It is notable that that the studies consisted of non-representative samples of children of distinct age and ethnic groups and therefore the findings can not be generalized to adults or other non-similar groups of children. All four of the intervention trials in adults (two with crossover designs in which subjects served as their own controls, and two randomized, controlled studies) that aimed to increase consumption of sweetened beverages demonstrated significant weight gain over periods ranging from 3 to 10 weeks. These findings provide support for the hypothesis that sweetened beverage consumption can contribute to excess weight gain, and that interventions to reduce sweetened beverage consumption can lead to a measurable reduction in BMI.

VII. Meta-Analyses That Combine Results from Studies That Vary in Design

Two meta-analyses have been recently published that examine the relationship of sweetened beverage intake on body weight (Vartanian, 2007; Forshee, 2008). Results of a group of studies are combined in a meta-analysis in order to improve statistical power. Vartanian et al. (2007) combined studies in both adults and children, including cross-sectional and longitudinal observational studies as well as intervention trials. Because results from different studies used different measures of adiposity, the findings were presented using a standardized measure of effect size. Based on 33 studies, a significant effect size of 0.08 was estimated (0.03 from 22 studies involving children; 0.11 from 11 studies involving adults). An effect size is a statistical technique used to quantify the strength of a relationship between two variables, in this case, between sweetened beverage consumption and a measure of adiposity. Larger effect sizes were observed for intervention trials, for studies involving adults, for studies focusing on soft drinks (as opposed to other sweetened beverages), and for studies not funded by the food industry. Based on calculated associations of sweetened beverage intake with increased energy intake,

decreased intake of milk, calcium and other nutrients, and increased body weight and health problems such as type 2 diabetes, the authors concluded that recommendations to reduce soft drink consumption were clearly supported by science.

The second meta-analysis by Forshee et al. (2008) included only longitudinal and intervention studies (total of 10) involving children or adolescents. The maximum estimated effect size of 0.02 (expressed in units of change in BMI during the time period defined by the studies for each daily serving change in sweetened beverage consumption) was not statistically different from zero. Given the variable length of studies—ranging from 3 months to 10 years—it is not possible to translate this effect size into anticipated weight gain. The authors concluded that reducing consumption of sweetened beverages would not have a large impact on BMI among youth. Of note, this meta-analysis was performed by authors who received funding from the American Beverage Association. As such, the results of this meta-analysis must be considered with caution. However, the meta-analysis by Vartanian et al. (2007) also found that the effect size in studies of children was smaller than for adults. It is possible that children are better than adults at compensating for extra calories consumed in liquid form. To our knowledge, this has not been tested in energy challenge trials; very few such trials have included children (Mattes, 1996). It is also possible that other differences between children and adults (e.g., variations in accuracy of dietary recall, variations in rate of growth) make it more difficult to detect an effect of sweetened beverages on weight change in children.

VIII. To What Extent Have Sweetened Beverages Contributed to the Increase in Calorie Intake Since the Late 1970s?

By examining the increase in per capita calorie intake that occurred over recent decades during which obesity rates have risen, an estimate of the percent of the increase in calories which came from sweetened beverages can be calculated. Given that it is unlikely that physical activity levels have increased substantially in recent decades (see pages 10-11), these additional calories were likely to have been in excess of average energy needs and therefore contributed to excess weight gain in the U.S. population. For this examination, data presently available from the largest nationally representative surveys were used – the 1977-78 NFCS and the 1999-2001 NHANES. Limitations of these data include reliance upon self-reported intakes, subject to recall errors and other reporter biases, and the fact that probing methods used to collect dietary intakes were improved over the years. However, national food supply data could not be used for this purpose because the calories from sweetened beverages were only available beginning in the mid 1980s, after which time national obesity rates had already begun to rise. Further, although food supply data are adjusted for anticipated waste and spoilage and are invaluable for showing intake trends, food supply data has been shown to overestimate actual consumption (Bleich, 2008). Other dietary surveys have been used to collect food intake of individuals over time, but because they are not nationally representative and don't always cover the time period of interest, their use would limit generalizability.

Including individuals 2 years old and older, per capita calorie intake increased from 1790 calories in 1977-78 to 2068 calories in 1999-2001 (Nielsen, 2004), an increase of 278 calories per person (Table 5). Over the same time period calories from sweetened beverages (defined as soft drinks and fruit drinks) increased from 70 to 190 calories per person per day, an increase of 120 calories. Therefore the increase in these sweetened beverages is equivalent to 43% of the increase in calorie consumption.

Table 5. Proportion of increase in total calorie intake from sweetened beverages, 1977-2001

	1977-1978	1999-2001	Difference
Total calories/person/day	1,790	2,068	278
Calories/person/day from sweetened beverages	70	190	120
Percentage of increase in total calorie intake from sweetened beverages			43% (120/278)
<i>(Source: NFCS, CSFII, and NHANES as reported in Nielsen and Popkin, 2004)</i>			

IX. Conclusion: Does the Intake of Sweetened Beverages Increase the Risk of Overweight?

All four lines of evidence examined (secular trends, plausible mechanisms, observational studies and intervention trials) provide support for the hypothesis that the intake of sweetened beverages increases the risk of overweight (Table 6). The population-wide increase in the intake of sweetened beverages corresponds with the increases in calories and obesity observed in recent decades. Studies show that calorie intake from sugar in liquid form is not well compensated for by reductions in subsequent intake and that sweetened beverage intake is consistently associated with higher calorie intakes. The majority of observational studies, particularly those of more rigorous design and higher quality, have demonstrated that sweetened beverage intake is significantly associated with greater adiposity when examined both longitudinally and cross-sectionally. Most importantly, two randomized controlled trials demonstrated successful reduction of sweetened beverage intake which resulted in reductions in adiposity among children. Conversely, trials to increase intake of sweetened beverages have demonstrated weight gain among free-living adults. Research examining gender and ethnic differences in the relationship between sweetened beverage intake and adiposity are limited; however, the data suggest that sweetened beverage intake can increase the risk of overweight regardless of ethnicity, age or gender. Likewise, evidence was limited with regard to the impact of different categories of sweetened beverages, other than soda; but the findings suggest that all sweetened

beverages have potential to increase the risk for overweight. Soda, because it is the largest beverage source of calories in the American diet, has likely contributed more than other sweetened beverages to the obesity epidemic. In recognition of this growing body of evidence in support of the link between obesity and sweetened beverages, researchers and health professionals are beginning to make recommendations for dramatically reducing sweetened beverage consumption, particularly among children (Crawford, 2008).

Table 6. Summary of Evidence Supporting Contribution of Sweetened Beverages to Obesity in the U.S.

Type of Evidence	Major findings
Intake trends	<ul style="list-style-type: none"> • Sweetened beverage intake – whether assessed from food production or reported food intakes – has increased concurrent with obesity epidemic • All age, gender, race/ethnicity, and socioeconomic groups increased intakes, but largest increases in intakes by groups most affected by obesity • Sweetened beverages account for 43% of observed increase in population energy intake between 1977 and 2001
Mechanistic studies	<ul style="list-style-type: none"> • Humans fail to compensate completely for calories from liquid challenges • Sweetened beverage intake consistently associated with more daily calories consumed
Observational studies	<ul style="list-style-type: none"> • Numerous observational studies, including nationally representative samples and longitudinal designs, report significant positive association between sweetened beverage intake and adiposity • No observational studies found the inverse – that higher intake of sweetened beverages are related to less obesity
Intervention trials	<ul style="list-style-type: none"> • Randomized, controlled trials that successfully reduced sweetened beverage intake lowered adiposity among some groups • Randomized, controlled or crossover trials showed that increasing sweetened beverage intake results in weight gain

Acknowledgements

The authors wish to acknowledge the contribution of Sarah Kim who abstracted and rated the observational studies referred to in this report. We also gratefully acknowledge the following national panel of experts for their review and input of this analysis:

- George Bray, MD, Boyd Professor, Pennington Center, Louisiana State University
- Patricia Crawford, DrPH, Co-director, University of California, Berkeley, Dr. Robert C. and Veronica Atkins Center for Weight and Health
- Steven Hooker PhD, Director, Prevention Research Center, University of South Carolina
- Richard Mattes, MPH, PhD, RD, Professor, Foods and Nutrition, Purdue University
- Barry Popkin, PhD, Professor, Department of Nutrition, Gillings School of Public Health, and Director, Interdisciplinary Obesity Center, University of North Carolina
- Jim Sallis, PhD, Professor Department of Psychology, San Diego State University and Director of The Robert Wood Johnson Foundation Active Living Research
- Marlene Schwartz, PhD, Deputy Director, Rudd Center for Food Policy and Obesity, Yale University
- Mary Story, PhD, Professor, Division of Epidemiology, School of Public Health, University of Minnesota and Director of The Robert Wood Johnson Foundation Active Living Research
- Y. Claire Wang, MD, Assistant Professor of Health Policy & Management, Mailman School of Public Health, Columbia University

References

- Abramson H. (2008). Consultant to Berkeley Media Studies Group, personal communication.
- Akhavan T, Anderson GH. (2007). Effects of glucose-to-fructose ratios in solutions on subjective satiety, food intake, and satiety hormones in young men. *Am J Clin Nutr*; 86(5): 1354-1363.
- Almiron-Roig E, Chen Y, Drewnowski A. (2003). Liquid calories and the failure of satiety: How good is the evidence? *Obes Rev*; 4: 201-212.
- Andersen LF, Lillegaard ITL, Overby N, Lytle L, Klepp K, Johansson L. (2005). Overweight and obesity among Norwegian schoolchildren: Changes from 1993-2000. *Scan J Public Health*; 33(2), 99-106.
- Anderson JV, et al. (2001). 5 A Day fruit and vegetable intervention improves consumption in a low income population. *J Am Diet Assoc*; 101(2), 195-202.
- Ariza AJ, Chen EH, Binns HJ, Christoffel KK. (2004). Risk factors for overweight in five to six year old Hispanic American children: A pilot study. *J Urban Health: Bull NY Acad Med*; 81(1), 150-161.
- Bachman CM, Baranowski T, Nicklas TA. (2006). Is there an association between sweetened beverages and adiposity? *Nutr Rev*; 64:153-174.
- Bandini LG, Vu D, Must A, Cyr H, Goldberg A, Dietz WH. (1999). Comparison of high-calorie, low-nutrient-dense food consumption among obese and non-obese adolescents. *Obes Res*; 7: 438-443.
- Baskin ML, Ard J, Franklin F, Allison DB. (2005). Prevalence of obesity in the United States. *Obes Rev*; 6: 5-7.
- Bates LM, Acevedo-Garcia D, Alegria M, Krieger N. (2008). Immigration and generational trends in body mass index and obesity in the United States: Results of the National Latino and Asian American Survey, 2002-2003. *Am J Public Health*; 98(1): 70-77.
- Beech BM, Klesges RC, Kumanyika SK, Murray DM, Klesges L, McClanahan B, Slawson D, Nunnally C, Rochon J, McLain-Allen, Pree-Cary J. (2003). Child- and parent-targeted interventions: the Memphis GEMS pilot study. *Ethn Dis*; 13: S40-S52.
- Berkey CS, Rockett HRH, Field AE, Gillman MW, Colditz GA. (2004). Sugar-added beverages and adolescent weight change. *Obes Res*; 12(5): 778-788.
- Bes-Rastrollo M, Sanchez-Villegas A, Gomez-Gracia E, Martinez JA, Pajares RM, Martinez-Gonzalez MA. (2006). Predictors of weight gain in a Mediterranean cohort: The Seguimiento Universidad de Navarra Study. *Am J Clin Nutr*; 83: 362-70.
- Beverage Digest (1998). *Fact Book 1998*. Bedford Hill, NY, pp. 54-55.
- Beverage Digest (2008). *Global Beverage Industry 2007*. June 6, 2008. Vol. 52, No. 13
- Beverage World (2007). New age report: health and wellness beverages continue to have a good prognosis; State of the Industry '07. April 15, 2007. Vol. 126, No. 4.
- Binkley J, Golub A. (2007). Comparison of grocery purchase patterns of diet soda buyers to those of regular soda buyers. *Appetite*; 49: 561-571.
- Bleich SN, Cutler D, Murray C, Adams A. (2008). Why is the developed world obese? *Annu Rev Public Health*; 29:273-295.
- Bleich SN, Wang CY, Wang Y, Gortmaker SL. (2009). Increasing consumption of sugar-sweetened beverages among US Adults: 1988-1994 to 1999-2004. *Am J Clin Nutr*; 89(1):372-381.

- Block G. (2004). Foods contributing to energy intake in the US: Data from NHANES III and NHANES 1999-2000. *J Food Comp Anal*; 17: 439-47.
- Blum JW, Jacobsen DJ, Donnelly JE. (2005). Beverage consumption patterns in elementary school aged children across a two-year period. *J Am College Nutr*; 24(2): 93-98.
- Bowman SA. (2002). Beverage choices of young females: changes and impact on nutrient intakes. *J Am Diet Assoc*; 102: 1234-1239.
- Bray GA, Nielsen SJ, Popkin BM. (2004). Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. *Am J Clin Nutr*; 79: 537-543.
- Brownson RC, Boehmer TK, Luke DA. (2005). Declining rates of physical activity in the United States: What are the contributors? *Annu Rev Public Health*; 26: 421-443.
- California Center for Public Health Advocacy. (2005). The growing epidemic: Child overweight rates on the rise in California assembly district. Accessed at <http://www.publichealthadvocacy.org/PDFs/FactSheetEpidemic.pdf>.
- California Department of Health Services. (2005). The Economic costs of Physical Inactivity, Obesity, and Overweight in California Adults: Health Care, Workers' Compensation, and Lost Productivity. Sacramento, CA: California Department of Health Services. Accessed at www.dhs.ca.gov/ps/cdic/cpns/press/downloads/GoctofObesityFullTechnicalReport.pdf.
- Centers for Disease Control and Prevention (CDC). Behavioral Risk Factor Surveillance System – Prevalence Data. Accessed at <http://apps.nccd.cdc.gov/brfss/>.
- Centers for Disease Control and Prevention (CDC). (2008). Prevalence of self-reported physically active adults – United States, 2007. *MMWR*; 57(48): 1297-1300.
- CMS (Children's Medical Services) (2008). California Dept. of Public Health Pediatric Nutrition Surveillance System (PedNSS) in California Data tables. Accessed Feb, 2008 at <http://www.dhs.ca.gov/pcfh/cms/onlinearchive/pdf/chdp/informationnotices/2004/chdpin04c/contents.htm>.
- Crawford PB, Woodward-Lopez G, Ritchie L, Webb K. (2008). How discretionary can we be with sweetened beverages for children? *J Am Diet Assoc*; 108 (9): 1440-4.
- Crawford, PB, Story, M, Wang, MC, Ritchie, LD, Sabry, ZI. (2001). Ethnic issues in the epidemiology of childhood obesity. *Child Adolesc Obes*; 48(4): 855-878.
- Cullen KW, Ash DM, Warneke C, de Moor C. (2002). Intake of soft drinks, fruit-flavored beverages, and fruits and vegetables by children in grades 4 through 6. *Am J Public Health*; 92: 1475-1477.
- DiMeglio DP, Mattes RD. (2000). Liquid versus solid carbohydrate: effects on food intake and body weight. *Int J Obes*; 24: 794-800.
- Drewnowski A, Bellisle F. (2007). Liquid calories, sugar, and body weight. *Am J Clin Nutr*; 85: 651-661.
- Dubois L, Farmer A, Girard M, Peterson K. (2007). Regular sugar-sweetened beverage consumption between meals increases risk of overweight among preschool-aged children. *J Am Diet Assoc*; 107: 924-934.
- Duffey KJ, Popkin BM. (2007). Shifts in patterns and consumption of beverages between 1965 and 2002. *Obesity*; 15(11): 2739-2747.

- Ebbeling CB, Feldman HA, Osganian SK, Chomitz VR, Ellenbogen SJ, Ludwig DS. (2006). Effects of decreasing sugar-sweetened beverage consumption on body weight in adolescents: A randomized, controlled pilot study. *Pediatrics*; 117(3): 673-680.
- Flegal KM, Carroll MD, Kuczmarski RJ, Johnson CL. (1998). Overweight and obesity in the United States: prevalence and trends, 1960-1994. *Int J Obes*; 22, 39-47.
- Flood JE, Roe LS, Rolls BJ. (2006). The effect of increased beverage portion size on energy intake at a meal. *J Am Diet Assoc*; 106: 1984-1990.
- Forshee RA, Anderson PA, Storey ML. (2004). The role of beverage consumption, physical activity, sedentary behavior, and demographics on body mass index of adolescents. *Int J Food Sci Nutr*; 55(6): 463-478.
- Forshee RA, Anderson PA, Storey ML. (2008). Sugar-sweetened beverages and body mass index in children and adolescents: a meta-analysis. *Am J Clin Nutr*; 87: 1662-1671.
- Forshee RA, Storey ML, Allison DB, Glinsmann WH, Hein GL, Lineback DR, Miller SA, Nicklas TA, Weaver GA, White JS. (2007). A critical examination of the evidence relating high fructose corn syrup and weight gain. *Crit Rev Food Sci Nutr*; 47: 561-582.
- Forshee RA, Storey ML. (2003). Total beverage consumption and beverage choices among children and adolescents. *Int J Food Sci Nutr*; 54: 297-307.
- Fox MK, Pac S, Devaney B, Janowski L. (2004). Feeding Infants and Toddlers Study: what foods are infants and toddlers eating? *J Am Diet Assoc*; 2004; 104:S31-S37.
- Freedman DS, Dietz WH, Srinivasan SR, Berenson GS. (1999). The relation of overweight to cardiovascular risk factors among children and adolescents: the Bogalusa heart Study. *Pediatrics*; 103(6 Pt 1): 1175-1182.
- French SA, Jeffery RW, Forster JL, McGovern PG, Kelder SH, Baxter, JE. (1994). Predictors of weight change over two years among a population of working adults: the Healthy Worker Project. *Int J Obes*; 18: 145-154.
- French SA, Story M, Jeffery RW, Snyder P, Eisenberg M, Sidebottom A, Murray D. (1997). Pricing strategy to promote fruit and vegetable purchase in high school cafeterias. *J Am Diet Assoc*; 97(9): 1008-1010.
- French SA, Story M, Perry CL. (1995). Self-esteem and obesity in children and adolescents: A literature review. *Obes Res*; 3: 479-480.
- French SA. (2003). Pricing effects on food choices. *J Nutr*; 133: 841S-843S.
- French SA. (2005). Public Health strategies for dietary change: schools and workplaces. *J Nutr*; 135: 910-912.
- Gallo AE. (1999). Food advertising in the United States. In: Frazao E, ed. *America's Eating Habits: Changes and Consequences*. Agriculture Information Bulletin, Number 750. Washington, DC: U.S. Department of Agriculture.
- Giammattei J, Blix G, Marshak HH, Wollitzer AO, Pettitt DJ. (2003). Television watching and soft drink consumption: associations with obesity in 11 to 13 year old schoolchildren. *Arch Ped Adolesc Med*; 157(9): 882-886.

- Gibson S, Neate D. (2007). Sugar intake, soft drink consumption and body weight among British children: Further analysis of National Diet and Nutrition Survey data with adjustment for under-reporting and physical activity. *Int J Food Sci Nutr*; 58(6): 445-460.
- Gillis LJ, Bar-Or O. (2003). Food away from home, sugar-sweetened drink consumption and juvenile obesity. *J Am College Nutr*; 22(6): 539-545.
- Gordon-Larsen P, Nelson MC, Beam K. (2005). Associations among active transportation, physical activity, and weight status in young adults. *Obes Res*; 13(5): 868-875.
- Gortmaker SL, Dietz WH Jr, Cheung LW. (1990). Inactivity, diet, and the fattening of America. *J Am Diet Assoc*; 90(9): 1247-1252, 1255.
- Governor's Council. (1995). Healthy California 2000 Physical Activity & Fitness Objectives 1995 Update.
- Guenther PM. (1986). Beverages in the diets of American teenagers. *J Am Diet Assoc*; 86: 493-499.
- Guthrie JF, Morton JF. (2000). Food sources of added sweeteners in the diets of Americans. *J Am Diet Assoc*; 100(1): 43-51.
- Guthrie JF. (1996). Dietary patterns and personal characteristics of women consuming recommended amounts of calcium. *Fam Econ Nutr Rev*; 9: 33-49.
- Habas ME, Macdonald IA. (1998). Metabolic and cardiovascular responses to liquid and solid test meals. *Br J Nutr*; 79: 241-247.
- Hamilton MT, Healy GN, Dunstan DW, Zderic TW, Owen N. (2008). Too little exercise and too much sitting: inactivity physiology and the need for new recommendations on sedentary behavior. *Curr Cardio Risk Rep*; 2: 292-298.
- Harnack L, Stang J, Story M. (1999). Soft drink consumption among U.S. children and adolescents: nutritional consequences. *J Am Diet Assoc*; 99: 436-441.
- Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegal KM. (2004). Prevalence of overweight and obesity among U.S. children, adolescents, and adults, 1999-2002. *JAMA*; 291(23): 2847-2850.
- Herman D. (2008). Low-income women purchase a variety of fruits and vegetables when provided with an economic incentive. *FASEB J*; 19(5s): A1697.
- Hill JO, Wyatt HR, Reed GW, Peters JC. (2003). Obesity and the environment: where do we go from here? *Science*; 299(7): 853-855.
- IOM (Institute of Medicine). (2005). Institute of Medicine Preventing Childhood Obesity: Health in the Balance, 2005. Accessed at www.iom.edu.
- IOM (Institute of Medicine). (2006). Food marketing to children and youth: Threat or opportunity? The National Academies Press.
- Jacobson MF. (2005). Liquid candy: how soft drinks are harming Americans' health. Accessed June 30, 2008 at <http://www.cspinet.org/liquidcandy/>.
- James J, Thomas P, Cavan D, Kerr D. (2004). Preventing childhood obesity by reducing consumption of carbonated drinks: Cluster randomized controlled trial. *BMJ*; 328: 1237

Johnson DB, Beaudoin S, Smith LT, Beresford SA, LoGerfo JP. (2004). Increasing fruit and vegetable intake in homebound elders: the Seattle senior farmers' market nutrition pilot program. *Prev Chronic Dis*; Jan;1(1): A03.

Johnson L, Mander AP, Jones LR, Emmett PM, Jebb SA. (2007). Is sugar-sweetened beverage consumption associated with increased fatness in children? *Nutrition*; 23: 557-563.

Johnson RK and Frary C. (2001). Choose beverages and foods to moderate your intake of sugars: The 2000 dietary guidelines for Americans--what's all the fuss about? *J Nutr*; 131(10): 2766S-2771S.

Kruger J, Kohl HW III, Miles IJ. (2007). Prevalence of regular physical activity among adults – United States, 2001 and 2005. *MMWR*; 56: 1209-1212.

Kvaavik E, Andersen LF, Klepp KI. (2004). The stability of soft drinks intake from adolescence to adult age and the association between long-term consumption of soft drinks and lifestyle factors and body weight. *Public Health Nutr*; 8(2): 149-157.

LaRowe TL, Moeller SM, Adams AK. (2007). Beverage Patterns, Diet Quality, and Body Mass Index of U.S. Preschool and School-Aged Children. *J Am Diet Assoc*; 107: 1124-1133.

Liebman, M, Pelican S, Moore SA, Holmes B, Wardlaw MK, Melcher LM, Liddil AC, Paul LC, Dunnagan T, Haynes GW. (2003). Dietary intake, eating behavior, and physical activity-related determinants of high body mass index in rural communities in Wyoming, Montana, and Idaho. *Int J Obes*; 27: 684-692.

Lin BH, Huang CL, French SA. (2004). Factors associated with women's and children's body mass indices by income status. *Int J Obes Relat Metab Disord*; 28(4): 536-42.

Livingstone MBE, Robson PJ, Wallace JMW, McKinley MC. (2003). How active are we? Levels of routine physical activity in children and adults. *Proc Nutr Society*; 62: 681-701.

Ludwig DS, Peterson KE, Gortmaker SL. (2001). Relation between consumption of sugar-sweetened drinks and childhood obesity. *Lancet*; 357: 505-508.

Lytle LA, Seifert S, Greenstein J, McGovern P. (2000). How do children's eating patterns and food choices change over time? Results from a cohort study. *Am J Health Promot*. Mar-Apr; 14(4): 222-228.

Malik VS, Schulze MB, Hu FB. (2006). Intake of sugar-sweetened beverages and weight gain: A systematic review. *Am J Clin Nutr*; 84: 274-288.

Mattes R. (2006). Fluid calories and energy balance: the good, the bad, and the uncertain. (2006). *Physiol Behav*; 89(1): 66-70.

Mattes RD. (1996). Dietary compensation by humans for supplemental energy provided as ethanol or carbohydrate in fluids. *Physiol Behav*; 59: 179-187.

Matthews CE, Chen KY, Freedson PS, Buchowski MS, Beech BM, Pate RR, Troiano RP. (2008). Amount of time spent in sedentary behaviors in the United States, 2003-2004. *Am J Epidemiol*; 167(7): 875-881.

McDonald NC. (2007). Active transportation to school: trends among U.S. schoolchildren, 1969-2001. *Am J Prev Med*; 32(6): 509-516.

Miech RA, Kumanyika SK, Stettler N, Link BG, Phelan JC, Chang VW. (2006). Trends in the association of poverty with overweight among U.S. adolescents 1971-2004. *JAMA*; 295(20): 2385-2393.

- Mourao DM, Bressan J, Campbell WW, Mattes RD. (2007). Effects of food form on appetite and energy intake in lean and obese young adults. *Int J Obes (Lond)*; 31: 1688-1695.
- Must A, Spandano J, Coakley EH, Field AE, Colditz GA, Dietz WH. (1999). The disease burden associated with overweight and obesity. *JAMA*; 282(16): 1523-1529.
- Narayan KMV, Boyle JP, Thompson TJ, Sorensen SW, Williamson DF. (2003). Lifetime risk for diabetes mellitus in the United States. *JAMA*; 290(14): 1884-1890.
- Newby PK, Peterson KE, Berkey CS, Leppert J, Wellett WC, Colditz GA. (2004). Beverage consumption is not associated with changes in weight and body mass index among low-income preschool children in North Dakota. *J Am Diet Assoc*; 104: 1086-1094.
- NHANES (National Health and Nutrition Examination Survey) III. Accessed at http://www.cdc.gov/nchs/products/elec_prods/subject/nhanes3.htm.
- Nicklas TA, Yang S, Baranowski T, Zakeri I, Berenson G. (2003). Eating patterns and obesity in children: The Bogalusa Heart Study. *Am J Prev Med*; 25(1): 9-16.
- Nielsen SJ, Popkin BM. (2003). Patterns and trends in food portion sizes, 1977-1998. *J Am Med Assoc*; 289: 450-453.
- Nielsen SJ, Popkin BM. (2004). Changes in beverage intake between 1977 and 2001. *Am J of Prev Med*; 27(3): 205-210.
- O'Connor TM, Yang S, Nicklas TA. (2006). Beverage intake among preschool children and its effect on weight status. *Pediatrics*; 118: e1010-e1018.
- O'Dea JA. (1999). Children and adolescents identify food concerns, forbidden foods, and food-related beliefs. *J Am Diet Assoc*; 99: 970-973.
- Ochoa MC, Moreno-Aliaga MJ, Martinez-Gonzalez MA, Martinez JA, Marti A, GENOI Members. (2007). Predictor factors for childhood obesity in a Spanish case-control study. *Nutrition*; 23: 379-384.
- Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. (2006). Prevalence of overweight and obesity in the United States, 1999-2004. *JAMA*; 295(13): 1549-1555.
- Ogden CL, Carroll MD, Flegal KM. (2008). High body mass index for age among U.S. children and adolescents, 2003-2006. *JAMA*; 299(20): 2401-2405.
- Ogden CL, Flegal KM, Carroll MD, Johnson CL. (2002). Prevalence and trends in overweight among U.S. children and adolescents, 1999-2000. *JAMA*; 288(14): 1728-1732.
- Overby NC, Lillegaard ITL, Johansson L, Andersen LF. (2003). High intake of added sugar among Norwegian children and adolescents. *Public Health Nutr*; 7(2): 285-293.
- Patterson ML, Stern S, Crawford PB, MacMahon RP, Similo SL, Schreiber GB, Morrison JA, Waclawiw MA. (1997). Sociodemographic factors and obesity in preadolescent black and white girls: NHLBI's Growth and Health Study. *J Nat Med Assoc*; 89: 594-600.
- Phillips SM, Bandini LG, Naumova EN, Cyr H, Colclough S, Dietz WH, Must A. (2004). Energy-dense snack food intake in adolescence: Longitudinal relationship to weight and fatness. *Obes Res*; 12(3): 461-472.
- Popkin BM, Nielsen SJ. (2003). The sweetening of the world's diet. *Obes Res*; 11(11): 1325-1332.
- Prentice A, Jebb S. (2004). Energy intake/physical activity interactions in the homeostasis of body weight regulation. *Nutr Rev*; 62(7): S98-S104.

- Raben A, Vasilaras TH, Møller AC, Astrup A. (2002b). Sucrose compared with artificial sweeteners: Different effects on ad libitum food intake and body weight after 10 wk of supplementation in overweight subjects. *Am J Clin Nutr*; 76: 721-729.
- Raben A.(2002a). Should obese patients be counselled to follow a low-glycaemic index diet? No. *Obes Rev*; 3(4): 245-256.
- Reid M, Hammersley R, Hill AJ, Skidmore P. (2007). Long-term dietary compensation for added sugar: effects of supplementary sucrose drinks over a 4-week period. *Br J Nutr*; 97: 193-203.
- Rideout V, Roberts DE, Foehr UG. (2005). Generation M: Media in the lives of 8-18 year-olds. Kaiser Family Foundation.
- Rockett HRH, Berkey CS, Field AE, Colditz GA. (2001). Cross-sectional measurement of nutrient intake among adolescents in 1996. *Prev Med*; 33: 27-37.
- Rodríguez-Artalejo F, García EL, Gorgojo L, Garcés C, Royo MA, Martín Moreno JM, Benavente M, Macías A, De Oya M. (2003). On behalf of the investigators of the Four Provinces Study. Consumption of bakery products, sweetened soft drinks and yogurt among children aged 6-7 years: Association with nutrient intake and overall diet quality. *Br J Nutr*; 89: 419-429.
- Sanigorski AM, Bell AC, Swinburn BA. (2007). Association of key foods and beverages with obesity in Australian schoolchildren. *Public Health Nutr*; 10(2), 152-157.
- Schulze MB, Manson JE, Ludwig DS, Colditz GA, Stampfer MJ, Willett WC, Hu FB. (2004). *JAMA*; 292(8): 927-934.
- Sfiligoi E. (1998). Beverage market index 1998. *Beverage World*; 117: 52-60.
- Shimakawa T, Sorlie P, Carpenter MA, Dennis B, Tell GS, Watson R, Williams OD. (1994). Dietary intake patterns and sociodemographic factors in the atherosclerosis risk in communities study. ARIC Study Investigators. *Prev Med*; 23(6): 769-780.
- Skinner JD, Carruth BR. (2001). A longitudinal study of children's juice intake and growth: The juice controversy revisited. *J Am Diet Assoc*; 101: 432-437.
- Smiciklas-Wright H, Mitchell DC, Mickle SJ, Cook AJ, Goldman JD. (2002). Foods Commonly Eaten in the United States: Quantities Consumed Per Eating Occasion and in a Day, 1994-1996. U.S. Department of Agriculture NFS Report No. 967-5, pre-publication version.
- Soenen, S. and M. S. Westerterp-Plantenga (2007). No differences in satiety or energy intake after high-fructose corn syrup, sucrose, or milk preloads. *Am J Clin Nutr*; 86(6): 1586-1594.
- Stanford Prevention Research Center. (2007). Building "Generation Play:" Addressing the crisis of inactivity among America's children. Accessed June 30, 2008 at <http://www.playeveryday.org/StanfordReport.pdf>.
- Striegel-Moore RH, Thompson D, Affenito SG, Franko DL, Obarzanek E, Barton BA, Schreiber GB, Daniels SR, Schmidt M, Crawford PB. (2006). Correlates of beverage intake in adolescent girls: The National Heart, Lung, and Blood Institute Growth and health Study. *J Ped*; 183-187.
- Sturm R. (2005). Childhood obesity -- what we can learn from existing data on societal trends, part 2. *Prev Chronic Dis*; 2(2):A20.

- Sugimori H, Yoshida K, Imano T, Miyakawa M, Suka M, Sekine M, Yamagami T, Kagamimori S. (2004). Analysis of factors that influence body mass index from ages 3 to 6 years: a study based on the Toyama cohort study. *Ped Int*; 46: 302-310.
- Sun SZ, Empie MW. (2007). Lack of findings for the association between obesity risk and usual sugar-sweetened beverage consumption in adults – A primary analysis of databases of CSF II 1989-1991, CSFII 1994-1998, NHANES III, and combined NHANES 1999-2002. *Food & Chem Tox*; 45: 1523-1536.
- Tam CS, Garnett SP, Cowell CT, Campbell K, Cabrera G, Baur LA. (2006). Soft drink consumption and excess weight gain in Australian school students: Results from the Nepean study. *Int J Obes*; 30: 1091-1093
- Teff K, Elliott S, Townsend R, et al. (2001). Consuming high fructose meals reduces circulating insulin and leptin concentrations in women. *Diabetes*; 50 (suppl 2): A532.
- Teff K. (1994). Cephalic phase insulin release in humans: Mechanism and function. In: Fernstrom JD, Miller GD, eds. *Appetite and body weight regulation: Sugar, fat, and macronutrient substitutes*. Boca Raton, FL: CFC Press, Inc.; 37-50.
- Teufel NI, Ritenbaugh CK. (1998). Development of a primary prevention program: Insight gained in the Zuni Diabetes Prevention Program. *Clin Pediatr*; 37: 131-142.
- Tittelbach TJ, Mattes RD, Gretebeck RJ. (2000). Post-exercise substrate utilization after a high glucose vs. high fructose meal during negative energy balance in the obese. *Obes Res*; 8: 496-505.
- TNS Media Intelligence Report From 1/1/06-12/31/07
- Tordoff MG, Alleva AM. (1990). Effect of drinking soda sweetened with aspartame or high-fructose corn syrup on food intake and body weight. *Am J Clin Nutr*; 51: 963-969.
- Tournier A, Louis-Sylvestre J. (1991). Effect of the physical state of a food on subsequent intake in human subjects. *Appetite*; 16: 17-24.
- Troiano RP, Berrigan D, Dodd KW, Mâsse LC, Tilert T, McDowell M. (2008). Physical activity in the United States measured by accelerometer. *Med Sci Sports Exerc*; 40(1): 181-188.
- Troiano RP, Briefel RR, Carroll MD, Bialostosky K. (2000). Energy and fat intakes of children and adolescents in the United States: Data from the National Health and Nutrition Examination Surveys. *Am J Clin Nutr*; 72(suppl): 1343S-53S.
- Troiano RP, Flegal KM. (1998). Overweight children and adolescents: Description, epidemiology and demographics. *Pediatrics*; 101: 497-503.
- U.S. Census Bureau: State and County quick facts (2008). Accessed at <http://quickfacts.census.gov/qfd/states/06000.html>.
- USDA (United States Department of Agriculture). MyPyramid.gov (2008). Accessed January 31, 2008 at <http://www.mypyramid.gov/mypyramid/index.aspx>.
- USDA/ARS (United States Department of Agriculture), Agricultural Research Service. Data tables: Results from USDA's 1994-96 Continuing Survey of Food Intakes by Individuals and 1994-96 Diet and Health Knowledge Survey. 1997. Accessed at <http://www.barc.usda.gov/bhnrc/foodsurvey/pdf/Csfii3yr.pdf>.

- USDA/ERS (a) (U.S. Department of Agriculture, Economic Research Service). Food Consumption per Capita Data System. Accessed May 2003 at <http://www.ers.usda.gov/data/food-consumption/DataSystem.asp?ERSTab=3>.
- USDA/ERS (b) (U.S. Department of Agriculture, Economic Research Service). ERS Food Availability Spreadsheets. Accessed May 2003 at <http://www.ers.usda.gov/data/FoodConsumption/FoodAvailSpreadsheets.htm>.
- USDA/ERS (U.S. Department of Agriculture, Economic Research Service). (April 2004). Indicators: In the long run. *Amber Waves*; 2(2): 48.
- USDA/ERS (U.S. Department of Agriculture, Economic Research Service). (2005). Sweetener Consumption in the U.S.: Distribution by Demographic and Product Characteristics. Outlook Report No. (SSS243-01).
- USDA/ERS (U.S. Department of Agriculture, Economic Research Service). Carbonated soft drinks: Per capita availability, 1984–2005. Accessed February 13, 2008 at <http://www.ers.usda.gov/Data/FoodConsumption/spreadsheets/beverage.xls>.
- Van Gaal L, Mertens I, Vansant G, De Leeuw I. (1999). Carbohydrate-induced thermogenesis in obese women. Effect of insulin and catecholamines. *J Endocrinol Invest*; 22: 109-114.
- Van Wymelbeke V, Béridot-Thérond ME, de La Guéronnière V, Fantino M. (2004). Influence of repeated consumption of beverages containing sucrose or intense sweeteners on food intake. *Eur J Clin Nutr*; 58: 154-161.
- Vartanian LR, Schwartz MB, Brownell KD. (2007). Effects of soft drink consumption on nutrition and health: A systematic review and meta-analysis. *Am J Public Health*; 97: 667-675.
- Wang Y, Gortmaker SL, Sobol AM, Kuntz KM. (2006). Estimating the energy gap among U.S. children: a counterfactual approach. *Pediatrics*; 118:e1721-e1733.
- Wang Y, Zhang Q. (2006). Are American children and adolescents of low socioeconomic status at increased risk of obesity? Changes in the association between overweight and family income between 1971 and 2002. *Am J Clin Nutr*; 84: 707-716.
- Wang YC, Bleich SN, Gortmaker SL. (2008). Increasing caloric contribution from sugar-sweetened beverages and 100% fruit juices among US children and adolescents, 1988-2004. *Pediatrics*; 121(6): e1604-1614.
- Warner ML, Harley K, Bradman A, Vargas G, Eskenazi B. (2006). Soda consumption and overweight status of 2-year-old Mexican-American children in California. *Obesity*; 14(11): 1966-1974.
- Welsh JA, Cogswell ME, Rogers S, Rockett H, Mei Z, Grummer-Strawn LM. (2005). Overweight among low-income preschool children associated with the consumption of sweet drinks: Missouri, 1999-2002. *Pediatrics*; 115: e223-e229.
- West S, King V, Carey TS, et al. (2002). Systems to Rate the Strength of Scientific Evidence. Evidence Report/Technology Assessment No. 47 (Prepared by the Research Triangle Institute-University of North Carolina Evidence-based Practice Center under Contract No. 290-97-0011). AHRQ Publication No. 02-E016. Rockville, MD: Agency for Healthcare Research and Quality.
- Westerterp KR, Speakman JR. (2008). Physical activity energy expenditure has not declined since the 1980s and matches energy expenditures of wild mammals. *Int J Obes*; 1-8.

Wilson JF. (2000). Lunch eating behavior of preschool children. Effects of age, gender, and type of beverage served. *Physiol Behav*; 70: 27-33.

Woodward-Lopez G, Ritchie L, Gerstein D, Crawford P. (2006). *Obesity: Dietary and Developmental Influences*. Boca Raton, FL: CRC Press.

Young LR, Nestle M. (2003). Expanding portion sizes in the U.S. marketplace: implications for nutrition counseling. *J Am Diet Assoc*; 103: 231-234.

Zhang Q, Wang Y. (2004). Trends in the association between obesity and socioeconomic status in U.S. adults: 1971-2000. *Obes Res*; 12(10): 1622-1632.

**THE ECONOMIC COSTS OF
OVERWEIGHT, OBESITY, AND
PHYSICAL INACTIVITY AMONG
CALIFORNIA ADULTS — 2006**

A study for the California Center for Public Health Advocacy

Conducted by Chenoweth & Associates, Inc.

New Bern, North Carolina | July 2009



www.PublicHealthAdvocacy.org

EXECUTIVE SUMMARY

Overweight, obesity, and physical inactivity are major risk factors for health conditions related to premature illness, disability, and death, and contribute significantly to the nation's rising medical care costs. In California in 2006, nearly 60% of adults were overweight or obese and almost half of California adults did not meet the recommended level and intensity of daily physical activity.

The California Center for Public Health Advocacy commissioned Chenoweth & Associates, Inc. to estimate the economic costs of overweight, obesity, and physical inactivity in the state of California and its counties. The results are based on an assessment of both health care costs and costs associated with lost productivity. The study also determined projected costs for overweight, obesity, and physical inactivity through 2011.

This study estimated the cost to California for overweight, obesity, and physical inactivity in 2006 to be \$41.2 billion. Of the total costs, \$21.0 billion was attributable to overweight and obesity and \$20.2 billion was attributable to physical inactivity. Half of the total amount was spent on health care and half came from lost productivity. If this trend continues, total costs for the state will increase to \$52.7 billion in 2011. Among California's counties, Los Angeles County, with its large population, accounted for more than one-quarter of all costs, followed by Orange and San Diego counties.

If the state of California is able to achieve a modest reduction in the prevalence of overweight, obesity, and physical inactivity of just 5% per year for each of these risk factors, the savings realized would average nearly \$2.4 billion per year.

Because employers and taxpayers share much of the burden of the economic costs associated with overweight, obesity, and physical inactivity, both the public and private sectors would benefit from the development and implementation of strategies that promote healthy eating and physical activity.

The estimated cost to California for overweight, obesity, and physical inactivity in 2006 was \$41.2 billion. If this trend continues, total costs for the state will increase to more than \$52.7 billion in 2011.

DEFINITIONS

Overweight:

Body mass index of 25.0–29.9

Obesity:

Body mass index of 30.0 or above

Physical Inactivity:

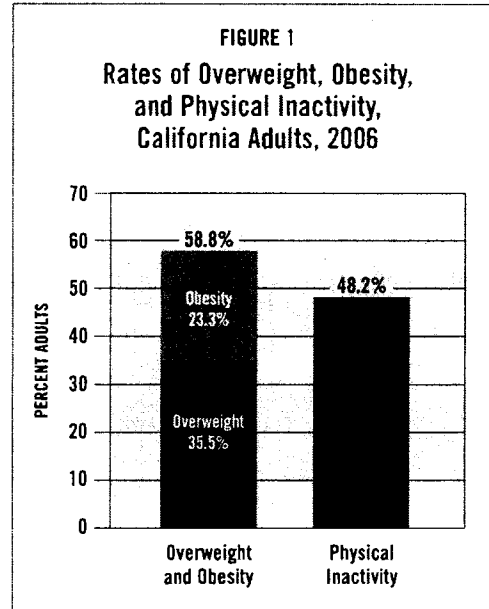
Engaging in less than 30 minutes of moderate physical activity on most days

SOURCE: Centers for Disease Control & Prevention

BACKGROUND

Overweight, obesity, and physical inactivity are major risk factors for many health conditions related to premature illness, disability, and death — among them, coronary heart disease, type 2 diabetes, some forms of cancer, and stroke¹⁻⁴ — and contribute significantly to the nation's rising medical care costs.⁵⁻¹²

In 2006, the Centers for Disease Control and Prevention (CDC) reported that a total of 58.8% of California adults were overweight or obese (35.5% and 23.3%, respectively).¹³ The two most recent CDC surveys reported a statewide adult physical inactivity rate for California of 46.6% in 2005 and 49.8% in 2007.¹⁴ A median prevalence rate of 48.2% was used in this study to estimate an approximate level of physical inactivity in 2006 (see Figure 1).



Overweight, obesity, and physical inactivity are major risk factors for many health conditions related to premature illness, disability, and death.

PURPOSE OF THE STUDY

The purpose of the study was to determine the current and future economic impact of overweight, obesity, and physical inactivity in the state of California. The last time such a study was published was in 2005 based on data for the year 2000.¹⁵ The current study also provides findings for California's counties. Economic costs at the county level were intended to allow local policy makers, business and community leaders, and community residents to know the economic effect of these three conditions in their geographic areas.

Specifically, the study sought to determine the following:

- Total medical care and prescription drug costs of medical conditions related to overweight, obesity, and physical inactivity for the state of California and its counties
- Lost productivity costs for each risk factor at the state and county level
- Future cost projections for each risk factor, assuming current prevalence and inflationary trends continue
- Projected cost savings for the state if even 5% of California adults who are currently overweight, obese, and/or physically inactive reduced their body weight or increased their physical activity to the recommended levels

Overweight, obesity, and physical inactivity have profound health and economic consequences.

METHODOLOGY

A statewide econometric analysis of costs related to overweight, obesity, and physical inactivity was conducted for California and its counties using health care and productivity data from several California and national databases. Health care cost estimates for each risk factor include direct medical care and prescription drug costs; lost productivity costs for each risk factor include costs associated with absenteeism, short term disability, and presenteeism (defined as the portion of an employee's work load they are unable to do because of their compromised health status). The aggregate cost of each of the three risk factors was calculated for each county and the entire state. Finally, medical care/prescription drug costs and lost productivity costs were projected for future years to estimate how these costs would change if the prevalence rates for the three risk factors continued at the current pace and what cost savings could be achieved if those risk factors were reduced even minimally.

Cost estimates assigned to each of the selected risk factors were based on conservative estimates of underlying factors. Thus, findings are likely to be conservative estimates as well. The Appendix provides a detailed description of the study methodology and limitations.

FINDINGS

Health Care and Lost Productivity Costs

The total estimated cost to California for overweight, obesity, and physical inactivity in 2006 was \$41.2 billion.

Of the total costs, \$21.0 billion was attributable to overweight

and obesity, and \$20.2 billion was attributable to physical inactivity. Half of the total amount was spent on health care (medical care and prescription drugs) and half came from lost productivity (see Table 1). Conditions stemming from overweight and obesity contributed \$12.8 billion (62%) to health care costs, while those related to physical inactivity accounted for \$7.9 billion (38%). Total lost productivity costs associated with overweight, obesity, and physical inactivity in California in 2006 were \$20.4 billion, including \$8.2 billion related to overweight and obesity (40%) and \$12.3 billion related to physical inactivity (60%) (see Figure 2).

Table 2 (on next page) presents the costs of health care and lost productivity for the three risk factors by county and for the state as a whole. Due to the size of their populations, Los Angeles, Orange, and San Diego counties accounted for nearly half of the state's total costs.

TABLE 1
Health Care and Lost Productivity Costs from Overweight, Obesity, and Physical Inactivity, California, 2006

	Overweight & Obesity	Physical Inactivity	TOTALS
Health Care Costs	\$12.8 billion	\$7.9 billion	\$20.7 billion
Lost Productivity Costs	\$8.2 billion	\$12.3 billion	\$20.4 billion
TOTALS	\$21.0 billion	\$20.2 billion	\$41.2 billion*

*Figures may not add to total due to rounding.

FIGURE 2: Percentage of Costs to California for Overweight, Obesity, and Physical Inactivity, 2006



TABLE 2
Economic Costs Associated with Overweight, Obesity, and
Physical Inactivity in California Counties, 2006

COUNTY	OVERWEIGHT & OBESITY		PHYSICAL INACTIVITY		TOTAL
	HEALTH CARE	LOST PRODUCTIVITY	HEALTH CARE	LOST PRODUCTIVITY	
Alameda	\$1,022,493,320	\$370,977,757	\$189,635,029	\$595,643,405	\$2,178,749,511
Butte	\$101,396,770	\$32,399,599	\$65,758,445	\$43,463,232	\$243,018,045
Contra Costa	\$404,221,810	\$272,232,863	\$255,603,709	\$386,509,777	\$1,318,568,159
El Dorado	\$59,641,096	\$31,626,939	\$39,983,414	\$44,781,471	\$176,032,920
Fresno	\$267,397,527	\$181,083,857	\$149,737,716	\$216,618,388	\$814,837,488
Humboldt	\$40,700,227	\$19,822,518	\$26,035,970	\$25,055,640	\$111,614,355
Imperial	\$56,344,348	\$27,113,157	\$31,538,647	\$29,852,954	\$144,849,106
Kern	\$281,023,090	\$153,339,517	\$172,825,417	\$199,394,032	\$806,582,056
Kings	\$42,523,486	\$28,055,537	\$25,821,065	\$32,069,645	\$128,469,732
Lake	\$36,298,603	\$9,101,561	\$21,502,216	\$11,119,542	\$78,021,922
Los Angeles	\$3,601,500,613	\$2,380,889,464	\$2,389,631,908	\$3,509,485,298	\$11,881,507,282
Madera	\$35,757,909	\$26,745,791	\$21,813,037	\$32,062,484	\$116,379,222
Marin	\$55,823,745	\$43,404,436	\$48,414,014	\$82,121,072	\$229,763,267
Mendocino	\$9,041,988	\$14,673,312	\$5,164,952	\$18,172,965	\$47,053,217
Merced	\$122,833,747	\$47,636,058	\$64,206,122	\$52,823,237	\$287,499,163
Monterey	\$186,716,905	\$110,934,183	\$109,920,445	\$126,813,230	\$534,384,763
Napa	\$63,033,157	\$29,541,415	\$42,867,363	\$42,794,998	\$178,236,933
Nevada	\$55,814,482	\$13,826,790	\$48,269,253	\$22,146,490	\$140,057,014
Orange	\$776,396,969	\$691,959,910	\$586,129,199	\$1,219,456,431	\$3,273,942,509
Placer	\$81,770,064	\$64,181,888	\$56,055,632	\$97,173,505	\$299,181,088
Riverside	\$443,401,567	\$345,544,640	\$370,674,371	\$459,833,591	\$1,619,454,168
Sacramento	\$558,107,329	\$363,575,032	\$301,772,622	\$437,819,850	\$1,661,274,834
San Bernardino	\$371,988,689	\$401,747,270	\$192,254,829	\$524,830,196	\$1,490,820,984
San Diego	\$817,945,377	\$647,077,040	\$577,254,569	\$999,779,198	\$3,042,056,184
San Francisco	\$244,703,445	\$193,072,957	\$225,528,252	\$423,071,502	\$1,086,376,156
San Joaquin	\$357,643,950	\$129,502,359	\$191,599,880	\$161,820,055	\$840,566,243
San Luis Obispo	\$179,805,931	\$44,329,042	\$168,087,338	\$61,456,910	\$453,679,220
San Mateo	\$351,116,006	\$216,493,810	\$223,291,405	\$361,466,707	\$1,152,367,927
Santa Barbara	\$133,523,535	\$89,644,429	\$82,771,771	\$128,916,568	\$434,856,303
Santa Clara	\$420,089,065	\$496,770,143	\$227,377,058	\$911,184,787	\$2,055,421,054
Santa Cruz	\$116,932,507	\$48,507,742	\$78,952,361	\$72,688,675	\$317,081,285
Shasta	\$111,090,845	\$30,900,455	\$69,350,965	\$41,393,440	\$252,735,705
Solano	\$158,429,455	\$97,507,493	\$97,239,872	\$129,336,401	\$482,513,221
Sonoma	\$114,668,973	\$84,373,927	\$90,816,010	\$146,866,048	\$436,724,958
Stanislaus	\$362,487,458	\$111,753,779	\$208,431,543	\$128,436,390	\$811,109,170
Sutter	\$32,084,565	\$14,578,464	\$19,343,231	\$17,654,708	\$83,660,969
Tulare	\$143,835,345	\$50,338,408	\$86,403,564	\$62,434,963	\$343,012,280
Ventura	\$287,718,588	\$154,743,132	\$204,090,472	\$222,866,813	\$869,419,005
Yolo	\$58,250,081	\$40,487,741	\$41,322,192	\$57,404,447	\$197,464,460
STATEWIDE	\$12,789,271,376	\$8,198,210,169	\$7,948,454,479	\$12,250,512,800	\$41,186,448,824

* Results for counties with populations less than 50,000 (Alpine, Amador, Calaveras, Colusa, Del Norte, Glenn, Inyo, Lassen, Mariposa, Modoc, Mono, Plumas, San Benito, Sierra, Siskiyou, Tehama, Trinity, Tuolumne, and Yuba) are not included in the table because county-specific risk factor data were not available. Costs from these counties were included in the statewide total.

If the state of California is able to achieve a modest reduction in the prevalence of overweight, obesity, and physical inactivity of just 5% per year, the cost savings to be realized would average nearly \$2.4 billion per year.

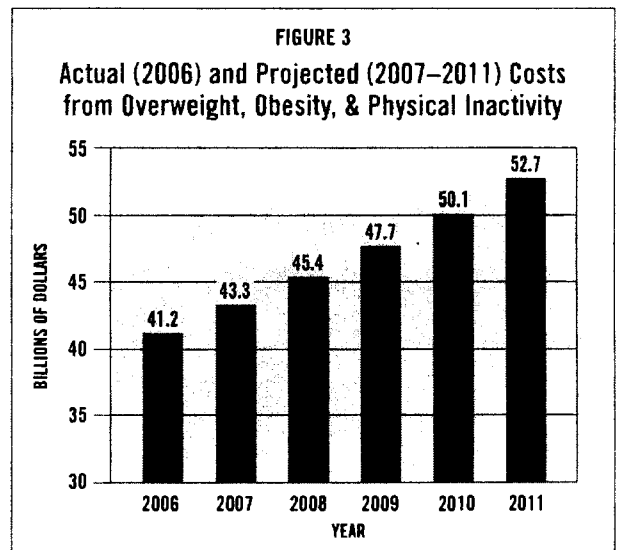
Projected Costs and Potential Cost Savings

The final phase of this analysis focused on the projected costs of overweight, obesity, and physical inactivity from 2007 through 2011 and the potential cost savings that could be achieved if the prevalence rates of these risk factors could be reduced.

Even if the prevalence rates remained constant, over time the economic costs associated with these risk factors would rise because of population growth and increased health care and employment costs.

Specifically, if California's population continues to rise at an expected rate of about 1% per year, medical care and prescription drug costs continue to rise at least 6% per year, and employment costs continue to rise at least 3% per year, then the combined health care and lost productivity costs associated with the three risk factors are conservatively estimated to increase to \$52.7 billion in 2011, or a cumulative five-year increase of 28% (see Figure 3).

If, however, the state of California is able to achieve a modest reduction in the prevalence of overweight, obesity, and physical inactivity of just 5% per year for each risk factor, the savings realized would average nearly \$2.4 billion per year.



DISCUSSION

Overweight, obesity, and physical inactivity have profound health consequences for the people of California. This analysis shows that the three risk factors — individually and collectively — also have profound economic consequences. California businesses, the backbone of the state's economy, are particularly affected. Because employers pay much of the cost of health care benefits, steady increases in health insurance premiums, in part due to increasing illness caused by poor diet and lack of physical activity, affect their bottom line, as does lost productivity resulting from these risk factors and their resulting illnesses. Taxpayers, too, have a huge financial stake in reversing these public health liabilities, as they pay for resulting illnesses through Medi-Cal and Medicare.

In order to reduce the unacceptably high prevalence of overweight, obesity, and physical inactivity, along with the costly and preventable illnesses associated with them, both the public and private sectors would benefit from promoting healthy eating and physical activity. While Californians must be encouraged to improve their individual behaviors, public policies must also be established to make it easier for Californians to adopt healthier lifestyles.

APPENDIX

Study Methodology

This econometric evaluation of costs related to overweight, obesity, and physical inactivity for California and its counties used available medical care and productivity data sources obtained from several California and national databases (see Table A-1).

Dollar year	Year 2006 dollars
Population	Statewide and 58 counties
Risk factors included	Overweight, obesity, and physical inactivity
Medical conditions included	Circulatory, digestive, injury, mental, metabolic, musculo-skeletal, neoplasm, nervous, pregnancy complications, and signs/symptoms ill-defined
State-level risk factor prevalence rates	Self-reported height and weight from the 2006 California Behavioral Risk Factor Surveillance Surveys (BRFSS); physical inactivity rates from the 2005 and 2007 BRFSS
County-level risk factor prevalence rates	Self-reported height and weight reported in the 2005 California Health Interview Survey (CHIS); self-reported physical inactivity rates reported in the 2001 California Health Interview Survey (CHIS)
Data source for inpatient medical costs: employer and private pay	2006 claims data from California's Office of Statewide Health Planning and Development (OSHPD) for 2006 by patient county residence and Diagnosis Related Group (DRG)
Data source for outpatient medical costs: employer and private pay	Estimated 2006 California corporate medical claims data (based on 2000 data from the authors) and 2006 claims data from OSHPD for ambulatory surgery and emergency department by patient county residence and Diagnosis Related Group (DRG)
Data source for outpatient medical costs: public pay (Medi-Cal)	Claims data from Medi-Cal for enrolled adults for the period of January 1, 2004 to December 31, 2004, projected to 2006 dollar values
Data source for prescription drug costs	Year 2006 cost norms from the 2007 Express Scripts Drug Trend Report and California prescription drug retail sales data from The Henry J. Kaiser Family Foundation
Lost productivity	Official Disability Guidelines injury frequency norms, 23 published studies, and California Employment Development Division average annual worker earnings

Overweight, Obesity, and Physical Inactivity Prevalence Rates

In order to estimate 2006 overweight and obesity prevalence rates, 2005 California Health Interview Survey (CHIS) results for height and weight for California counties were statistically adjusted to make them consistent with statewide-level Behavioral Risk Factor Surveillance Survey (BRFSS) findings for 2006.

The physical inactivity rates used in this study were based on the most recent available state and national health survey data. Because BRFSS did not collect physical inactivity prevalence rates in 2006, this study used the median between the statewide rates reported by BRFSS in 2005 and 2007. Because 2005 CHIS did not determine what proportion of Californians engage in less than 30 minutes of moderate physical activity on most days, this study utilized 2001 county-level CHIS

physical inactivity rates and statistically adjusted them to make them consistent with the estimated 2006 state-level physical inactivity rates from BRFSS.

Health Care Costs: Medical Care

Medical care costs were determined using health care claims data for California adults for medical conditions that have been shown in the published scientific literature as being directly linked to overweight, obesity, and physical inactivity. These conditions are represented by more than 100 diagnosis-related groups (DRGs) within the following ten major diagnostic categories: circulatory, digestive, injury, mental, metabolic, musculoskeletal and nervous conditions, some cancers, some pregnancy complications, and other signs and symptoms of an ill-defined nature (see Table A-2).

TABLE A-2 Medical Conditions Associated with Targeted Risk Factors—Diagnosis-Related Groups		
<p>Circulatory (DRGs: 014-017, 103-112, 120-145)</p> <p>Cardiovascular disease Myocardial infarction Hypertension Deep vein thrombosis Chronic venous insufficiency Stroke Atherosclerosis Coronary atherosclerosis Angina pectoris Congestive heart failure</p> <p>Digestive (DRGs: 179, 193-198, 203-204, 207-208, 316-317)</p> <p>Gallbladder disease Liver disease End stage renal disease Acute/chronic pancreatitis</p> <p>Injury (DRGs: 418, 452-453)</p> <p>Infection following wounds Heat disorders Surgical complications Hip fracture</p>	<p>Mental (DRGs: 426-427)</p> <p>Neurotic depression* Depressive disorder Anxiety states <i>* Excludes brief depressive reactive and prolonged depressive reaction</i></p> <p>Metab/ Endo/ Nutrition (DRGs: 294-295, 488-490)</p> <p>Diabetes Gout Impaired immune response</p> <p>Musculo-Skeletal (DRGs: 237, 241-246, 243, 248)</p> <p>Osteoarthritis knee or hip Rheumatoid arthritis Low back pain Low back strain/sprain Tendon/myo/bursitis Pain in joint Stiffness in joint Polymyalgia/rheum. Osteoporosis</p>	<p>Neoplasms (Cancers) (DRGs: 149-149, 152, 154-156, 203, 290, 274-275, 306-307, 318-319, 354-359, 401-404)</p> <p>Esophageal/gastric Colorectal Breast Endometrial Bladder Renal (kidney) Lymphoma Carcinoma <i>in situ</i> Prostate</p> <p>Nervous (DRG: 6)</p> <p>Carpal tunnel syndrome</p> <p>Pregnancy (DRGs: 354, 358, 366, 368, 370, 372, 390)</p> <p>Obstetric & gynecol. complications</p> <p>Signs/Symptoms Ill-Defined (DRGs: 87-88)</p> <p>Impaired respiratory function Sleep apnea Urinary stress incontinence</p>

As the first step toward estimating the direct medical care costs of each risk factor in relation to the targeted conditions, medical care claims utilization and cost data were obtained on as many California adults as possible for 2006 on a county-by-county basis. The California Office of State Health Planning and Development (OSHPD), the organization charged with acquiring, tracking, and managing all inpatient encounters, provided the inpatient claims data for the selected medical conditions.

Although no centralized database on outpatient claims for California is available, OSHPD tracks outpatient ambulatory surgery (AS) and emergency department (ED) encounters. These claims data were obtained for 2006. Because financial charge and payment data are not provided on either AS or ED encounters, an in-house California corporate medical claims database compiled by the authors was used. This database includes medical encounters and costs from numerous medical claims data

analyses that the authors performed for several California employers in the late 1990s. Because those employers are located in northern, central, and southern California, they provide a representative sample of health care utilization and cost patterns throughout the state. That database provided per-encounter payment norms (which were adjusted to year 2006 cost values) for AS and ED claims for the specific conditions.

Claims and costs for adults enrolled in Medi-Cal were based on 2004 data from California's Department of Health Services, Office of Fiscal Forecasting and Data Management. Due to the two-year lag, the 2004 claims were adjusted to 2006 values,¹⁶ and payments per selected condition were inflated to reflect actual California state-specific medical cost changes during that period.

Next, the prevalence of these three risk factors was combined with the medical care data for each county through a process developed by the authors known as the Proportionate Risk Factor Cost Appraisal™ (PRFCA). The PRFCA uses findings from published studies in peer-reviewed scientific journals to estimate the proportion of people who have a given risk factor (the risk factor weight) for designated medical conditions (i.e., any of the 100 or so DRGs).

Finally, the estimated number of people in each county who have the medical condition was multiplied by the average cost to treat that condition to get the total cost to treat that condition by county. Treatment costs for all conditions were then summed to determine the cost of medical care for conditions associated with each risk factor.

To estimate indirect health care costs associated with a health condition, health care economists generally multiply direct medical costs by a factor ranging from 2 to 9.^{17, 18} Indirect costs reflect any additional expense or lost opportunity that occurs in addition to the direct (immediate) medical cost associated with a medical condition. Examples of indirect costs include lingering or unexpected health problems that require additional medical care and/or prescription drugs, create additional stress or depression leading to a lower quality of life, or negatively affect an individual's ability to work at a level necessary for job promotion, greater earnings, and other advancement opportunities. In order to be conservative, the indirect costs were added as a multiple of 3.

Health Care Costs: Prescription Drugs

Prescription drug costs were assessed as complementary medical costs because they typically occur in conjunction with the provision of health care diagnoses or treatment. Prescription drug expenses associated with each of the targeted medical conditions are not available in a statewide database. Therefore, in order to calculate the approximate prescription drug costs associated with all of the targeted medical conditions for each of the three risk factors, claims data from several industry-leading drug utilization reports were used.^{19, 20}

Lost Productivity Costs

For the analysis of lost productivity costs associated with overweight, obesity, and physical inactivity, three outcome measures were used: absenteeism, short-term disability, and presenteeism (i.e., the portion of an employee's work load they are unable to do because of their compromised health status). The analysis is based on published scientific research on the effect of each of the three risk factors on each of the three measures of lost productivity.²¹

To determine lost productivity costs associated with each of the three outcome measures, estimates were made of the average annual number of hours of lost work time per individual associated with the presence of each the three risk factors. These were then summed to reflect the overall average estimated impact of each risk factor for an individual (see Table A-3 on next page).

Based on applicable regional and state data sources, the total cost of the lost productivity was then computed for each county using county- and state-specific data on risk-factor prevalence, the number of workers, and the average salary in the county.

TABLE A-3
Estimated Average Annual Number of Hours of Lost Work Time, per Individual, Associated with Overweight, Obesity, and Physical Inactivity, California, 2006

	Overweight	Obesity	Physical Inactivity
Absences	4.08 hours	12.43 hours	15.75 hours
Short-term disability	4.86 hours	14.78 hours	13.00 hours
Presenteeism	8.94 hours	27.19 hours	28.75 hours
TOTAL	17.88 hours	54.40 hours	57.50 hours
% Annual work*	0.89%	2.72%	2.80%

** Based on an annual workload of 2,000 hours.*

Study Limitations

Although this study was based on the best data available, the findings are limited by the following factors:


- The prevalence rates of overweight, obesity, and physical inactivity that were applied to each county are based on self-reports from respected state and national population-based surveys. Self-reported data are generally recognized as being underreported.²²
- The risk factor weights were based on a review of published studies for the general adult population. These weights could change as research findings are refined over time.
- In cases where specific health care cost data were not available, estimates were made. These include Medi-Cal managed care plan data, pharmaceutical drug costs paid by private and employer-paid sources, and employer-paid outpatient medical claims and cost data. The latter were estimated based on norms developed from the author's in-house California corporate database.
- Because county-specific lost productivity data were not available, national norms were used to estimate risk-factor-based absenteeism, short-term disability, and presenteeism rates.
- Lost productivity costs by county were based on the assumption that people work in the counties in which they live.

REFERENCES

1. U.S. Dept. of Health and Human Services. *Healthy People 2010: Understanding & Improving Health. 2nd Edition*, (2000). Office of Disease Prevention and Health Promotion.
2. Flegal, K., et al. (2005). Excess Deaths Associated with Underweight, Overweight, and Obesity. *JAMA*, 293, 1861-1867.
3. Gregg, E., et al. (2005). Secular Trends in Cardiovascular Disease Risk Factors According to Body Mass Index in US Adults. *JAMA*, 293, 1868-1874.
4. *The Surgeon General's Report on Physical Activity and Health* (1996). U.S. Department of Health & Human Services, Washington, D.C.
5. Goetzel, R., et al. (1998). The Association Between Ten Modifiable Risk Factors and Health Care Expenditures. *J Occup Environ Med*, 40, 10, 1-12.
6. Wasserman, J., et al. (2000). The Gender Specific Effects of Modifiable Risk Factors on Coronary Heart Disease and Related Health Care Expenditures. *J Occup Environ Med*, 42, 11, 973-985.
7. Anderson, D., et al. (2000). The Relationship Between Modifiable Health Risks and Group-Level Health Care Expenditures. *Am J Health Promot*, 15, 1, 45-52.
8. Pratt, M., Macera, G., and Wang, G. (2000). Higher Direct Medical Costs Associated with Physical Inactivity. *The Physician and Sportsmedicine*, 28, 10, 63-70.
9. Sturm, R. (2002). The Effect of Obesity, Smoking, and Drinking on Medical Problems and Costs. *Health Affairs*, 21, 2, 245-253.
10. Finkelstein, E., Fiebelkorn, I., and Wang, G. (2004). State-level Estimates of Annual Medical Expenditures Attributable to Obesity. *Obes Res*, 12, 1, 18-24.
11. Colditz, G. (1999). Economic Costs of Obesity and Inactivity. *Medicine & Science in Sports & Exercise*, 31, 11 (Supplement): S663-S667.
12. Analyses conducted by Chenoweth and Associates: (a) Chenoweth D. Economic Cost of Physical Inactivity in New York State. *J Am Med Ath Assn*, 14:1, 5-8, 2000; (b) *The Financial Cost of Specific Risk Factors in the Commonwealth of Massachusetts*. A Report to the Massachusetts Department of Public Health. November 7, 2003; (c) *The Economic Cost of Physical Inactivity in Michigan: A Study for the Michigan Fitness Foundation*. East Lansing, MI., May 21, 2003; (d) Chenoweth D. The Medical Cost of High Serum Cholesterol in Harris County, Texas. *J Tex Med*, 100: 5, 49-53, 2004; (e) *An Economic Cost Appraisal of Physical Inactivity, Obesity, and Overweight Among Maine Adults*, 2006. Retrieved May 20, 2008 from www.anthem.com/maine/weightstudy; (f) *The Economic Cost of Physical Inactivity Among Washington State Adults*. A Report for the Washington State Department of Health and the Washington Coalition to Promote Physical Activity. February 3, 2004; (g) *The Economic Cost of Selected Cardiovascular Risk Factors and Conditions Among North Carolina Adults* (2008). Be Active North Carolina, Inc. Durham, NC. Retrieved July 15, 2008 from www.beactiveinc.org.
13. Centers for Disease Control and Prevention. *Prevalence and Trends Data, California—2006, Overweight and Obesity*. Retrieved November 14, 2008, from <http://apps.nccd.cdc.gov/brrfs/display.asp?cat=0B&yr=2007&qkey=4409&state=CA>.
14. Centers for Disease Control and Prevention. *2007 vs. 2005 Prevalence Data: California, Physical Activity*. Retrieved May 20, 2008 from www.cdc.gov/nccd.
15. *The Economic Costs of Physical Inactivity, Obesity, and Overweight in California Adults During the Year 2000: A Technical Analysis* (April 2005). Sacramento, CA: California Department of Health Services.
16. Annual changes from 2004-2006 were relatively flat, averaging 5%. Sources: *California: Percent Change in Monthly Medicaid Enrollment and California: Average Annual Growth in Spending, FY 1990-2006* (both at www.statehealthfacts.org).
17. Goetzel, R., Hawkins, K., Ozminkowski, R., and Wang, S. (2003). The Health and Productivity Cost Burden of the Top 10 Physical and Mental Health Conditions Affecting Six Large U.S. Employers. *J Occup Environ Med*, 45, 5-14.
18. Gallagher, P., and Morgan, C. Measuring Indirect Costs in Workers' Compensation. Retrieved May 15, 2004 from www.milliman.com/health/publications/consultants_corner/mr_healthcc.55.html.
19. Express Scripts 2007 Drug Trend Report, April 2008.
20. Specialty Anti-Inflammatories See Huge Increase in Utilization. 2006 Express Scripts Specialty Drug Trend Report. (www.managed.caremag.com/archives/0607/0607.formfiles.html.)
21. Sources for lost productivity data include the following: Burton, W., et al. (1999). The Role of Health Risk Factors and Disease on Workers' Productivity. *J Occup Environ Med*, 41,10; Pronk, N., et al. (2004). The Association Between Work Performance and Physical Activity, Cardiorespiratory Fitness, and Obesity. *J Occup Environ Med*, 46, 1, 19-25; Goetzel, R., et al. (2004). Health, Absence, Disability, and Presenteeism Cost Estimates of Certain Physical and Mental Health Conditions Affecting U.S. Employers. *J Occup Environ Med*, 46, 398-412; Gates, D., et al. (2008). Obesity and Presenteeism: The Impact of Body Mass Index on Workplace Productivity. *J Occup Environ Med*, 50, 1, 39-45; Ricci, J., et al. (2005). Lost Productive Time Associated with Excess Weight in the U.S. Workforce. *J Occup Environ Med*, 47, 12, 1227-1234; Ostbye, T., et al. (2007). Obesity and Workers' Compensation: Results from the Duke Health and Safety Surveillance System. *Arch Intl Med*, 167, 766-773; Collins, J., et al. (2005). The Assessment of Chronic Health Conditions on Work Performance, Absence and Total Economic Impact for Employers. *J Occup Environ Med*, 47, 547-557; Burton, W., et al. (2005). The Association of Health Risks with On-the-Job Productivity. *J Occup Environ Med*, 47, 8, 769-777; Pronk, N., et al. (2004). The Association Between Work Performance and Physical Activity, Cardiorespiratory Fitness, and Obesity. *J Occup Environ Med*, 46, 1, 19-25; Tucker, L., and Friedman, G. (1998). Obesity and Absenteeism: An Epidemiologic Study of 10,825 Employed Adults. *Am J Health Promot*, 12, 3, 202-207; Goetzel, R., et al. (2004). Health, Absence, Disability, and Presenteeism Cost Estimates of Certain Physical and Mental Health Conditions Affecting U.S. Employers. *J Occup Environ Med*, 46, 398-412; Collins, J., et al. (2005). The Assessment of Chronic Work Conditions on Work Performance. *J Occup Environ Med*, 47, 547-557; Stewart, W., et al. (2003). Lost Productive Work Time Costs from Health Conditions in the United States: Results from the American Productivity Audit. *J Occup Environ Med*, 45, 12, 1234-1246. A complete list of references is available from the authors.
22. Ezzati, M., et al. (2006). Trends and National and State-Level Obesity in the USA After Correction for Self-Report Bias: Analysis of Health Surveys. *J Royal Soc Med*, 99:250-257.

Acknowledgments

Support for this project was provided by The California Endowment.

 The
California
Endowment

The California Center for Public Health Advocacy gratefully acknowledges Nancy Adess for editing and Cici Kinsman of C² Graphics for graphic design.

Citation

The Economic Costs of Overweight, Obesity, and Physical Inactivity Among California Adults—2006 (July 2009). The California Center for Public Health Advocacy.

Chenoweth & Associates, Inc.

Chenoweth & Associates, Inc. (C&A) is an econometrics consulting firm based in New Bern, North Carolina. Since 1979, C&A has provided strategic econometric services to business, industrial, health care, and government organizations and has conducted chronic disease risk factor cost analyses for state health departments and other health-related organizations in various states, including California, New York, North Carolina, Maine, Massachusetts, Michigan, Texas, and Washington.

128 St. Andrews Circle, New Bern, North Carolina 28562
(252) 636-3241 | www.chenoassociates.com

The California Center for Public Health Advocacy

The California Center for Public Health Advocacy is an independent, nonpartisan, non-profit organization that raises awareness about public health issues and mobilizes communities to promote the establishment of effective health policies.



Post Office Box 2309, Davis, CA 95617
(530) 297-6000 | FAX: (530) 297-6200

2201 Broadway, Suite 502, Oakland, CA 94512
(510) 302-3387 | FAX: (510) 444-8253

12921 Ramona Boulevard, Suite D, Irwindale, CA 91706
(626) 962-5900 | FAX: (626) 961-1609

www.PublicHealthAdvocacy.org

Sweetened Beverage Taxes



APRIL 30, 2009

Ounces of Prevention — The Public Policy Case for Taxes on Sugared Beverages

Kelly D. Brownell, Ph.D., and Thomas R. Frieden, M.D., M.P.H.

Sugar, rum, and tobacco are commodities which are nowhere necessities of life, which are become objects of almost universal consumption, and which are therefore extremely proper subjects of taxation.

Adam Smith, *The Wealth of Nations*, 1:76

The obesity epidemic has inspired calls for public health measures to prevent diet-related diseases. One controversial idea is now the subject of public debate: food taxes.

Forty states already have small taxes on sugared beverages and snack foods, but in the past year, Maine and New York have proposed large taxes on sugared beverages, and similar discussions have begun in other states. The size of the taxes, their potential for generating revenue and reducing consumption, and vigorous opposition by the beverage industry have resulted in substantial

controversy. Because excess consumption of unhealthful foods underlies many leading causes of death, food taxes at local, state, and national levels are likely to remain part of political and public health discourse.

Sugar-sweetened beverages (soda sweetened with sugar, corn syrup, or other caloric sweeteners and other carbonated and uncarbonated drinks, such as sports and energy drinks) may be the single largest driver of the obesity epidemic. A recent meta-analysis found that the intake of sugared beverages is associated with increased body weight, poor

nutrition, and displacement of more healthful beverages; increasing consumption increases risk for obesity and diabetes; the strongest effects are seen in studies with the best methods (e.g., longitudinal and interventional vs. correlational studies); and interventional studies show that reduced intake of soft drinks improves health.¹ Studies that do not support a relationship between consumption of sugared beverages and health outcomes tend to be conducted by authors supported by the beverage industry.²

Sugared beverages are marketed extensively to children and adolescents, and in the mid-1990s, children's intake of sugared beverages surpassed that of milk. In the past decade, per capita intake of calories from sugar-sweetened beverages has increased by nearly 30% (see bar graph)³; beverages now account for 10 to 15% of the

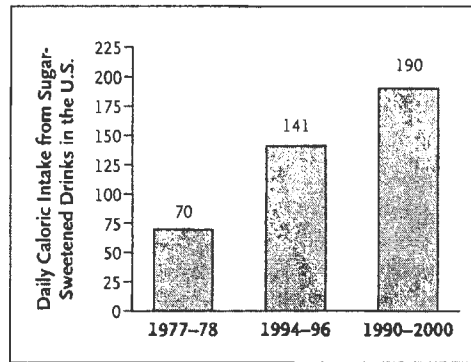
calories consumed by children and adolescents. For each extra can or glass of sugared beverage consumed per day, the likelihood of a child's becoming obese increases by 60%.⁴

Taxes on tobacco products have been highly effective in reducing consumption, and data indicate that higher prices also reduce soda consumption. A review conducted by Yale University's Rudd Center for Food Policy and Obesity suggested that for every 10% increase in price, consumption decreases by 7.8%. An industry trade publication reported even larger reductions: as prices of carbonated soft drinks increased by 6.8%, sales dropped by 7.8%, and as Coca-Cola prices increased by 12%, sales dropped by 14.6%.⁵

Such studies — and the economic principles that support their findings — suggest that a tax on sugared beverages would encourage consumers to switch to more healthful beverages, which would lead to reduced caloric intake and less weight gain.

The increasing affordability of soda — and the decreasing affordability of fresh fruits and vegetables (see line graph) — probably contributes to the rise in obesity in the United States. In 2008, a group of child and health care advocates in New York proposed a one-penny-per-ounce excise tax on sugared beverages, which would be expected to reduce consumption by 13% — about two servings per week per person. Even if one quarter of the calories consumed from sugared beverages are replaced by other food, the decrease in consumption would lead to an estimated reduction of 8000 calories per

person per year — slightly more than 2 lb each year for the average person. Such a reduction in calorie consumption would be expected to substantially reduce the risk of obesity and diabetes and may also reduce the risk of heart disease and other conditions.



Daily Caloric Intake from Sugar-Sweetened Drinks in the United States.

Data are from Nielsen and Popkin.³

Some argue that government should not interfere in the market and that products and prices will change as consumers demand more healthful food, but several considerations support government action. The first is externality — costs to parties not directly involved in a transaction. The contribution of unhealthy diets to health care costs is already high and is increasing — an estimated \$79 billion is spent annually for overweight and obesity alone — and approximately half of these costs are paid by Medicare and Medicaid, at taxpayers' expense. Diet-related diseases also cost society in terms of decreased work productivity, increased absenteeism, poorer school performance, and reduced fitness on the part of military recruits, among other negative effects.

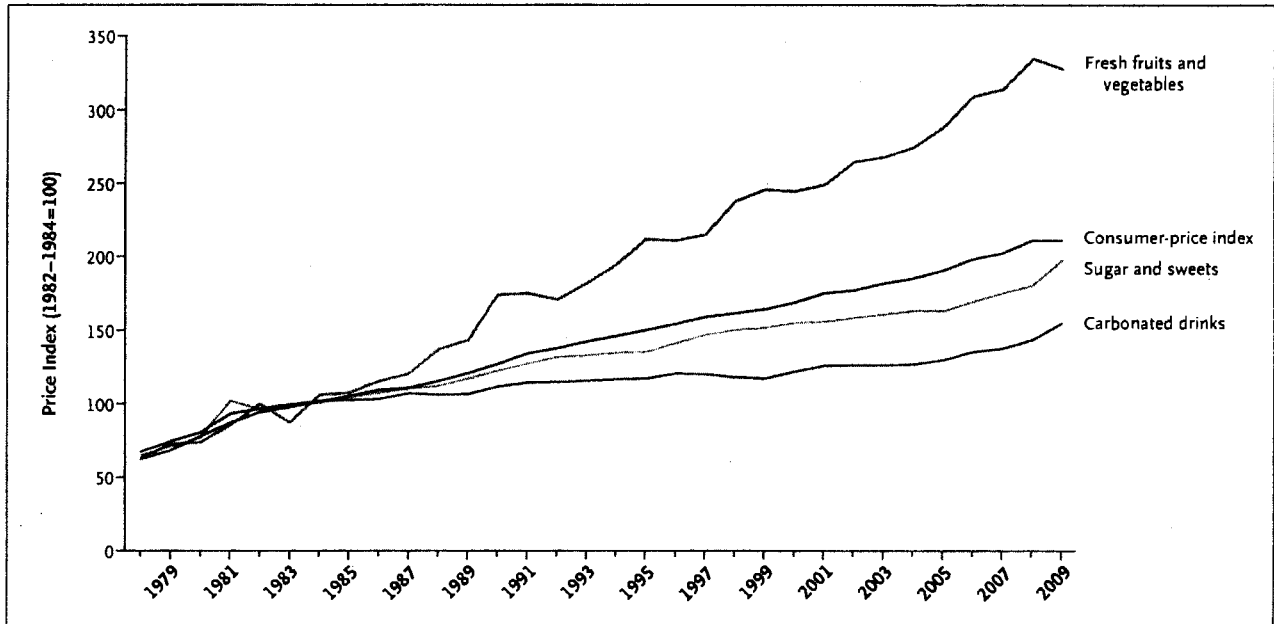
The second consideration is information asymmetry between the parties to a transaction. In

the case of sugared beverages, marketers commonly make health claims (e.g., that such beverages provide energy or vitamins) and use techniques that exploit the cognitive vulnerabilities of young children, who often cannot distinguish a television program from an advertisement.

A third consideration is revenue generation, which can further increase the societal benefits of a tax on soft drinks. A penny-per-ounce excise tax would raise an estimated \$1.2 billion in New York State alone. In times of economic hardship, taxes that both generate this much revenue and promote health are better options than revenue initiatives that may have adverse effects.

Objections have certainly been raised: that such a tax would be regressive, that food taxes are not comparable to tobacco or alcohol taxes because people must eat to survive, that it is unfair to single out one type of food for taxation, and that the tax will not solve the obesity problem. But the poor are disproportionately affected by diet-related diseases and would derive the greatest benefit from reduced consumption; sugared beverages are not necessary for survival; Americans consume about 250 to 300 more calories daily today than they did several decades ago, and nearly half this increase is accounted for by consumption of sugared beverages; and though no single intervention will solve the obesity problem, that is hardly a reason to take no action.

The full impact of public policies becomes apparent only after they take effect. We can estimate changes in sugared-drink con-



Relative Price Changes for Fresh Fruits and Vegetables, Sugars and Sweets, and Carbonated Drinks, 1978–2009.

Data are from the Bureau of Labor Statistics and represent the U.S. city averages for all urban consumers in January of each year.

sumption that would be prompted by a tax, but accompanying changes in the consumption of other foods or beverages are more difficult to predict. One question is whether the proportions of calories consumed in liquid and solid foods would change. And shifts among beverages would have different effects depending on whether consumers substituted water, milk, diet drinks, or equivalent generic brands of sugared drinks.

Effects will also vary depending on whether the tax is designed to reduce consumption, generate revenue, or both; the size of the tax; whether the revenue is earmarked for programs related to nutrition and health; and where in the production and distribution chain the tax is applied. Given the heavy consumption of sugared beverages, even small taxes will generate substantial revenue, but only heftier taxes will significantly reduce consumption.

Sales taxes are the most common form of food tax, but because they are levied as a percentage of the retail price, they encourage the purchase of less-expensive brands or larger containers. Excise taxes structured as a fixed cost per ounce provide an incentive to buy less and hence would be much more effective in reducing consumption and improving health. In addition, manufacturers generally pass the cost of an excise tax along to their customers, including it in the price consumers see when they are making their selection, whereas sales taxes are seen only at the cash register.

Although a tax on sugared beverages would have health benefits regardless of how the revenue was used, the popularity of such a proposal increases greatly if revenues are used for programs to prevent childhood obesity, such as media campaigns, facilities and programs for phys-

ical activity, and healthier food in schools. Poll results show that support of a tax on sugared beverages ranges from 37 to 72%; a poll of New York residents found that 52% supported a “soda tax,” but the number rose to 72% when respondents were told that the revenue would be used for obesity prevention. Perhaps the most defensible approach is to use revenue to subsidize the purchase of healthful foods. The public would then see a relationship between tax and benefit, and any regressive effects would be counteracted by the reduced costs of healthful food.

A penny-per-ounce excise tax could reduce consumption of sugared beverages by more than 10%. It is difficult to imagine producing behavior change of this magnitude through education alone, even if government devoted massive resources to the task. In contrast, a sales tax on sugared drinks would generate considerable rev-

enue, and as with the tax on tobacco, it could become a key tool in efforts to improve health.

No potential conflict of interest relevant to this article was reported.

Dr. Brownell is a professor and director of the Rudd Center for Food Policy and Obesity, Yale University, New Haven, CT. Dr. Frieden is the health commissioner for the City of New York.

This article (10.1056/NEJMp0902392) was published at NEJM.org on April 8, 2009.

1. Vartanian LR, Schwartz MB, Brownell KD. Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis. *Am J Public Health* 2007;97:667-75.

2. Forshee RA, Anderson PA, Storey ML. Sugar-sweetened beverages and body mass index in children and adolescents: a meta-analysis. *Am J Clin Nutr* 2008;87:1662-71.

3. Nielsen SJ, Popkin BM. Changes in beverage intake between 1977 and 2001. *Am J Prev Med* 2004;27:205-10. [Erratum, *Am J Prev Med* 2005;28:413.]

4. Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. *Lancet* 2001;357:505-8.

5. Elasticity: big price increases cause Coke volume to plummet. *Beverage Digest*. November 21, 2008:3-4.

Copyright © 2009 Massachusetts Medical Society.

GLOBAL HEALTH

Rationing Antiretroviral Therapy in Africa — Treating Too Few, Too Late

Nathan Ford, D.H.A., Edward Mills, Ph.D., and Alexandra Calmy, M.D.

Related article, p. 1815

The past 6 years have seen striking advances in access to antiretroviral therapy in Africa. From 2002 onward, the international drive to scale up antiretroviral treatment gained considerable momentum, most notably with the establishment of the Global Fund to Fight AIDS, Tuberculosis, and Malaria, the “3 by 5” Initiative of the World Health Organization (WHO), and the U.S. President’s Emergency Plan for AIDS Relief (PEPFAR). Today, an estimated 3 million people in the developing world are receiving antiretroviral therapy.

The momentum has now begun to wane, with various groups arguing that the focus on AIDS has had its day and that health care funding should now be redirected to other areas, such as maternal and child health and primary care. But before the international community gives up on prioritizing care for patients with HIV infection, we believe that on-the-ground discussions must address not only whether enough has been done to scale up treatment but also whether

the treatment that patients are receiving is good enough.

The standard approach to HIV treatment in Africa is to wait until people are visibly sick, treat them with effective but poorly tolerated drugs, and then wait until they are sick again before switching regimens. There are several problems with this approach.

The first is that too few people are receiving treatment. The 3 million people receiving antiretroviral therapy are usually said to account for about 30% of the need for such treatment, but even this rate reflects the use of stringent eligibility criteria that have been abandoned in wealthier countries.

Second, we are waiting until people are symptomatic before they are treated. In most African countries, patients begin receiving treatment when the CD4+ count falls below 200 cells per cubic millimeter, at which point most patients already have symptomatic and severe (WHO stage 3 or 4) infection. In the United States and Europe, treatment is initiated earlier — as

soon as the CD4+ count reaches 350 cells per cubic millimeter — and increasingly, experts are arguing that even that is too late.

In many patients in Africa, the CD4+ count takes only about a year to decline from the cutoff for such early initiation to that for the later initiation now practiced in developing countries.¹ Although delaying therapy may mean saving money on drugs during this period, the long-term cost of such delays is increased substantially by the need for more intensive clinical care, decreased income, and likely regimen switches. Cost is thus no longer a tenable justification for delaying therapy. More important, recent observational data presented by Kitahata et al. in this issue of the *Journal* (pages 1815–1826) show that the risk of death increases by 69% when the initiation of therapy is delayed until the CD4+ count drops below 350 cells per cubic millimeter. Patients’ immunologic nadir — how low their CD4+ count is allowed to drop — is predictive of the degree of benefit they will

HEALTH POLICY REPORT

The Public Health and Economic Benefits of Taxing Sugar-Sweetened Beverages

Kelly D. Brownell, Ph.D., Thomas Farley, M.D., M.P.H., Walter C. Willett, M.D., Dr.P.H., Barry M. Popkin, Ph.D., Frank J. Chaloupka, Ph.D., Joseph W. Thompson, M.D., M.P.H., and David S. Ludwig, M.D., Ph.D.

The consumption of sugar-sweetened beverages has been linked to risks for obesity, diabetes, and heart disease¹⁻³; therefore, a compelling case can be made for the need for reduced consumption of these beverages. Sugar-sweetened beverages are beverages that contain added, naturally derived caloric sweeteners such as sucrose (table sugar), high-fructose corn syrup, or fruit-juice concentrates, all of which have similar metabolic effects.

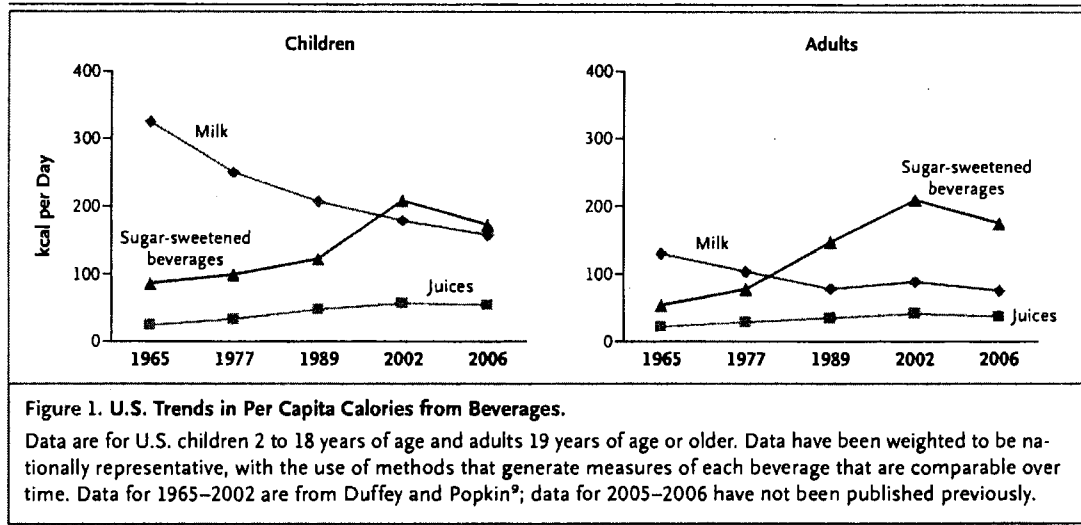
Taxation has been proposed as a means of reducing the intake of these beverages and thereby lowering health care costs, as well as a means of generating revenue that governments can use for health programs.⁴⁻⁷ Currently, 33 states have sales taxes on soft drinks (mean tax rate, 5.2%), but the taxes are too small to affect consumption and the revenues are not earmarked for programs related to health. This article examines trends in the consumption of sugar-sweetened beverages, evidence linking these beverages to adverse health outcomes, and approaches to designing a tax system that could promote good nutrition and help the nation recover health care costs associated with the consumption of sugar-sweetened beverages.

CONSUMPTION TRENDS AND HEALTH OUTCOMES

In recent decades, intake of sugar-sweetened beverages has increased around the globe; for example, intake in Mexico doubled between 1999 and 2006 across all age groups.⁸ Between 1977 and 2002, the per capita intake of caloric beverages doubled in the United States across all age groups⁹ (Fig. 1). The most recent data (2005–2006) show that children and adults in the United States consume about 172 and 175 kcal daily, respectively, per capita from sugar-sweetened beverages.

The relationship between the consumption of sugar-sweetened beverages and body weight has been examined in many cross-sectional and longitudinal studies and has been summarized in systematic reviews.^{1,2} A meta-analysis showed positive associations between the intake of sugar-sweetened beverages and body weight — associations that were stronger in longitudinal studies than in cross-sectional studies and in studies that were not funded by the beverage industry than in those that were.² A meta-analysis of studies involving children¹⁰ — a meta-analysis that was supported by the beverage industry — was interpreted as showing that there was no evidence of an association between consumption of sugar-sweetened beverages and body weight, but it erroneously gave large weight to several small negative studies; when a more realistic weighting was used, the meta-analysis summary supported a positive association.¹¹ A prospective study involving middle-school students over the course of 2 academic years showed that the risk of becoming obese increased by 60% for every additional serving of sugar-sweetened beverages per day.¹² In an 8-year prospective study involving women, those who increased their consumption of sugar-sweetened beverages at year 4 and maintained this increase gained 8 kg, whereas those who decreased their intake of sugar-sweetened beverages at year 4 and maintained this decrease gained only 2.8 kg.¹³

Short-term clinical trials provide an experimental basis for understanding the way in which sugar-sweetened beverages may affect adiposity. Tordoff and Alleva¹⁴ found that as compared with total energy intake and weight during a 3-week period in which no beverages were provided, total energy intake and body weight increased when subjects were given 530 kcal of sugar-sweetened beverages per day for 3 weeks but decreased when



subjects were given noncaloric sweetened beverages for the same length of time. Raben et al.¹⁵ reported that obese subjects gained weight when they were given sucrose, primarily in the form of sugar-sweetened beverages, for 10 weeks, whereas they lost weight when they were given noncaloric sweeteners for the same length of time.

Four long-term, randomized, controlled trials examining the relationship between the consumption of sugar-sweetened beverages and body weight have been reported; the results showed the strongest effects among overweight persons. A school-based intervention to reduce the consumption of carbonated beverages was assessed among 644 students, 7 to 11 years of age, in the United Kingdom with the use of a cluster design.¹⁶ After 1 year, the intervention group, as compared with the control group, had a nonsignificantly lower mean body-mass index (the weight in kilograms divided by the square of the height in meters) and a significant 7.7% lower incidence of obesity. In a study involving 1140 Brazilian schoolchildren, 9 to 12 years of age, that was designed to discourage the consumption of sugar-sweetened beverages, no overall effect on body-mass index was observed during the 9-month academic year.¹⁷ Among students who were overweight at baseline, the body-mass index was nonsignificantly decreased in the intervention group as compared with the control group; the difference was significant among overweight girls. In another clinical trial, 103 high-school students in Boston were assigned to a control group or to an intervention group that received

home delivery of noncaloric beverages for 25 weeks. The body-mass index was nonsignificantly reduced in the overall intervention group, but among students in the upper third of body-mass index at baseline, there was a significant decrease in the body-mass index in the intervention group, as compared with the control group (a decrease of 0.63 vs. an increase of 0.12).¹⁸ The effects of replacing sugar-sweetened beverages with milk products were examined among 98 overweight Chilean children.¹⁹ After 16 weeks, there was a nonsignificantly lower increase in the percentage of body fat in the intervention group than in the control group (0.36% and 0.78% increase, respectively), whereas there was a significantly greater increase in lean mass in the intervention group (0.92 vs. 0.62 kg).

Three prospective, observational studies — one involving nurses in the United States, one involving Finnish men and women, and one involving black women — each showed positive associations between the consumption of sugar-sweetened beverages and the risk of type 2 diabetes.^{13,20,21} Among the 91,249 women in the Nurses' Health Study II who were followed for 8 years, the risk of diabetes among women who consumed one or more servings of sugar-sweetened beverages per day was nearly double the risk among women who consumed less than one serving of sugar-sweetened beverages per month¹³; about half the excess risk was accounted for by greater body weight. Among black women, excess weight accounted for most of the excess risk.

Among 88,520 women in the Nurses' Health

Study, the risk of coronary heart disease among women who consumed one serving of sugar-sweetened beverages per day, as compared with women who consumed less than one serving per month, was increased by 23%, and among those who consumed two servings or more per day, the risk was increased by 35%.³ Increased body weight explained some, but not all, of this association.

MECHANISMS LINKING
SUGAR-SWEETENED BEVERAGES
WITH POOR HEALTH

A variety of behavioral and biologic mechanisms may be responsible for the associations between the consumption of sugar-sweetened beverages and adverse health outcomes, with some links (e.g., the link between intake of sugar-sweetened beverages and weight gain) better established than others. The well-documented adverse physiological and metabolic consequences of a high intake of refined carbohydrates such as sugar include the elevation of triglyceride levels and of blood pressure and the lowering of high-density lipoprotein cholesterol levels, which would be expected to increase the risk of coronary heart disease.²² Because of the high glycemic load of sugar-sweetened beverages, consumption of these beverages would be expected to increase the risk of diabetes by causing insulin resistance and also through direct effects on pancreatic islet cells.²³ Observational research has shown that consumption of sugar-sweetened beverages, but not of noncalorically sweetened beverages, is associated with markers of insulin resistance.²⁴

Intake of sugar-sweetened beverages may cause excessive weight gain owing in part to the apparently poor satiating properties of sugar in liquid form. Indeed, adjustment of caloric intake at subsequent meals for energy that had been consumed as a beverage is less complete than adjustment of intake for energy that had been consumed as a solid food.²⁵ For example, in a study involving 323 adults, in which 7-day food diaries were used, energy from beverages added to total energy intake instead of displacing other sources of calories.²⁶ The results of a study of school-age children were consistent with the data from adults and showed that children who drank 9 oz or more of sugar-sweetened beverages per day consumed nearly 200 kcal per day more than

those who did not drink sugar-sweetened beverages.²⁷

Short-term studies of the effect of beverage consumption on energy intake support this mechanism. Among 33 adults who were given identical test lunches on six occasions but were given beverages of different types (sugar-sweetened cola, noncaloric cola, or water) and amounts (12 oz [355 ml] or 18 oz [532 ml]),²⁸ the intake of solid food did not differ across conditions; the result was that there was significantly greater total energy consumption when the sugar-sweetened beverages were served.

Sugar-sweetened beverages may also affect body weight through other behavioral mechanisms. Whereas the intake of solid food is characteristically coupled to hunger, people may consume sugar-sweetened beverages in the absence of hunger, to satisfy thirst or for social reasons. Sugar-sweetened beverages may also have chronic adverse effects on taste preferences and food acceptance. Persons — especially children — who habitually consume sugar-sweetened beverages rather than water may find more satiating but less sweet foods (e.g., vegetables, legumes, and fruits) unappealing or unpalatable, with the result that their diet may be of poor quality.

ECONOMIC RATIONALE

Economists agree that government intervention in a market is warranted when there are “market failures” that result in less-than-optimal production and consumption.^{29,30} Several market failures exist with respect to sugar-sweetened beverages. First, because many persons do not fully appreciate the links between consumption of these beverages and health consequences, they make consumption decisions with imperfect information. These decisions are likely to be further distorted by the extensive marketing campaigns that advertise the benefits of consumption. A second failure results from time-inconsistent preferences (i.e., decisions that provide short-term gratification but long-term harm). This problem is exacerbated in the case of children and adolescents, who place a higher value on present satisfaction while more heavily discounting future consequences. Finally, financial “externalities” exist in the market for sugar-sweetened beverages in that consumers do not bear the full costs of their consumption decisions. Because of the contribu-

tion of the consumption of sugar-sweetened beverages to obesity, as well as the health consequences that are independent of weight, the consumption of sugar-sweetened beverages generates excess health care costs. Medical costs for overweight and obesity alone are estimated to be \$147 billion — or 9.1% of U.S. health care expenditures — with half these costs paid for publicly through the Medicare and Medicaid programs.³¹

AN EFFECTIVE TAX POLICY
AND PROJECTED EFFECTS

Key factors to consider in developing an effective policy include the definition of taxable beverages, the type of tax (sales tax or excise tax), and the tax rate. We propose an excise tax of 1 cent per ounce for beverages that have any added caloric sweetener. An alternative would be to tax beverages that exceed a threshold of grams of added caloric sweetener or of kilocalories per ounce. If this approach were used, we would recommend that the threshold be set at 1 g of sugar per ounce (30 ml) (32 kcal per 8 oz [237 ml]). Another option would be a tax assessed per gram of added sugar, but such an approach would be difficult to administer. The advantage of taxing beverages that have any added sugar is that this kind of tax is simpler to administer and it may promote the consumption of no-calorie beverages, most notably water; however, a threshold approach would also promote calorie reductions and would encourage manufacturers to reformulate products. A consumer who drinks a conventional soft drink (20 oz [591 ml]) every day and switches to a beverage below this threshold would consume approximately 174 fewer calories each day.

A specific excise tax (a tax levied on units such as volume or weight) per ounce or per gram of added sugar would be preferable to a sales tax or an ad valorem excise tax (a tax levied as a percentage of price) and would provide an incentive to reduce the amount of sugar per ounce of a sugar-sweetened beverage. Sales taxes added as a percentage of retail cost would have three disadvantages: they could simply encourage the purchase of lower-priced brands (thus resulting in no calorie reduction) or of large containers that cost less per ounce; consumers would become aware of the added tax only after making the decision to purchase the beverage; and the syrups

that are used in fountain drinks, which are often served with multiple refills, would remain untaxed. A number of states currently exempt sugar-sweetened beverages from sales taxes along with food, presumably because food is a necessity. This practice should be eliminated, whether or not an excise tax is enacted.

Excise taxes could be levied on producers and wholesalers, and the cost would almost certainly be passed along to retailers, who would then incorporate it into the retail price; thus, consumers would become aware of the cost at the point of making a purchase decision. Taxes levied on producers and wholesalers would be much easier to collect and enforce than taxes levied on retailers because of the smaller number of businesses that would have to comply with the tax; in addition, the sugar used in syrups could be taxed — a major advantage because of the heavy sales of fountain drinks. Experience with tobacco and alcohol taxes suggests that specific excise taxes have a greater effect on consumption than do ad valorem excise taxes and can also generate more stable revenues because they are less dependent on industry pricing strategies.³² In addition, tax laws should be written with provisions for the regular adjustment of specific excise taxes to keep pace with inflation, in order to prevent the effect of the taxes on both prices and revenues from eroding over time.

A tax of 1 cent per ounce of beverage would increase the cost of a 20-oz (591-ml) soft drink by 15 to 20%. The effect on consumption can be estimated through research on price elasticity (i.e., consumption shifts produced by price). The price elasticity for all soft drinks is in the range of -0.8 to 1.0.³³ (Elasticity of -0.8 suggests that for every 10% increase in price, there would be a decrease in consumption of 8%, whereas elasticity of 1.0 suggests that for every 10% increase in price, there would be a decrease in consumption of 10%.) Even greater price effects are expected from taxing only sugar-sweetened beverages, since some consumers will switch to diet beverages. With the use of a conservative estimate that consumers would substitute calories in other forms for 25% of the reduced calorie consumption, an excise tax of 1 cent per ounce would lead to a minimum reduction of 10% in calorie consumption from sweetened beverages, or 20 kcal per person per day, a reduction that is sufficient for weight loss and reduction in risk (unpublished

data). The benefit would be larger among consumers who consume higher volumes, since these consumers are more likely to be overweight and appear to be more responsive to prices.⁷ Higher taxes would have greater benefits.

A controversial issue is whether to tax beverages that are sweetened with noncaloric sweeteners. No adverse health effects of noncaloric sweeteners have been consistently demonstrated, but there are concerns that diet beverages may increase calorie consumption by justifying consumption of other caloric foods or by promoting a preference for sweet tastes.³⁴ At present, we do not propose taxing beverages with noncaloric sweeteners, but we recommend close tracking of studies to determine whether taxing might be justified in the future.

REVENUE-GENERATING POTENTIAL

The revenue generated from a tax on sugar-sweetened beverages would be considerable and could be used to help support childhood nutrition programs, obesity-prevention programs, or health care for the uninsured or to help meet general revenue needs. A national tax of 1 cent per ounce on sugar-sweetened beverages would raise \$14.9 billion in the first year alone. Taxes at the state level would also generate considerable revenue — for example, \$139 million in Arkansas, \$183 million in Oregon, \$221 million in Alabama, \$928 million in Florida, \$937 million in New York, \$1.2 billion in Texas, and \$1.8 billion in California. A tax calculator that is available online can generate revenue numbers for states and 25 major cities.³⁵

OBJECTIONS, INDUSTRY REACTION, PUBLIC SUPPORT, AND FRAMING

One objection to a tax on sugar-sweetened beverages is that it would be regressive. This argument arose with respect to tobacco taxes but was challenged successfully by proponents of the taxes, who pointed out that the poor face a disproportionate burden of smoking-related illnesses, that nearly all smokers begin to smoke when they are teenagers, and that both groups are sensitive to price changes.⁷ In addition, some of the tobacco revenue has been used for programs developed specifically for the poor and for youth. The poor are most affected by illnesses that are

related to unhealthy diets, and brand loyalties for beverages tend to be set by the teenage years. In addition, sugar-sweetened beverages are not necessary for survival, and an alternative (i.e., water) is available at little or no cost; hence, a tax that shifted intake from sugar-sweetened beverages to water would benefit the poor both by improving health and by lowering expenditures on beverages. Designating revenues for programs promoting childhood nutrition, obesity prevention, or health care for the uninsured would preferentially help those most in need.

A second objection is that taxing sugar-sweetened beverages will not solve the obesity crisis and is a blunt instrument that affects even those who consume small amounts of such beverages. Seat-belt legislation and tobacco taxation do not eliminate traffic accidents and heart disease but are nevertheless sound policies. Similarly, obesity is unlikely to yield to any single policy intervention, so it is important to pursue multiple opportunities to obtain incremental gains. Reducing caloric intake by 1 to 2% per year would have a marked impact on health in all age groups, and the financial burden on those who consumed small amounts of sugar-sweetened beverages would be minimal.

Opposition to a tax by the beverage industry is to be expected, given the possible effect on sales; opposition has been seen in jurisdictions that have considered such taxes and can be predicted from the behavior of the tobacco industry under similar circumstances.³⁶ PepsiCo threatened to move its corporate headquarters out of New York when the state considered implementing an 18% sales tax on sugar-sweetened beverages.³⁷ The tobacco industry fought policy changes by creating front groups with names that suggested community involvement. The beverage industry has created Americans Against Food Taxes.³⁸ These reactions suggest that the beverage industry believes that a tax would have a substantial impact on consumption.

Public support for food and beverage taxes to address obesity has increased steadily. Questions about taxes in polls have been asked in various ways, and the results are therefore not directly comparable from year to year, but overall trends are clear. Support for food taxes rose from 33% in 2001 to 41% in 2003 and then to 54% in 2004.³⁹ A 2008 poll of New York State residents showed that 52% of respondents support a soda

tax; 72% support such a tax if the revenue is used to support programs for the prevention of obesity in children and adults. The way in which the issue is framed is essential; support is highest when the tax is introduced in the context of promoting health and when the revenues are earmarked for programs promoting childhood nutrition or obesity prevention.

CONCLUSIONS

The federal government, a number of states and cities, and some countries (e.g., Mexico⁹) are considering levying taxes on sugar-sweetened beverages. The reasons to proceed are compelling. The science base linking the consumption of sugar-sweetened beverages to the risk of chronic diseases is clear. Escalating health care costs and the rising burden of diseases related to poor diet create an urgent need for solutions, thus justifying government's right to recoup costs.

As with any public health intervention, the precise effect of a tax cannot be known until it is implemented and studied, but research to date suggests that a tax on sugar-sweetened beverages would have strong positive effects on reducing consumption.^{5,33} In addition, the tax has the potential to generate substantial revenue to prevent obesity and address other external costs resulting from the consumption of sugar-sweetened beverages, as well as to fund other health-related programs. Much as taxes on tobacco products are routine at both state and federal levels because they generate revenue and they confer a public health benefit with respect to smoking rates, we believe that taxes on beverages that help drive the obesity epidemic should and will become routine.

Supported in part by grants from the Rudd Foundation (to Dr. Brownell), the National Institutes of Health (R01-CA121152, to Dr. Popkin), and the Robert Wood Johnson Foundation (to Dr. Chaloupka).

No potential conflict of interest relevant to this article was reported.

From the Rudd Center for Food Policy and Obesity, Yale University, New Haven, CT (K.D.B.); the Department of Health and Mental Hygiene, City of New York (T.F.); the Department of Nutrition, Harvard School of Public Health (W.C.W.), and the Optimal Weight for Life Program, Children's Hospital, and Harvard Medical School (D.S.L.) — all in Boston; the Department of Nutrition and the University of North Carolina Interdisciplinary Obesity Center, University of North Carolina, Chapel Hill (B.M.P.); the Department of Economics and the University of Chicago at Illinois Health Policy Center, University of Illinois, Chicago (F.J.C.); and the University of Arkansas for Medical Sci-

ences and the Surgeon General's Office, State of Arkansas, Little Rock (J.W.T.).

This article (10.1056/NEJMhpr0905723) was published on September 16, 2009, at NEJM.org.

1. Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systematic review. *Am J Clin Nutr* 2006;84:274-88.
2. Vartanian LR, Schwartz MB, Brownell KD. Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis. *Am J Public Health* 2007;97:667-75.
3. Fung TT, Malik V, Rexrode KM, Manson JE, Willett WC, Hu FB. Sweetened beverage consumption and risk of coronary heart disease in women. *Am J Clin Nutr* 2009;89:1037-42.
4. Brownell KD. Get slim with higher taxes. *New York Times*. December 15, 1994:A29.
5. Brownell KD, Frieden TR. Ounces of prevention — the public policy case for taxes on sugared beverages. *N Engl J Med* 2009;360:1805-8.
6. Jacobson MF, Brownell KD. Small taxes on soft drinks and snack foods to promote health. *Am J Public Health* 2000;90:854-7.
7. Powell LM, Chaloupka FJ. Food prices and obesity: evidence and policy implications for taxes and subsidies. *Milbank Q* 2009;87:229-57.
8. Barquera S, Hernandez-Barrera L, Tolentino ML, et al. Energy intake from beverages is increasing among Mexican adolescents and adults. *J Nutr* 2008;138:2454-61.
9. Duffey KJ, Popkin BM. Shifts in patterns and consumption of beverages between 1965 and 2002. *Obesity (Silver Spring)* 2007;15:2739-47.
10. Forshee RA, Anderson PA, Storey ML. Sugar-sweetened beverages and body mass index in children and adolescents: a meta-analysis. *Am J Clin Nutr* 2008;87:1662-71. [Erratum, *Am J Clin Nutr* 2009;89:441-2.]
11. Malik VS, Willett WC, Hu FB. Sugar-sweetened beverages and BMI in children and adolescents: reanalyses of a meta-analysis. *Am J Clin Nutr* 2009;89:438-9.
12. Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. *Lancet* 2001;357:505-8.
13. Schulze MB, Manson JE, Ludwig DS, et al. Sugar-sweetened beverages, weight gain, and incidence of type 2 diabetes in young and middle-aged women. *JAMA* 2004;292:927-34.
14. Tordoff MG, Alleva AM. Effect of drinking soda sweetened with aspartame or high-fructose corn syrup on food intake and body weight. *Am J Clin Nutr* 1990;51:963-9.
15. Raben A, Vasilaras TH, Moller AC, Astrup A. Sucrose compared with artificial sweeteners: different effects on ad libitum food intake and body weight after 10 wk of supplementation in overweight subjects. *Am J Clin Nutr* 2002;76:721-9.
16. James J, Thomas P, Cavan D, Kerr D. Preventing childhood obesity by reducing consumption of carbonated drinks: cluster randomised controlled trial. *BMJ* 2004;328:1237. [Erratum, *BMJ* 2004;328:1236.]
17. Sichert R, Paula Trotte A, de Souza RA, Veiga GV. School randomised trial on prevention of excessive weight gain by discouraging students from drinking sodas. *Public Health Nutr* 2009;12:197-202.
18. Ebbeling CB, Feldman HA, Osganian SK, Chomitz VR, Ellenbogen SJ, Ludwig DS. Effects of decreasing sugar-sweetened beverage consumption on body weight in adolescents: a randomized, controlled pilot study. *Pediatrics* 2006;117:673-80.
19. Albala C, Ebbeling CB, Cifuentes M, Lera L, Bustos N, Ludwig DS. Effects of replacing the habitual consumption of sugar-sweetened beverages with milk in Chilean children. *Am J Clin Nutr* 2008;88:605-11.
20. Montonen J, Jarvinen R, Knekt P, Heliövaara M, Reunanen A.

- Consumption of sweetened beverages and intakes of fructose and glucose predict type 2 diabetes occurrence. *J Nutr* 2007;137:1447-54.
21. Palmer JR, Boggs DA, Krishnan S, Hu FB, Singer M, Rosenberg L. Sugar-sweetened beverages and incidence of type 2 diabetes mellitus in African American women. *Arch Intern Med* 2008;168:1487-92.
 22. Appel LJ, Sacks FM, Carey VJ, et al. Effects of protein, mono-unsaturated fat, and carbohydrate intake on blood pressure and serum lipids: results of the OmniHeart randomized trial. *JAMA* 2005;294:2455-64.
 23. Ludwig DS. The glycemic index: physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. *JAMA* 2002;287:2414-23.
 24. Yoshida M, McKeown NM, Rogers G, et al. Surrogate markers of insulin resistance are associated with consumption of sugar-sweetened drinks and fruit juice in middle and older-aged adults. *J Nutr* 2007;137:2121-7.
 25. Mourao DM, Bressan J, Campbell WW, Mattes RD. Effects of food form on appetite and energy intake in lean and obese young adults. *Int J Obes (Lond)* 2007;31:1688-95.
 26. De Castro JM. The effects of the spontaneous ingestion of particular foods or beverages on the meal pattern and overall nutrient intake of humans. *Physiol Behav* 1993;53:1133-44.
 27. Harnack L, Stang J, Story M. Soft drink consumption among US children and adolescents: nutritional consequences. *J Am Diet Assoc* 1999;99:436-41.
 28. Flood JE, Roe LS, Rolls BJ. The effect of increased beverage portion size on energy intake at a meal. *J Am Diet Assoc* 2006;106:1984-90.
 29. Cawley J. An economic framework for understanding physical activity and eating behaviors. *Am J Prev Med* 2004;27:117-25.
 30. Finkelstein EA, Ruhm CJ, Kosa KM. Economic causes and consequences of obesity. *Annu Rev Public Health* 2005;26:239-57.
 31. Finkelstein EA, Trogdon JG, Cohen JW, Dietz W. Annual medical spending attributable to obesity: payer- and service-specific estimates. *Health Aff (Millwood)* 2009 July 29 (Epub ahead of print).
 32. Chaloupka FJ, Peck RM., Tauras JA, Yurekli A. Cigarette excise taxation: the impact of tax structure on prices, revenues, and cigarettes smoking. Geneva: World Health Organization (in press).
 33. Andreyeva T, Long MW, Brownell KD. The impact of food prices on consumption: a systematic review of research on price elasticity of demand for food. *Am J Public Health* (in press).
 34. Mattes RD, Popkin BM. Nonnutritive sweetener consumption in humans: effects on appetite and food intake and their putative mechanisms. *Am J Clin Nutr* 2009;89:1-14.
 35. Rudd Center for Food Policy and Obesity. Revenue calculator for soft drink taxes. (Accessed September 4, 2009, at <http://www.yaleruddcenter.org/sodatax.aspx>.)
 36. Brownell KD, Warner KE. The perils of ignoring history: big tobacco played dirty and millions died: how similar is big food? *Milbank Q* 2009;87:259-94.
 37. Hakim D, McGeehan P. New York vulnerable to poaching in recession. *New York Times*. March 1, 2009.
 38. Americans Against Food Taxes home page. (Accessed September 4, 2009, at <http://nofoodtaxes.com>.)
 39. Brownell KD. The chronicling of obesity: growing awareness of its social, economic, and political contexts. *J Health Polit Policy Law* 2005;30:955-64.

Copyright © 2009 Massachusetts Medical Society.

By Roland Sturm, Lisa M. Powell, Jamie F. Chriqui, and Frank J. Chaloupka

Soda Taxes, Soft Drink Consumption, And Children's Body Mass Index

DOI: 10.1377/hlthaff.2009.0061
HEALTH AFFAIRS 29,
NO. 5 (2010): –
©2010 Project HOPE—
The People-to-People Health
Foundation, Inc.

ABSTRACT Taxes on sugar-sweetened beverages have been proposed to combat obesity. Using data on state sales taxes for soda and individual-level data on children, we examine whether small taxes are likely to change consumption and weight gain or whether larger tax increases would be needed. We find that existing taxes on soda, which are typically not much higher than 4 percent in grocery stores, do not substantially affect overall levels of soda consumption or obesity rates. We do find, however, that subgroups of at-risk children—children who are already overweight, come from low-income families, or are African American—may be more sensitive than others to soda taxes, especially when soda is available at school. A greater impact of these small taxes could come from the dedication of the revenues they generate to other obesity prevention efforts rather than through their direct effect on consumption.

Roland Sturm (sturm@rand.org) is a senior economist at the RAND Corporation in Santa Monica, California.

Lisa M. Powell is a research professor at the Institute for Health Research and Policy, University of Illinois at Chicago.

Jamie F. Chriqui is a senior research scientist at the Institute for Health Research and Policy.

Frank J. Chaloupka is a distinguished professor of economics and public health and director of the Health Policy Center, University of Illinois at Chicago.

Carbonated soft drinks, or soda, and other sugar-sweetened beverages such as fruit punch, sweetened tea, and sports drinks are commonly targeted in anti-obesity initiatives. One of the most common—and most controversial—proposals is the notion of taxing these beverages, based on the success of tobacco excise taxes in reducing tobacco consumption.

A number of studies have found that soda consumption is price-sensitive, with a 10 percent increase in price leading to an 8 percent average reduction in consumption.¹ However, there is limited research on the extent to which increases in soft drink taxes would translate into reduced weight. No such evidence is available for children. A few recent studies found that higher soda taxes are very weakly associated with adolescent and adult weight levels.²⁻⁴

In 2007 twenty-eight states taxed soda at a higher rate than the sales tax on other types of food.⁵ In this paper we estimate the potential effect of taxes on children's consumption and weight by taking advantage of existing variations

in soda sales taxes and sales tax exemptions across states. Does the range of current state-level soda tax rates have a significant effect on consumption patterns and weight gain among children?

There are both practical and substantive reasons why proposals to tax soda or sugar-sweetened beverages are so prominent. Carbonated soft drinks or sugar-sweetened beverages are more easily defined than other categories of snack items, which makes it easier to implement such taxes. Youth have increased their consumption of calories from sugar-sweetened beverages continuously since the 1970s; by now, more than 200 calories daily, or 10 percent of daily energy needs, come from sugar-sweetened beverages. These are calories that otherwise meet no nutritional needs.^{6,7} Soda accounts for most of the consumption of sugar-sweetened beverages.^{6,7}

Reviews of the literature show that consumption of sugar-sweetened beverages is associated with higher energy intake, lower nutrient intake, and increased weight gain or risk for obesity. Even though no single food is responsible for

the energy imbalance, the intake of sugar-sweetened beverages by itself is much higher than the energy imbalance that underlies the obesity epidemic among youth.⁷⁻⁹

High-profile taxes on soda or sweetened beverages have been introduced at the local, state, and federal levels. The City of San Francisco proposed in 2007 to levy a fee on sugar-sweetened beverages to recapture medical care costs attributable to obesity.¹⁰ In 2009, New York State's executive budget proposed an additional 18 percent sales tax on nondiet soft drinks and fruit drinks containing less than 70 percent natural fruit juice.¹¹ The stated goal was that "by increasing the price, [the tax] will discourage individuals, especially children and teenagers, from excessive consumption of these beverages."¹¹ However, the proposal has since been dropped.

In connection with discussions on how to finance health care reform,¹² the U.S. Senate held hearings on soft drink taxes in May 2009. The industry then launched an aggressive national anti-soft drink tax campaign in the summer of 2009.¹³

Study Data And Methods

DATA

► **EARLY CHILDHOOD LONGITUDINAL STUDY—KINDERGARTEN COHORT:** We combined individual-level national data from the Early Childhood Longitudinal Study—Kindergarten Cohort (ECLS-K) with data on state-level grocery store soda tax rates that were in effect during the year in which the longitudinal study data were collected. We examined children's body mass index (BMI, weight in kilograms divided by height in meters squared), total consumption of sugar-sweetened beverages in the past week, and consumption of such beverages at school.

The Early Childhood Longitudinal Study—Kindergarten Cohort is a panel data set of elementary school students that began with a nationally representative cohort of U.S. kindergartners in the fall of 1998 and followed them over time. Data on food consumption were collected in fifth grade (spring 2004) but not in earlier waves, and height and weight were measured by study staff in all waves. This is a distinct advantage of this data set, because most other data sets only have self- or parent-reported height and weight.

The child food consumption questionnaire asked: "During the past seven days, how many times did you drink soda, sports drinks, not 100 percent juice?" There were seven response categories, which we converted into a continuous measure. Children were asked how many times they bought those drinks in the past week

at school, if available. Given that soda consumption accounts for the majority of sugar-sweetened beverage consumption among children,^{6,7} we refer to these drinks as "soda" in the remainder of the paper and in the exhibits, even though the survey question includes other sugar-sweetened beverages.

We have data on soda consumption and soda purchases at school from 7,414 children and data on BMI for 7,300 children. More details on data, methods, and results can be found in the online Appendix.¹⁴

The dependent variables in this analysis were soda consumption in the past week, soda purchases at school, and change in BMI between the third and fifth grades.

Other individual-level control variables included the child's age in months; race and ethnicity indicators, with non-Hispanic white as the reference group; female; a continuous income measure plus additional indicators of family income under \$25,000 and over \$75,000; indicator variables of the mother's education level (less than high school, some college, and college degree, with high school diploma as the reference group); parents' reports of number of times the child engaged in vigorous physical activity per week; weekly television hours; and two scales of parent-child interaction (one about help with homework, the other about how often they talk about school and friends). When analyzing BMI, we also included birth weight.

► **STATE-LEVEL DATA ON SODA TAXES:** Data on state-level sales tax rates for soda purchased through grocery stores came from data collected for the Robert Wood Johnson Foundation-supported Bridging the Gap program. The term "states" includes the fifty states and the District of Columbia. Sales tax rates were compiled from state statutory and administrative laws via primary legal research and were verified by the states.⁵ The sales tax rates we used here were specific to carbonated drinks and did not necessarily apply to other sugar-sweetened beverages such as fruit punch or sports drinks. For purposes of this analysis, taxes on carbonated drinks are referred to as soda taxes.

To match the tax data to the fifth-grade wave of the Early Childhood Longitudinal Study individual-level data collected in the spring of 2004, we used tax rates that were in effect in January 2004. Our primary measures were, first, the difference between taxes on soda versus those on other food sold in grocery stores; and second, an indicator of whether the soda tax rate was greater than the general food tax rate. We considered differences because we wished to estimate the effect of price changes for soda, not the effect of cost-of-living increases where all prices change in the same

way. New soda taxes would change the prices differentially.

STATISTICAL METHODS We used specification tests to find statistical models that best fit the data.¹⁴ For the relationship between taxes and consumption, the best-fitting model is a gamma regression model with a log link; for taxes and BMI, it is ordinary least squares. Because coefficients in nonlinear models are hard to interpret, we show the marginal effect (or the discrete change of a dummy variable from 0 to 1) at the mean in the exhibits. In other words, the numbers show our estimated effect of a one-percentage-point change in the tax rate or a switch from 0 to 1 for a dichotomous variable. A more detailed explanation of the analytic models and methods is included in the online Appendix.¹⁴ The variation in tax rates is cross-sectional (that is, tax differences across states in 2004), even if the individual outcome variable (BMI change) is longitudinal.

STUDY LIMITATIONS One limitation of the study is that the sample size has little statistical power to detect small policy effects, even though there is good statistical power to estimate the effects of individual behavior, such as television watching. The clustered sampling design at the school level further reduces the statistical power.

Another limitation is that the sales tax differentials were for carbonated soft drinks versus other types of food, while the Early Childhood Longitudinal Study questions used for this analysis included carbonated soft drinks and other sugar-sweetened beverages.

Study Results

SUMMARY OF FINDINGS

► **CHILDREN'S SODA CONSUMPTION:** Children report a mean consumption of more than six sodas per week (the median consumption is two sodas per week), with wide variance (Exhibit 1). Fifteen percent of children have zero consumption, 25 percent drink soda daily, and 10 percent consume two or more drinks a day. The much larger mean—three times greater than the median—is a reflection of the fact that there are a small number of children with much greater-than-average consumption.

The average number of soda purchases at school is small. Four-fifths of children buy no soda at school, although the remaining children average three soda purchases per school week.

► **BODY MASS INDEX PERCENTILE:** The average BMI in the sample is situated at the sixty-seventh percentile of the growth charts. Growth charts are based on historical data. This means that in the past, 67 percent of the population had a lower BMI than the average BMI in this data.

This statistic is a good indicator of the obesity problem. If children in the sample had the same weight distribution as the historical data on which the growth charts are based, the average should be around the fiftieth percentile.^{15,16}

The mean increase in BMI between third and fifth grades was 1.9, and the median increase was 1.5. In contrast, the median BMI increase for this age range according to the growth charts should have been 0.4. The change observed indicates a substantial excess weight gain for the children in this study.

► **BEHAVIOR:** Two individual behaviors particularly relevant to obesity are TV watching, a sedentary behavior, and vigorous physical activity. The average TV time for our study was 7.6 hours per week, and the average number of days per week with vigorous activity was 3.8.

► **TAXES:** For the children in the sample, the average tax on soda sold in grocery stores is 4.2 percent; it ranged from 0 to 7 percent. This is, on average, 3.5 percentage points higher than the tax on other types of food. Twenty states have a “differential tax” on soda—that is, a tax on soda greater than the tax on other food items. Sixty-five percent of the children in this sample live in states with differential taxes.

ASSOCIATIONS BETWEEN TAXES, SODA CONSUMPTION, AND BMI Exhibit 2 reports effects of changes in state-level taxes (measured as the difference between sales taxes on soda and on other types of food) for the entire sample on the dependent variables listed: total soda consumption, soda purchases at school, and BMI change. Exhibit 3 repeats the analysis for various subgroups in the sample that are at risk for obesity—namely, at or above the eighty-fifth percentile for BMI (based on growth charts), children from low-income households, children who watch a great deal of TV, and African American children. Exhibit 4 explores the school findings in more depth by limiting the data to children who reported that sugar-sweetened beverages were available at school.

As noted earlier, we were interested in whether a state had a differential tax for soda (see column in exhibits labeled “higher soda tax indicator”), and, if so, by how much (see column labeled “higher soda tax amount.”) We were also interested in the effects these have on the mean values of the dependent variables for both the entire sample (Exhibit 2) and the subgroups (Exhibits 3 and 4).

In Exhibit 2, column 1 (“higher soda tax amount”) shows the effect of a one-percentage-point increase in soda tax (in excess of tax on other food items), and column 2 (“higher soda tax indicator”) shows the effect of implementing the average differential tax rate from our sample.

EXHIBIT 1

Descriptive Statistics For Outcome, Policy, And Child/Family Control Variables, Study Of Childhood Soda Consumption

Variable	Mean/frequency	Standard deviation	Minimum	Maximum
DEPENDENT VARIABLES				
Number of drinks per week	6.1	7.5	0	28
Number of drinks bought at school per week	0.35	1.43	0	20
Body mass index (BMI)	20.5	4.6	7.7	47.3
BMI percentile	67	29	0	99.8
Change in BMI	1.91	1.63	-2.27	7.00
CHILD/FAMILY VARIABLES				
Age (months)	134.4	4.2	111	152
Annual family income (\$ thousands)	60.9	46.2	4	200 (top code)
Weekly hours of TV watching	7.6	4.0	0	31
Parent-child interaction—homework	4.5	3.1	0	11
Parent-child interaction—friends	5.3	0.9	0	6
Number of days per week with more than 20 minutes of vigorous physical activity	3.8	1.9	0	7
Female	49.8%	50	0	1
Family income under \$25,000	21.6%	41	0	1
Family income over \$75,000	35.1%	35	0	1
African American	14.2%	35	0	1
Hispanic	17.5%	38	0	1
Asian	2.3%	15	0	1
Mother has less than high school education	10.2%	30	0	1
Mother has high school diploma	24.3%	43	0	1
Mother has some college education	37.5%	48	0	1
Mother has completed college or higher degree	28.0%	45	0	1
TAX VARIABLES				
Tax on soda if sold through grocery store	4.2%	2.5	0	7
Differential soda—other food tax in grocery stores	3.5%	2.8	0	7
Indicator for higher soda tax	66%	47	0	1

SOURCE Authors' analysis of data from the Early Childhood Longitudinal Study—Kindergarten Cohort. **NOTES** Summary statistics are weighted using wave 6 child weights. Summary statistics for child/family and tax variables are based on N = 7,414 corresponding to the sample for total soda consumption, N = 7,403 for soda consumption at school, N = 7,300 for BMI, and N = 6,866 for BMI change.

Consider the last row of Exhibit 2. This shows that an increase in the differential soda tax by one percentage point is associated with a 0.013 reduction in average BMI, while implementing the average differential tax rate would be associated with a 0.085 reduction in average BMI. Exhibits 3 and 4 may be interpreted similarly for the subgroups shown.¹⁷

► **TAXES AND CONSUMPTION:** As shown in Exhibit 2, there was no significant relationship between differential soda taxes and overall soda consumption for the whole population. This means that, within the limitations of our analysis, increasing the differential tax on soda doesn't affect total soda consumption. We found a significant relationship between differential

EXHIBIT 2

Associations Between Soda Taxes And Outcomes, Marginal Effects

Dependent variable	Higher soda tax amount	Higher soda tax indicator
Total drinks per week	-0.004	-0.006
Drinks bought at school	-0.010	-0.064*
BMI change	-0.013*	-0.085**

SOURCE Authors' analysis of data from the Early Childhood Longitudinal Study—Kindergarten Cohort. **NOTES** Sample sizes are available in the Exhibit 1 Notes. The estimated models are GLM with log link and gamma distribution for soda consumption, identity link and normal distribution (ordinary least squares regression) for BMI. A full version of this exhibit, with additional variables and robust standard errors, is available in the online Appendix, which can be accessed by clicking on the Appendix link in the box to the right of the article online. *p < 0.10 **p < 0.05

EXHIBIT 3

Results For Subgroups At High Risk For Obesity, Marginal Effects

Outcome variable	Total consumption		School consumption		BMI change	
	Higher soda tax amount	Higher soda tax indicator	Higher soda tax amount	Higher soda tax indicator	Higher soda tax amount	Higher soda tax indicator
At risk of overweight (N = 2,917 for total consumption)	-0.026	-0.078	-0.011	-0.067	-0.033**	-0.222**
Family income <\$25,000 (N = 1,371 for total consumption)	-0.142*	-0.811	-0.039**	-0.239**	-0.000	-0.005
African American (N = 701 for total consumption)	-0.125	-0.767	-0.103***	-0.585***	0.029	0.086
9 hours or more of TV watching per week (N = 2,345 for total consumption)	-0.073	-0.376	-0.029**	-0.178**	-0.014	-0.091

SOURCE Authors' analysis of data from the Early Childhood Longitudinal Study—Kindergarten Cohort. **NOTES** Overweight is defined as body mass index at the eighty-fifth percentile or higher. A full version of this exhibit, with robust standard errors, is available in the online Appendix, which can be accessed by clicking on the Appendix link in the box to the right of the article online. **p* < 0.10 ***p* < 0.05 ****p* < 0.01

soda taxes and BMI change from third to fifth grades. But this finding does not hold up under different statistical analysis,¹⁴ and the effect may be attributable to children who are already at risk for being overweight (Exhibit 3).

Exhibit 2 does not show the detailed results for other control variables, which are not of primary interest in this analysis. The most important predictors of total consumption (all significant at the 0.01 level) are increased hours of TV viewing (which leads to increased consumption) and mother with a college education, female, and Asian (which all lead to reduced consumption). The most important predictors of soda purchases at school (all significant at the 0.01 level) are African American and increased hours of TV viewing (both of which lead to increased soda purchases). The most important predictors of a

larger BMI increase are African American and increased hours of TV, whereas higher income, mother with college education, and days with vigorous physical activity predict smaller BMI gains (all significant at the 0.01 level).

► **CHILDREN AT HIGHER RISK:** Because Exhibit 3 deals with much smaller samples (subsets of the entire sample), statistical power is reduced. For the groups shown, higher differential soda taxes are associated with lower total consumption, although not in a way that is statistically significant. For children in low-income families, African Americans, or heavy TV watchers, higher differential taxes predict significantly lower consumption at school. Finally, higher taxes are also associated with significantly lower BMI gain for the heavier children.

► **CHILDREN WITH ACCESS TO SODA AT**

EXHIBIT 4

Results For Subgroups At High Risk For Obesity, Subset Of Students Who Report Availability Of Soda At School

Outcome variable	Total consumption		School consumption	
	Higher soda tax amount	Higher soda tax indicator	Higher soda tax amount	Higher soda tax indicator
All children reporting availability at school	-0.10*	-0.62**	-0.03**	-0.26**
At risk of overweight (N = 1,108 for total consumption)	-0.165**	-1.046**	-0.023	-0.150
Family income <\$25,000 (N = 538 for total consumption)	-0.283**	-1.76**	-0.087**	-0.504**
African American (N = 301 for total consumption)	-0.500***	-2.62***	-0.252***	-1.41***
9 hours or more of TV watching per week (N = 951 for total consumption)	-0.225**	-1.35**	-0.063**	-0.385**

SOURCE Authors' analysis of data from the Early Childhood Longitudinal Study—Kindergarten Cohort. **NOTES** Overweight is defined as body mass index at the eighty-fifth percentile or higher. A full version of this exhibit, with robust standard errors, is available in the online Appendix, which can be accessed by clicking on the Appendix link in the box to the right of the article online. **p* < 0.10 ***p* < 0.05 ****p* < 0.01

SCHOOL: Exhibit 4 shows results only for children who reported that sugar-sweetened beverages were available at school. The results become even stronger, and now differential soda taxes are also predictive of total consumption, indicating the role that schools play in consumption. Reductions in school purchases account for about one-third of the decline in consumption and for a bigger decline among African Americans. There are no significant associations with weight gain, which is therefore not reported in Exhibit 4.

Discussion

This study estimated whether small taxes on soda affect consumption behavior of children and their weight gain. The existing variation in taxes on soda is not very large—up to 7 percent, with a mean differential of 3.5 percent. Many proposals, even those put forward by advocates of “junk food” taxes, call for taxes that are well within the range of existing variation.¹⁸ Similar to the findings from previous studies that linked tax data to individual-level adolescent and adult population data on weight outcomes,^{2,4} our results suggest that such small taxes are unlikely to have measurable effects on soda consumption or obesity among children overall. However, there may be more noticeable effects in population subgroups at higher risk for obesity.

We found statistically significant and substantively larger effects of differential soda sales taxes among children who are heavier, have lower family income, are African American, or watch a great deal of TV. This was particularly pronounced for children for whom sugar-sweetened beverages are available at school. For African Americans, the decline in soda purchases at school associated with any differential tax (1.4 drinks during the school week) accounted for more than half of the decline in total consumption (2.6 drinks).

Overall, the magnitudes are small, which may simply be a consequence of the small tax differentials in place. Larger increases (such as 18 percent, as was under consideration in New York State in 2009) would have larger effects than any existing differential sales tax.

EFFECT OF PLACE OF PURCHASE Most of our statistically significant findings apply to soda purchases at school. One reason this outcome may be more sensitive is that as tax rates rise, posted prices (inclusive of taxes) in cafeterias or vending machines may jump to higher rounded price points. Consequently, the effect of a percentage sales tax may be higher than at grocery stores, where the tax is applied at the cash register. This would affect lower-income groups

Among children at higher risk for obesity, even taxes in the range of current rates can affect outcomes.

more. Previous studies for other types of food have reported larger price effects on BMI among heavier and lower-income children and youths.¹⁹⁻²¹

Several more of our results would have been significant at the 5 percent level without the clustered sampling design effect described above. However, this would not change our conclusion that for the full population, the range of variation in taxes does not predict total consumption or BMI. Larger taxes could have more pronounced effects at the population level. Among children at higher risk for obesity, however, even taxes in the range of current rates can affect outcomes.

SODA TAX AMOUNTS The range of existing soda tax rates is relatively small. That may arguably be the relevant comparison, as new taxes are likely to be relatively small. In that case, we should not expect noticeable behavior or weight changes for children in the general population. A greater impact of these small taxes could come from the dedication of the revenues they generate to other obesity prevention efforts rather than through their direct impact on children’s consumption of soda.

On the other hand, the combination of a continuing obesity epidemic and states’ financial difficulties in the economic downturn may lead to much larger changes. The 18 percent soda tax rate originally proposed in New York’s Executive Budget is much larger than existing tax rates. Our estimated marginal effect of differential taxes on BMI increases between third and fifth grades is -0.013 BMI units at the population level. If effects were linear, an 18 percent differential soda tax would correspond to -0.23 BMI units, or a 20 percent reduction of the excess BMI gain. No other anti-obesity policy has demonstrated a reduction of that magnitude yet, so our results do not imply that excise taxes would be ineffective at the population level—only that small taxes in the range of existing differentials

are unlikely to have visible effects at the population level.

The economic theory of the design and effects of taxes is fairly clear, although the empirical evidence is limited and estimates cover a wide range.¹ If reducing the consumption of sugar-sweetened beverages is the goal, rather than collecting money, taxes need to be linked to consumption. An approach such as that considered in San Francisco, which would collect a fixed annual fee from retailers in order to sell sugar-sweetened beverages, fails that criterion.

EXCISE VERSUS SALES TAXES A specific excise tax would be preferable to a sales tax. A tax levied per ounce would be easiest to implement, although it is possible, but more complicated, to levy a tax based on sugar content. The latter will encourage substitution to cheaper, larger-

volume products, rather than a reduction in consumption. Also, an excise tax is preferable to a sales tax because it would be incorporated into the shelf price, making the higher costs more visible to consumers.

CONCLUDING COMMENTS Efforts to reduce obesity are accelerating, and a common target is reducing the consumption of sugar-sweetened beverages. For youth, initiatives so far have sought to limit the sale of soda in schools, but schools are only one source of consumption. We can expect that many localities will implement taxes on a variety of foods deemed “junk” foods, most likely starting with sugar-sweetened beverages, in the near future. To have a measurable effect on consumption, taxes need to be tied to consumption, and they need to be larger than the existing state variation in sales taxes. ■

The research for this paper was funded by the National Institute of Child Health and Human Development (Grant no. R01HD057193); the National Heart,

Lung, and Blood Institute (Grant no. R01HL096664); and the Robert Wood Johnson Foundation through Bridging the Gap's ImpacTeen project. The

content is solely the responsibility of the authors and does not necessarily represent the official views of the funders. [Published online 1 April 2010.]


NOTES

- 1 Andreyeva T, Long M, Brownell KD. The impact of food prices on consumption: a systematic review of research on price elasticity of demand for food. *Am J Public Health*. 2010;100(2):216–22.
- 2 Powell LM, Chriqui JF, Chaloupka FJ. Associations between state-level soda taxes and adolescent body mass index. *J Adolesc Health*. 2009;45: S57–63.
- 3 Kim D, Kawachi I. Food taxation and pricing strategies to thin out the obesity epidemic. *Am J Prev Med*. 2006;30(5):430–7.
- 4 Fletcher JM, Frisvold D, Tefft N. Can soft drink taxes reduce population weight? *Contemp Econ Policy*. 2010;28(1):23–35.
- 5 Chriqui JF, Eidson SS, Bates H, Kowalczyk S, Chaloupka FJ. State sales tax rates for soft drinks and snacks sold through grocery stores and vending machines, 2007. *J Public Health Policy*. 2008;29: 226–49.
- 6 Nielsen SJ, Popkin BM. Changes in beverage intake between 1977 and 2001. *Am J Prev Med*. 2004;27: 205–10.
- 7 Wang YC, Bleich SN, Gortmaker SL. Increasing caloric contribution from sugar-sweetened beverages and 100% fruit juices among U.S. children and adolescents, 1988–2004. *Pediatrics*. 2008;121:e1604–14.
- 8 Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systematic review. *Am J Clin Nutr*. 2006;84:274–88.
- 9 Vartanian LR, Schwartz MB, Brownell KD. Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis. *Am J Public Health*. 2007; 97:667–75.
- 10 McKinley J. San Francisco's mayor proposes fee on sales of sugary soft drinks. *New York Times*. 2007 Dec 18.
- 11 State of New York. *New York State, 2009–2010 Executive Budget, briefing book, revenue actions*. Albany (NY): State of New York; 2008 Dec 16.
- 12 Adamy J. Soda tax weighed to pay for health care. *Wall Street Journal*. 2009 May 12; A4.
- 13 Americans Against Food Taxes [home page on the Internet]. Available from: <http://www.nofoodtaxes.com/ads>
- 14 The online Appendix can be accessed by clicking the Appendix link in the box to the right of the article online.
- 15 Centers for Disease Control and Prevention. CDC growth charts [Internet]. Atlanta (GA): CDC; [cited 2010 Mar 17]. Available from: <http://www.cdc.gov/GrowthCharts/>
- 16 Even though height and weight were measured by study staff, four observations had a BMI of under 10, which is not credible and likely to reflect measurement or recording errors. For change in BMI, we dropped the top and bottom 1 percent values to avoid the influence of gross measurement errors.
- 17 The multivariate regression models include the following control variables (unless they are dropped for collinearity in the subgroup analyses): age, female, a continuous income measure, indicators of family income (less than \$25,000 or more than \$75,000), three race/ethnicity dummies (African American, Hispanic, Asian), three maternal education dummies (less than high school, some college, completed college), days of vigorous physical activity, weekly hours of TV watching, and two scales of parent-child interaction. The BMI change regression also included birth weight.
- 18 Jacobson MF, Brownell KD. Small taxes on soft drinks and snack foods to promote health. *Am J Public Health*. 2000;90:854–7.
- 19 Powell LM, Bao Y. Food prices, access to food outlets, and child weight. *Econ Hum Biol*. 2009;7: 64–72.
- 20 Sturm R, Datar A. Body mass index in elementary school children, metropolitan area food prices, and food outlet density. *Public Health*. 2005; 119:1059–68.
- 21 Sturm R, Datar A. Food prices and weight gain during elementary school: five-year update. *Public Health*. 2008;122(11):1140–3.

Bodily Effects of Sugar

Circulation

JOURNAL OF THE AMERICAN HEART ASSOCIATION

American Heart
Association® 
Learn and Live™

Sugar-Sweetened Beverages, Obesity, Type 2 Diabetes Mellitus, and Cardiovascular Disease Risk

Vasanti S. Malik, Barry M. Popkin, George A. Bray, Jean-Pierre Després and Frank
B. Hu

Circulation 2010;121:1356-1364

DOI: 10.1161/CIRCULATIONAHA.109.876185

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX
72514

Copyright © 2010 American Heart Association. All rights reserved. Print ISSN: 0009-7322. Online
ISSN: 1524-4539

The online version of this article, along with updated information and services, is
located on the World Wide Web at:

<http://circ.ahajournals.org/cgi/content/full/121/11/1356>

Subscriptions: Information about subscribing to *Circulation* is online at
<http://circ.ahajournals.org/subscriptions/>

Permissions: Permissions & Rights Desk, Lippincott Williams & Wilkins, a division of Wolters
Kluwer Health, 351 West Camden Street, Baltimore, MD 21202-2436. Phone: 410-528-4050. Fax:
410-528-8550. E-mail:
journalpermissions@lww.com

Reprints: Information about reprints can be found online at
<http://www.lww.com/reprints>

Sugar-Sweetened Beverages, Obesity, Type 2 Diabetes Mellitus, and Cardiovascular Disease Risk

Vasanti S. Malik, MSc; Barry M. Popkin, PhD; George A. Bray, MD;
Jean-Pierre Després, PhD; Frank B. Hu, MD, PhD

Obesity has recently emerged as a major global health problem. According to World Health Organization estimates, ≈ 1.6 billion adults worldwide were overweight (body mass index [BMI] ≥ 25 kg/m²) and at least 400 million were obese (BMI ≥ 30 kg/m²) in 2005, numbers that are expected to reach 2.3 billion and 700 million, respectively, by 2015. In the United States, the percentage of overweight and obese adults increased markedly from 47% and 15% in 1976 to 1980 to $>66\%$ and 33% in 2005 to 2006, with the greatest proportion of increase seen among non-Hispanic black and Mexican American women.^{1,2} The implications of excess body weight are far-reaching. Epidemiological studies indicate that overweight and obesity are important risk factors for type 2 diabetes mellitus (T2DM), cardiovascular disease, cancer, and premature death.³ In the United States, healthcare expenditures attributable to overweight and obesity are estimated to be \$147 billion or 9.1% of total healthcare costs per year.⁴ Such excess costs could have serious repercussions for resource-poor countries, which must manage the dual burdens of chronic and infectious disease.

In the setting of a pandemic of obesity and related chronic diseases, the American Heart Association recently released a scientific statement recommending reductions in added-sugar intake to no more than 100 to 150 kcal/d for most Americans.⁵ The statement identified sugar-sweetened beverages (SSBs) as the primary source of added sugars in the American diet.⁶ Although it has long been suspected that SSBs contribute at least in part to the obesity epidemic, only in recent years have large epidemiological studies been able to substantiate the relationship between SSB consumption and long-term weight gain, T2DM, and cardiovascular risk. It is thought that SSBs contribute to weight gain because of their high added-sugar content, low satiety, and potential incomplete compensation for total energy, leading to increased energy intake.^{7,8} In addition, because of their high amounts of rapidly absorbable carbohydrates such as various forms of sugar and high-fructose corn syrup (HFCS) and the large quantities consumed, SSBs may increase T2DM and cardiovascular risk independently of obesity as a contributor to a

high dietary glycemic load (GL), leading to inflammation, insulin resistance, and impaired β -cell function.⁹ Fructose from any sugar or HFCS may also increase blood pressure and promote the accumulation of visceral adiposity, dyslipidemia, and ectopic fat deposition because of increased hepatic de novo lipogenesis.¹⁰ Here, we review temporal patterns in SSB consumption and clinically relevant effects on obesity, T2DM, and cardiovascular disease risk, emphasizing potential underlying biological mechanisms, clinical implications, and consideration of methodological issues inherent in the literature.

SSB Global Pattern

Although carbonated beverages trace their history back to the 1760s when carbonation techniques were developed to reproduce naturally occurring carbonated mineral waters believed to be healthy, these beverages did not add sugar.¹¹ A century later, one of the most pivotal events in soft drink history occurred when Atlanta pharmacist J.S. Pemberton combined kola, a caffeinated nut from Africa, with coca, a stimulant from South America, to create Coca-Cola, which, like most other sweetened beverages developed in the 1800s, was marketed as a tonic.¹² In about 1904, Asa Candler purchased legal rights to the formula from Pemberton and soon developed the first mass factory.¹³ During World War II, Coca-Cola worked closely with the US Department of War to provide free Cokes to army soldiers. As a result of a lobbying campaign, they were allowed to break sugar ration rules and to create Coke plants in European countries with the support of the government, ultimately becoming synonymous globally with SSBs.¹³

During the past 30 years, there has been a marked increase in the consumption of SSBs across the globe. For instance, in the United States, intake of these beverages, which includes the full range of soft drinks, fruit drinks, energy drinks, and vitamin water drinks, increased from 3.9% of calories in the late 1970s to 9.2% in 2001, representing a 3-fold increase in intake.¹⁴ In other countries, there have been varying levels of increase in SSBs, with some countries such as Mexico reaching such magnitudes that serious government interven-

From the Departments of Nutrition and Epidemiology, Harvard School of Public Health (V.S.M., F.B.H.), and Channing Laboratory, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School (F.B.H.), Boston, Mass; Centre de Recherche de l'Institut Universitaire de Cardiologie et de Pneumologie de Québec, Québec City, Québec, Canada (J.P.D.); Dietary Obesity Laboratory, Pennington Biomedical Research Center, Baton Rouge, La (G.A.B.); and Department of Nutrition, Gillings School of Global Public Health, University of North Carolina, Chapel Hill (B.M.P.).

Correspondence to Frank B. Hu, Department of Nutrition, Harvard School of Public Health, 665 Huntington Ave, Boston, MA 02115. E-mail frank.hu@channing.harvard.edu

(*Circulation*. 2010;121:1356-1364.)

© 2010 American Heart Association, Inc.

Circulation is available at <http://circ.ahajournals.org>

DOI: 10.1161/CIRCULATIONAHA.109.876185

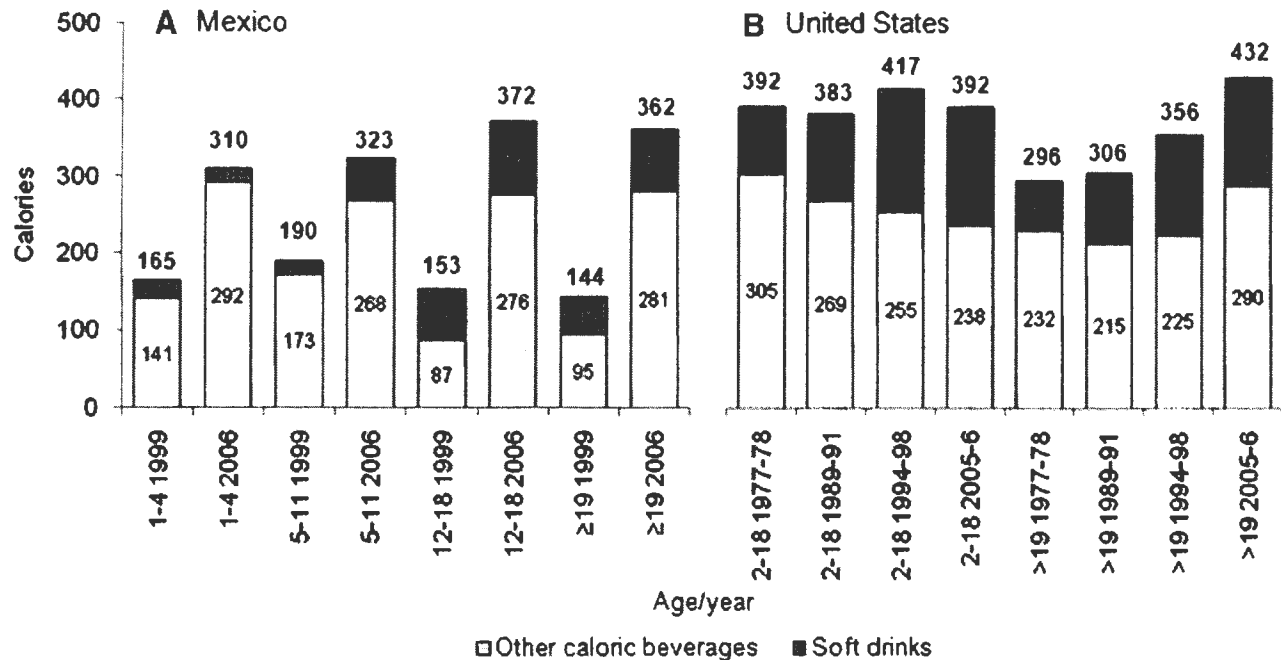


Figure 1. Trends in consumption of calories from soft drinks and all caloric beverages in Mexico and the United States (weighted to be nationally representative) by age groups (1 to 4, 5 to 11, 12 to 18, and ≥ 19 years) in 1999 and 2006. Soft drinks include carbonated, noncarbonated beverages with sugar added, and commercially processed, bottled/formula fountain soft drinks and fruit drinks but exclude agua frescas, Mexican hand-prepared added-sugar fruit juices, and fruit drinks.

tion to reduce intake is being undertaken.¹⁵ Nation-level food disappearance data from China, India, Vietnam, Thailand, and other South Asian countries also show rapid trajectories of an increase in SSB intake, as well as large per capita consumption across the Americas, Germany, Australia, Spain, and Great Britain.¹⁶ The most rigorous sources of nationally representative patterns in SSB intake come from the United States and Mexico, where large-scale dietary intake surveys have been repeated in the last decade.^{15,17} According to these data, all age groups in Mexico consume $\approx 10\%$ of their total energy intake from SSBs. As shown in Figure 1A, SSB intake has increased considerably among those ≥ 5 years of age in Mexico. Figure 1B presents the same data for the United States. As seen in both children 2 to 18 years of age and adults >19 years of age, substantial increases across each decade have continued.

SSB and Childhood Obesity

Childhood obesity is known to increase risk of obesity in adulthood and can lead to serious consequences for T2DM and cardiovascular disease risk later in life. In fact, recent evidence suggests consideration of lipid screening for children with BMI beginning at the 80th percentile rather than ≥ 85 th percentile, the point at which a child is considered overweight.¹⁸

Given the preponderance of SSB consumption among children and adolescents, several epidemiological studies have examined the relationship between SSB and weight gain or obesity in this group. Recently, we conducted a meta-analysis evaluating change in BMI per increase in 1 serving of SSB per day and found a significant positive association between SSB intake and weight gain (0.08; 95% confidence interval [CI], 0.03 to 0.13 kg)¹⁹ among studies that did not adjust for total energy intake.^{20–24} Because the association

between SSB intake and weight gain is partially mediated by total energy intake, adjusting for energy is expected to attenuate this effect. In these data, the effect was also strongest in larger studies with longer durations of follow-up that used robust dietary assessment methods such as food frequency questionnaires rather than a single 24-hour diet recall, which is not able to capture patterns in dietary intake.^{20,21} These results are supported by previous systematic reviews and meta-analyses,^{25–27} as well as more recent studies. For example, Dubois and colleagues²⁸ found that in >2000 children 2.5 years of age followed up for 3 years, regular consumers of SSBs between meals had a 2.4-fold greater odds of being overweight compared with nonconsumers ($P < 0.05$). Another study conducted among 5-year-old subjects in the UK with 4 years of follow-up did not find an association between SSB intake and fatness, possibly because intake levels were too low.²⁹ Recent studies have also shown that greater SSB consumption in childhood or adolescence predicted weight gain into adulthood.^{30,31}

Findings from intervention studies, which are few in number, generally support those from well-powered prospective cohort studies and show positive associations between SSB intake and weight gain either in the overall study²² or in subgroup analyses among participants overweight at baseline.^{24,32} A follow-up analysis of a school-based intervention that showed that reducing SSB intake decreased overweight and obesity²² did not see an effect 2 years after the intervention had been discontinued, which supports an effect of SSB consumption on weight gain.³³

SSB Consumption and Weight Gain in Adults

To date, a large number of studies have evaluated the relationship between SSB consumption and weight gain or

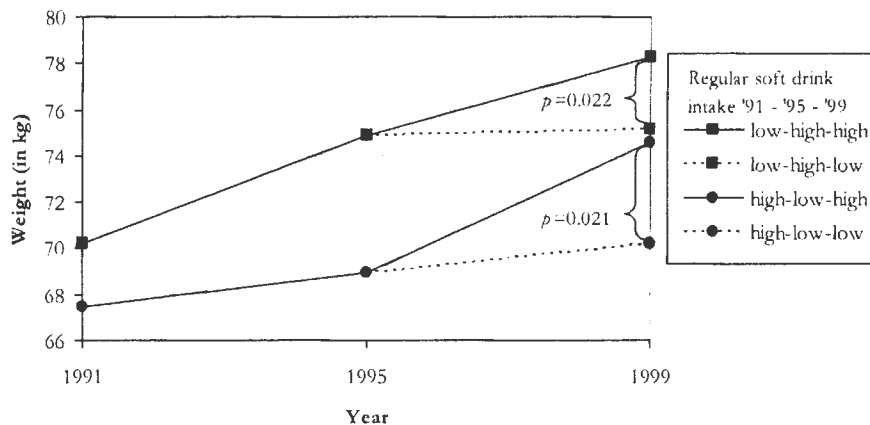


Figure 2. Low and high intakes were defined as ≤ 1 /week and ≥ 1 /day. The numbers of subjects were as follows: low-high-high=323, low-high-low=461, high-low-high=110, and high-low-low=746. Groups with similar intake in 1991 and 1995 were combined for estimates for these time points. Means were adjusted for age, alcohol intake, physical activity, smoking, postmenopausal hormone use, oral contraceptive use, cereal fiber intake, and total fat intake at each time point. Adapted with permission from Schulze et al.³⁴

risk of overweight and obesity among adults. However, differences in study design, methodologies, and data quality have made it difficult to observe a consistent effect. Cross-sectional studies are not optimal because of the high potential for intractable confounding and reverse causation. Experimental studies are not well suited to capture long-term patterns because compliance tends to wane with increasing duration, but they do provide important insight into potential underlying biological mechanisms. Prospective cohort studies tend to provide the most robust evidence despite a large degree of diversity between studies in terms of outcome measurements, size, and duration of follow-up. Therefore, greater emphasis should be placed on larger studies of longer duration that are better powered to detect an effect. In this literature, the longest and largest studies^{34,35} show stronger and more consistent associations compared with smaller and shorter studies.^{36,37} For example, in the study by Schulze et al³⁴ of >50 000 nurses followed up for two 4-year periods (1991 to 1995 and 1995 to 1999), a higher consumption of SSBs was associated with a greater magnitude of weight gain. After adjustment for potential confounders, women who increased their SSB consumption from 1991 to 1995 and maintained a high level of intake gained on average 8.0 kg during the 2 periods, whereas women who decreased their SSB intake between 1991 and 1995 and maintained a low level of intake gained on average 2.8 kg during the 2 periods (Figure 2). Similar results were reported by Palmer and colleagues³⁵ in >40 000 black women followed up for 6 years. Those whose SSB intake increased from ≤ 1 serving per week to ≥ 1 serving per day gained the most weight, whereas those who decreased their intake gained the least weight (6.8 and 4.1 kg, respectively) after adjustment for potential confounders. A smaller study from Spain³⁸ with >7000 participants followed up for ≈ 2 years found that a higher consumption of SSB was associated with significant weight gain among subjects who gained ≥ 3 kg in the 5 years before baseline. These participants had a higher absolute intake of SSB at baseline compared with participants with no previous weight gain, consistent with a positive association between SSB intake and weight gain. It is possible that the study was not long enough to evaluate weight gain in relation to SSB intake in subjects with no previous weight gain. In a large cohort from Germany ($n=17\ 369$) with a similar duration of follow-up, SSB intake was associated with weight

gain in men but not women.³⁹ In the Framingham Offspring Study⁴⁰ with an average duration of 4 years and >4000 participants, compared with infrequent consumers, participants who consumed ≥ 1 soft drinks per day had a 37% higher risk of obesity.⁴⁰ Because this analysis included both diet and regular soft drinks, it is difficult to disentangle the independent effect of SSBs because consumers of diet soft drinks may be weight conscious or trying to lose weight. In an observational analysis of the Clinical Trial of Comprehensive Lifestyle Modification for Blood Pressure Control (PREMIER) study ($n=810$), Chen et al⁴¹ found that a reduction in SSB intake of 1 serving per day was associated with a weight loss of 0.49 kg (95% CI, 0.11 to 0.82; $P=0.006$) at 6 months and of 0.65 kg (95% CI, 0.22 to 1.09; $P=0.003$) at 18 months. However, participants in this study were part of a trial to lower blood pressure and had higher baseline BMI than other cohorts and stage 1 hypertension, which could partly explain why such a strong effect was seen with relatively little power. At the same time, because this study adjusted for total energy, the effect of SSBs on weight gain may have been underestimated.

SSB Consumption and T2DM and the Metabolic Syndrome

Similar to the weight-gain literature, prospective cohort studies evaluating the effect of SSBs on risk of T2DM and the metabolic syndrome (MetSyn) have found the strongest and most consistent associations in large studies with long durations of follow-up. These aspects of study design are particularly important when assessing diet in relation to chronic disease origin because sufficient time is required for causal action and disease initiation and detection to occur. In >50 000 women followed up for 8 years, after adjustment for potential confounders, those consuming ≥ 1 SSBs per day had an 83% greater risk of developing T2DM compared with those consuming < 1 SSB per month (relative risk [RR], 1.83; 95% CI, 1.42 to 2.36; $P<0.001$ for trend; Figure 3).³⁴ The RR comparing extreme categories further controlling for BMI was 1.41 (95% CI, 1.09 to 1.83; $P<0.001$ for trend). This finding suggests that BMI accounts for about half of the excess risk. Similarly, in the Black Women's Health Study,³⁵ with >40 000 women followed up for 10 years, those who consumed ≥ 2 SSBs per day had a 24% greater risk of

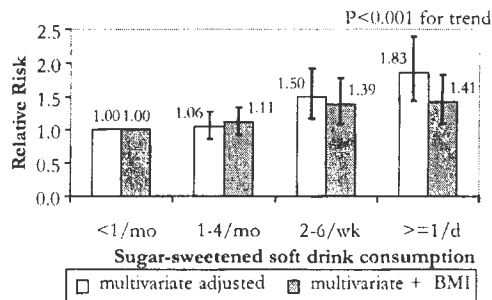


Figure 3. Multivariate RRs were adjusted for age, alcohol (0, 0.1 to 4.9, 5.0 to 9.9, ≥ 10 g/d), physical activity (quintiles), family history of diabetes mellitus, smoking (never, past, current), postmenopausal hormone use (never, ever), oral contraceptive use (never, past, current), intake (quintiles) of cereal fiber, magnesium, *trans* fat, polyunsaturated:saturated fat, and consumption of sugar-sweetened soft drinks, diet soft drinks, fruit juice, and fruit punch (other than the main exposure, depending on model). These data are based on data from Schulze et al.³⁴

developing T2DM compared with those who consumed <1 SSB per month (RR, 1.24; 95% CI, 1.06 to 1.45; $P=0.002$ for trend). After additional adjustment for BMI, the RR was no longer statistically significant, suggesting that in this population the majority of effect was mediated by BMI. Findings from these studies were replicated in a large cohort of $>70\,000$ women followed up for 18 years that showed that women who consumed 2 to 3 SSBs per day had a 31% greater risk of developing T2DM than women who consumed <1 SSB per month (RR, 1.31; 95% CI, 0.99 to 1.74; $P<0.01$ for trend).⁴² Because this study adjusted for BMI and total energy intake, both potential mediators of effect, the association between SSB intake and T2DM risk may actually be underestimated. In contrast to these studies, findings from the Atherosclerosis Risk in Communities Study ($n=12\,204$) did not show a consistent association between SSB intake and incidence of T2DM after 9 years of follow-up (men: RR, 1.09; 95% CI, 0.89 to 1.33; women: RR, 1.17; 95% CI, 0.94 to 1.46).⁴³ Compared with the study by Schulze et al.,³⁴ participants in this study were older (53.6 versus 36.1 years of age) and heavier (27.2 versus 24.6 kg/m²) at baseline. Because the effect of SSB on T2DM is mediated in part by BMI, once BMI is increased, it is possible that the additional effect of continued SSB intake is diminished; however, further research is needed to confirm this hypothesis.

Few studies have examined the effect of SSBs on the development of MetSyn, but they are in line with findings from studies evaluating T2DM. For example, findings from the Framingham Offspring Study ($n=6154$) showed that compared with nonconsumers, individuals who consumed ≥ 1 soft drinks per day had a 39% greater risk of developing MetSyn during the course of 4 years.⁴⁰ Although this analysis combined diet and regular soft drinks, it can be assumed that the majority of this effect was due to regular soft drink consumption. Other studies of MetSyn have found marginal effects of SSBs, but because they adjusted for total energy intake, the results may have been underestimated.^{44,45}

SSB Consumption and Cardiovascular Risk

The evidence relating SSB intake to cardiovascular risk is limited, although data are starting to accumulate that suggest

that greater SSB consumption may have a role in the development of hypertension, adverse lipid parameters, inflammation, and clinical coronary heart disease (CHD). The Framingham Offspring Study, which also looked at SSB intake in the context of MetSyn components in 6154 adults followed up for 4 years, found that individuals who consumed ≥ 1 soft drink per day had a 22% higher incidence of hypertension ($\geq 135/85$ mm Hg or on treatment) compared with nonconsumers (RR, 1.22; 95% CI, 1.05 to 1.41).⁴⁰ Similarly, in the Nurses' Health Studies I and II, women who consumed ≥ 4 SSBs per day had a 44% and 28% higher risk of incident hypertension, respectively, compared with infrequent consumers (RR, 1.44; 95% CI, 0.98 to 2.11; and RR, 1.28; 95% CI, 1.01 to 1.62, respectively).⁴⁶ Regarding lipids, daily soft drink consumers in the Framingham Offspring Study were found to have a 22% higher incidence of hypertriglyceridemia (≥ 1.7 mmol/L or on treatment) and low high-density lipoprotein cholesterol (<1.03 mmol/L for men or <1.3 mmol/L for women or on treatment) compared with nonconsumers (RR, 1.22; 95% CI, 1.07 to 1.41; and RR, 1.22; 95% CI, 1.04 to 1.44, respectively).⁴⁰ Results from the Multi-Ethnic Study of Atherosclerosis (MESA) study, which had fewer participants ($n=3878$), showed a significant effect of SSBs on hypertriglyceridemia (≥ 1.7 mmol/L or on treatment) and trends toward an effect on hypertension ($\geq 130/85$ mm Hg or on treatment) and low high-density lipoprotein cholesterol (<1.03 mmol/L for men or <1.3 mmol/L for women or on treatment) in daily SSB consumers compared with nonconsumers (RR, 1.28; 95% CI, 1.02 to 1.60; RR, 1.14; 95% CI, 0.91 to 1.43; and RR, 1.28; 95% CI, 0.99 to 1.64, respectively).⁴⁵ These findings are supported by a recent cross-sectional analysis of National Health and Nutrition Examination Survey data that found a positive association between SSB intake and blood pressure in adolescents.⁴⁷ A 10-week intervention study comparing the effects of sucrose- and artificially sweetened foods/beverages on markers of inflammation found that serum levels of haptoglobin, transferrin, and C-reactive protein increased in the sucrose group and decreased in the sweetener group (between-group differences: $P=0.006$, $P=0.01$, and $P=0.1$, respectively).⁴⁸ Indirect evidence for an effect of SSBs on inflammation also stems from observational studies that have found positive associations between dietary patterns that are high in SSBs with markers of inflammation such as C-reactive protein and tumor necrosis factor receptor 2⁴⁹ and dietary GL, to which SSB intake is a large contributor with C-reactive protein.⁵⁰ In addition, higher consumption of soft drinks has been associated with hyperuricemia⁵¹ and incidence of developing gout,⁵² a condition commonly associated with insulin resistance and MetSyn.

Recently, in the Nurses Health Study, a positive association between SSB intake and risk of CHD (nonfatal myocardial infarction or fatal CHD) was observed even after accounting for other unhealthy factors.⁵³ In $>88\,000$ women followed up for 24 years, those who consumed ≥ 2 SSBs per day had a 35% greater risk of developing CHD compared with those who consumed <1 SSB per month (RR, 1.35; 95% CI, 1.1 to 1.7; $P<0.001$ for trend). Additional adjustment for BMI, energy intake, and incident T2DM attenuated the

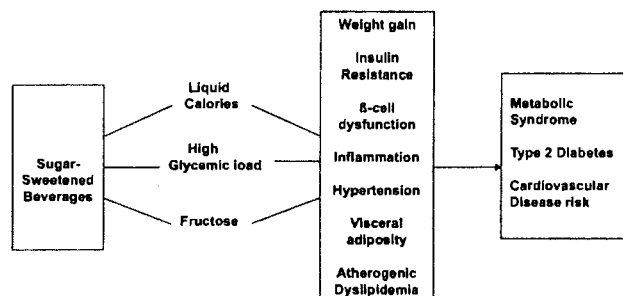


Figure 4. SSBs may lead to weight gain as a result of incomplete compensation for liquid calories at subsequent meals, resulting in positive energy balance. Independently of weight gain, SSBs may increase the risk of MetSyn, T2DM, and cardiovascular disease because of their large contribution to a high dietary GL and large fructose fraction, leading to the development of insulin resistance, β -cell dysfunction, inflammation, hypertension, visceral adiposity, and atherogenic dyslipidemia.

associations, but they remained statistically significant, suggesting that the effect of SSBs on CHD is not entirely mediated by these factors.

Potential Mechanisms

The prevailing mechanisms linking SSB intake to weight gain are decreased satiety and incomplete compensatory reduction in energy intake at subsequent meals after consumption of liquid calories (Figure 4).^{7,8} On average, SSBs contain 140 to 150 calories and 35 to 37.5 g sugar per 12-oz serving. If normal dietary intake does not decrease by an equivalent amount of calories per serving, then weight gain is expected.^{25,54} This has been illustrated in short-term feeding trials showing greater energy intake and weight gain after consumption of calorically sweetened beverages (sugar, sucrose, HFCS) compared with noncaloric artificially sweetened beverages.^{55,56} In addition, a number of studies have shown greater energy intake and weight gain after isocaloric consumption of beverages as opposed to solid food.⁷ These studies argue that sugar or HFCS in liquid beverages may not suppress intake of solid foods to the level needed to maintain energy balance; however, the mechanism responsible for that weaker compensatory response to fluids is unknown.⁵⁷

SSBs may contribute to T2DM and cardiovascular risk in part by their ability to induce weight gain, but an independent effect may also stem from the high amounts of rapidly absorbable carbohydrates such as any form of sugar or HFCS, the primary sweeteners used in SSBs (Figure 4). Consumption of SSBs has been shown to result in rapid and dramatic increases in blood glucose and insulin concentrations,⁵⁸ which, in conjunction with the large quantities that are often consumed, contribute to a high dietary GL. High-GL diets are thought to stimulate appetite and to promote weight gain and have been shown to induce glucose intolerance and insulin resistance.⁵⁹ An increase in GL has also been shown to exacerbate levels of inflammatory biomarkers such as C-reactive protein linked to T2DM and cardiovascular disease risk.⁵⁰ Inflammation is known to influence atherosclerosis, plaque stability, and thrombosis; therefore, SSB consumption may affect CHD risk within just a few years.⁵³ High dietary GL has also been associated with greater risk of

CHD.⁶⁰ In addition, the caramel coloring used in cola-type soft drinks is high in advanced glycation end products, which may further increase insulin resistance and inflammation.⁶¹ For instance, an 8-oz serving of cola delivers 16.3 kU advanced glycation end products.⁶²

Recent evidence also suggests that consuming fructose, which is found in similar amounts in sucrose and HFCS, may have particularly adverse effects on selective deposition of visceral and ectopic fat, lipid metabolism, de novo lipogenesis, blood pressure, and insulin sensitivity compared with glucose¹⁰ (Figure 4). The different pathways for the metabolism of fructose and glucose are clearly important potential mechanisms. Fructose alone is poorly absorbed but is enhanced by glucose in the gut, thus accounting for the rapid and complete absorption of both fructose and glucose when ingested as sucrose or HFCS. Studies in humans and animals have shown that fructose is preferentially metabolized to lipid in the liver, leading to increased triglyceride levels, which have been associated with the development of insulin resistance and cardiovascular disease.^{63–66} A recent study in overweight adults compared the effects of consuming glucose- or fructose-sweetened beverages providing 25% of energy requirements.¹⁰ After 10 weeks, both groups showed similar weight gain; however, only the fructose group showed a significant increase in visceral adiposity, which has also been observed in a number of recent studies.^{67–69} Although fasting plasma triglyceride levels increased only in the glucose group, hepatic de novo lipogenesis, postprandial triglycerides, and markers of altered lipid metabolism and lipoprotein remodeling such as fasting apolipoprotein B and small low-density lipoprotein particles significantly increased in the fructose group. In addition, fasting plasma glucose and insulin levels increased and insulin sensitivity decreased in the fructose group. Of interest, Ghanim and colleagues⁷⁰ did not find evidence of oxidative or inflammatory stress after intake of 300 kcal fructose or orange juice, whereas reactive oxygen species generation and nuclear factor- κ B binding were significantly increased after intake of glucose. However, the quantities of fructose contained in SSBs are far greater than those contained in these preloads.⁷⁰ Fructose can also increase blood uric acid concentrations.⁷¹ The production of uric acid in the liver by xanthine oxidase may reduce endothelial nitric oxide,⁷² which could partly mediate the association between SSBs and risk of CHD. Increases in blood pressure have also been observed when fructose is administered acutely, an effect not seen with glucose.⁷³ In addition, an increase in blood pressure spanning 10 weeks was found when individuals drank SSBs but not aspartame-sweetened beverages.⁵⁵ Fructose intake may also lead to weight gain by decreasing the production of insulin and leptin in peripheral tissues, thereby initiating the hunger cascade in the central nervous system⁶⁴; this area warrants further investigation because others have found greater satiety and lower total energy intake after fructose preloads compared with glucose preloads.⁷⁴

Clinical Implications

Controlling the intake of SSBs represents an important component of lifestyle management for weight control and

maintenance. Limiting SSBs may also confer favorable benefits on T2DM and cardiovascular risk such as improving lipid profiles and insulin sensitivity and reducing blood pressure, inflammation, and accumulation of visceral adiposity. The excess risk imparted by SSBs may have particular relevance for certain individuals or populations who are more susceptible to developing T2DM.⁷⁵ Limiting SSB intake among children and adolescents is imperative because overweight and obesity are rampant in this population, which can have serious downstream effects on cardiovascular health. Public policy approaches such as taxation have been proposed to reduce SSB consumption in the general population.⁷⁶

When SSBs are replaced with other beverages, it is important to select alternatives that are healthy and do not promote weight gain. The average individual needs at least 1 mL fluid for every calorie burned, which is approximately eight 8-oz glasses per day for a 2000-kcal diet.⁷⁷ Adequate hydration is essential for maintaining blood volume and kidney function and preventing constipation.⁷⁸ Water has no calories or additives and is widely available, inexpensive, and generally safe. Findings from epidemiological studies show that energy intake is significantly lower ($\approx 9\%$, or 194 kcal/d) in water drinkers compared with non-water drinkers,⁷⁹ which was supported by a recent randomized controlled trial in German schoolchildren.⁸⁰ This study found that 1 year of water intake was linked with a 31% reduction in the risk of being overweight.⁸⁰ Unlike SSBs, water does not contain liquid calories to be compensated for at subsequent meals. As shown in secondary analysis of a clinical weight loss trial, replacing SSBs with water was associated with lower total energy intake (predicted mean decrease of 200 kcal/d spanning 12 months).⁸¹ In addition, some evidence indicates that consuming water before or with a meal reduces feelings of hunger and increases satiety,^{79,82} in contrast to both diet and regular soft drinks, which are thought to stimulate appetite by their intense sweet flavor.^{83,84} Coffee and tea are also reasonable alternatives provided that caloric sweeteners and whitening agents are used sparingly. A number of studies have shown that regular consumption of coffee and tea can have favorable benefits on T2DM and cardiovascular disease risk, possibly by virtue of their polyphenol content.^{85,86} In recent decades, consumption of milk has decreased markedly in the United States. Displacement by SSBs in the pediatric population is of great concern because it can lead to lower intakes of protein, calcium, magnesium, zinc, vitamin A, and vitamin D and increase the risk for osteoporosis and bone fracture.^{14,87} Because of the excess calories and saturated fat content of whole milk, low-fat milk is recommended but should be consumed in moderation because one 8-oz serving of nonfat milk still provides 85 kcal. Some evidence suggests that low-fat dairy products may also be beneficial for weight loss and the prevention of hypertension, T2DM, and CHD.^{88,89}

Diet soda is a reasonable alternative to SSBs in that they have few to no calories, but they provide no nutritional value, and little is known about the health consequences of consuming artificial sweeteners during a lifetime.⁹⁰ In addition, some evidence suggests that the intense sweetness of artificial sweeteners could lead to conditioning for a greater preference for sweets and thus may actually enhance appetite, but this

area remains controversial.⁹¹ Several epidemiological studies have suggested a positive association between diet soda consumption and weight gain and risk of MetSyn.^{40,44,45} However, these observations may be due to reverse causation or residual confounding because, for example, diet soda consumption is higher among individuals with diabetes mellitus than those without diabetes mellitus.⁹² Studies with longer durations of follow-up and repeated measures, which are less prone to reverse causation, showed only marginal nonsignificant associations with diet soda.^{34,35,53} Some evidence suggests that a subset of diet soda consumers use diet soda as rationale for consuming other higher-calorie foods.⁹⁰

There is also growing concern about excessive fruit juice intake, but the evidence is limited. In a large cohort of women, high intake of fruit juices was positively associated with incidence of T2DM, whereas intake of whole fruits and green leafy vegetables was inversely associated.⁴² Although Schulze and colleagues did not find an association between fruit juice and risk of T2DM, they did find a positive association with weight gain.³⁴ Fruit juice has also been linked with increased weight among Australian children.⁹³ However, Ghanim and colleagues observed significantly lower reactive oxygen species generation and nuclear factor- κ B binding after consumption of orange juice compared with a glucose drink, possibly resulting from its flavonoid content.⁷⁰ Although fruit juice can provide some vitamins and nutrients, they often contain high amounts of sugar and calories and should therefore be consumed in moderation.

Methodological Issues

Although more studies are warranted to better understand the underlying biological mechanisms mediating the effect of SSBs on weight gain, T2DM, and cardiovascular risk, evidence from observational studies shows clear positive associations. Clinical trials, on which policies and recommendations are often based, are not well suited to this modality because they are greatly affected by intervention intensity and are limited by compliance, which tends to wane with increasing study length. To effectively evaluate the risk of chronic disease, sufficient follow-up time is needed for causal action and disease initiation and detection to occur, which would be difficult to emulate in the setting of a clinical trial. Thus, in the midst of an obesity epidemic that is fueling an epidemic of T2DM and cardiovascular risk, ample evidence exists from the observational studies at hand for nutrition recommendations and policy to discourage consumption of SSBs. However, certain limitations inherent in these studies are important to consider when interpreting the evidence.

Most studies discussed here adjusted their analyses for potential confounding by various lifestyle factors, and for the majority, a positive association persisted, suggesting an independent effect of SSBs. However, residual confounding by unmeasured or imperfectly measured factors cannot be ruled out. Higher SSB intake could be a marker of a globally undesirable diet because it tends to cluster with other unhealthy dietary and lifestyle habits such as higher intakes of saturated and *trans* fats^{44,94} and higher GL.³⁴ Therefore, incomplete adjustment for various lifestyle factors could lead

to an overestimation of the strength of the positive associations. However, the consistency of results from different cohorts reduces the likelihood that residual confounding is responsible for the findings. Because total energy intake partially mediates the effect of SSBs on weight gain, T2DM, and cardiovascular risk, whether a study has adjusted for total energy intake can seriously affect its results. For example, in our recent meta-analysis evaluating SSB intake and BMI in children,¹⁹ when energy-adjusted estimates were excluded, the summary effect estimate increased from a nonsignificant inverse trend (-0.03 ; 95% CI, -0.11 to 0.04) to a significant positive association (0.08 ; 95% CI, 0.03 to 0.13). Even after adjustment for total energy and other mediating factors such as BMI, some studies have still shown positive associations, supporting an effect of SSBs that is not mediated through energy intake or adiposity.^{53,95,96} Measurement error in dietary assessment is inevitable. However, in the setting of prospective cohort studies, misclassification of SSB intake does not likely differ by case status. Such nondifferential misclassification of exposure may actually attenuate the associations. Awareness of weight status, however, could result in systematic underreporting of SSB intake (as of body weight), which could weaken the association of SSBs with weight gain.

Longitudinal studies evaluating diet and weight change may also be prone to reverse causation (ie, people change their diet because of their weight, which could result in spurious associations). Ascertainment of repeated measures of diet and weight or stable intake patterns during long periods of follow-up may reduce the likelihood of this. Although most studies have been conducted among white populations from the West, the underlying biological process should be generalizable to other populations, although it is possible that some ethnic groups may be more prone to the deleterious effects of SSBs on cardiovascular risk. Further work in this area is clearly warranted.

Conclusions

SSB intake has increased considerably across the globe in recent decades, tracking positively with rising rates of obesity. Given the large number of comorbidities, reduced quality of life, and high healthcare expenditures, large-scale obesity prevention efforts are now a priority for many countries around the world. SSB intake is a significant contributor to weight gain and can lead to increased risk of T2DM and cardiovascular disease. In general, longer studies with greater numbers of participants that do not adjust for potential mediators of effect such as energy intake report stronger and more consistent results. SSBs are the greatest contributor to added-sugar intake in the United States and are thought to promote weight gain in part because of incomplete compensation for liquid calories at subsequent meals. SSBs may also increase T2DM and cardiovascular risk independently of obesity as a potential contributor to a high dietary GL and increased fructose metabolism, leading to inflammation, insulin resistance, impaired β -cell function, and high blood pressure, as well as accumulation of visceral adiposity/ectopic fat and atherogenic dyslipidemia. For these reasons and because they have little nutritional value, intake of SSBs

should be limited, and SSBs should be replaced by healthy alternatives such as water.

Acknowledgment

We thank Dr Jennifer Nettleton at the University of Texas School of Public Health for reanalyzing MESA data to provide effect estimates between regular soda intake and T2DM and MetSyn.

Source of Funding

Dr Hu's work is supported by National Institutes of Health grants HL60712 and DK58845.

Disclosures

Dr Després reports receiving research grants or consulting, speaker's, or advisory board fees from Abbott Laboratory, AstraZeneca, Eli Lilly Canada, GlaxoSmithKline, Pfizer Canada Inc, Novartis, Solvay Pharma, the Canadian Diabetes Association, and the Canadian Institutes of Health Research. Dr Hu reports receiving a research grant from Merck and a mentor-based postdoctoral fellowship from Unilever. The other authors report no conflict.

References

- Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999–2004. *JAMA*. 2006;295:1549–1555.
- Block JP, Scribner RA, DeSalvo KB. Fast food, race/ethnicity, and income: a geographic analysis. *Am J Prev Med*. 2004;27:211–217.
- Hu FB. *Obesity Epidemiology*. New York, NY: Oxford University Press; 2008.
- Finkelstein EA, Trogon JG, Cohen JW, Dietz W. Annual medical spending attributable to obesity: payer- and service-specific estimates. *Health Aff (Millwood)*. 2009;28:w822–w831.
- Johnson RK, Appel LJ, Brands M, Howard BV, Lefevre M, Lustig RH, Sacks F, Steffen LM, Wylie-Rosett J, for the American Heart Association Nutrition Committee of the Council on Nutrition, Physical Activity and Metabolism and the Council on Epidemiology and Prevention. Dietary sugars intake and cardiovascular health: a scientific statement from the American Heart Association. *Circulation*. 2009;120:1011–1020.
- Block G. Foods contributing to energy intake in the US: data from NHANES III and NHANES 1999–2000. *J Food Composition Anal*. 2004;14:439–447.
- DiMeglio DP, Mattes RD. Liquid versus solid carbohydrate: effects on food intake and body weight. *Int J Obes Relat Metab Disord*. 2000;24:794–800.
- Mattes RD. Dietary compensation by humans for supplemental energy provided as ethanol or carbohydrate in fluids. *Physiol Behav*. 1996;59:179–187.
- Schulze MB, Liu S, Rimm EB, Manson JE, Willett WC, Hu FB. Glycemic index, glycemic load, and dietary fiber intake and incidence of type 2 diabetes in younger and middle-aged women. *Am J Clin Nutr*. 2004;80:348–356.
- Stanhope KL, Schwarz JM, Keim NL, Griffen SC, Bremer AA, Graham JL, Hatcher B, Cox CL, Dyachenko A, Zhang W, McGahan JP, Seibert A, Krauss RM, Chiu S, Schaefer EJ, Ai M, Otokozawa S, Nakajima K, Nakano T, Beysen C, Hellerstein MK, Berglund L, Havel PJ. Consuming fructose-sweetened, not glucose-sweetened, beverages increases visceral adiposity and lipids and decreases insulin sensitivity in overweight/obese humans. *J Clin Invest*. 2009;119:1322–1334.
- Wolf A, Bray GA, Popkin BM. A short history of beverages and how our body treats them. *Obes Rev*. 2008;9:151–164.
- Emmins CI. Soft drinks. In: Kiple KF, Ornelas KC, eds. *The Cambridge World History of Food*. Cambridge, UK: Cambridge University Press; 2000:702–712.
- Pendergrast M. *For God, Country, and Coca-Cola: The Definitive History of the Great American Soft Drink and the Company That Makes It*. 2nd ed. New York, NY: Basic Books; 2000.
- Nielsen SJ, Popkin BM. Changes in beverage intake between 1977 and 2001. *Am J Prev Med*. 2004;27:205–210.
- Rivera JA, Muñoz-Hernández O, Rosas-Peralta M, Aguilar-Salinas CA, Popkin BM, Willett WC. Beverage consumption for a healthy life: recommendations for the Mexican population [in Spanish]. *Salud Publica Mexico*. 2008;50:173–195.

16. Ismail AI, Tanzer JM, Dingle JL. Current trends of sugar consumption in developing societies. *Community Dent Oral Epidemiol.* 1997;25:438–443.
17. Duffey KJ, Popkin BM. Shifts in patterns and consumption of beverages between 1965 and 2002. *Obesity (Silver Spring).* 2007;15:2739–2747.
18. Skinner AC, Mayer ML, Flower K, Perrin EM, Weinberger M. Using BMI to determine cardiovascular risk in childhood: how do the BMI cutoffs fare? *Pediatrics.* 2009;124:e905–e912.
19. Malik VS, Willett WC, Hu FB. Sugar-sweetened beverages and BMI in children and adolescents: reanalyses of a meta-analysis. *Am J Clin Nutr.* 2009;89:438–439.
20. Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. *Lancet.* 2001;357:505–508.
21. Berkey CS, Rockett HR, Field AE, Gillman MW, Colditz GA. Sugar-added beverages and adolescent weight change. *Obes Res.* 2004;12:778–788.
22. James J, Thomas P, Cavan D, Kerr D. Preventing childhood obesity by reducing consumption of carbonated drinks: cluster randomised controlled trial. *BMJ.* 2004;328:1237.
23. Phillips SM, Bandini LG, Naumova EN, Cyr H, Colclough S, Dietz WH, Must A. Energy-dense snack food intake in adolescence: longitudinal relationship to weight and fatness. *Obes Res.* 2004;12:461–472.
24. Ebbeling CB, Feldman HA, Osganian SK, Chomitz VR, Ellenbogen SJ, Ludwig DS. Effects of decreasing sugar-sweetened beverage consumption on body weight in adolescents: a randomized, controlled pilot study. *Pediatrics.* 2006;117:673–680.
25. Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systematic review. *Am J Clin Nutr.* 2006;84:274–288.
26. Olsen NJ, Heitmann BL. Intake of calorically sweetened beverages and obesity. *Obes Rev.* 2009;10:68–75.
27. Vartanian LR, Schwartz MB, Brownell KD. Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis. *Am J Public Health.* 2007;97:667–675.
28. Dubois L, Farmer A, Girard M, Peterson K. Regular sugar-sweetened beverage consumption between meals increases risk of overweight among preschool-aged children. *J Am Diet Assoc.* 2007;107:924–934.
29. Johnson L, Mander AP, Jones LR, Emmett PM, Jebb SA. Is sugar-sweetened beverage consumption associated with increased fatness in children? *Nutrition.* 2007;23:557–563.
30. Nissinen K, Mikkilä V, Mannisto S, Lahti-Koski M, Rasanen L, Viikari J, Raitakari OT. Sweets and sugar-sweetened soft drink intake in childhood in relation to adult BMI and overweight: the Cardiovascular Risk in Young Finns Study. *Public Health Nutr.* May 28 2009;1–9.
31. Viner RM, Cole TJ. Who changes body mass between adolescence and adulthood? Factors predicting change in BMI between 16 year and 30 years in the 1970 British Birth Cohort. *Int J Obes (Lond).* 2006;30:1368–1374.
32. Sichiari R, Paula Trotte A, de Souza RA, Veiga GV. School randomised trial on prevention of excessive weight gain by discouraging students from drinking sodas. *Public Health Nutr.* 2009;12:197–202.
33. James J, Thomas P, Kerr D. Preventing childhood obesity: two year follow-up results from the Christchurch obesity prevention programme in schools (CHOPPS). *BMJ.* 2007;335:762.
34. Schulze MB, Manson JE, Ludwig DS, Colditz GA, Stampfer MJ, Willett WC, Hu FB. Sugar-sweetened beverages, weight gain, and incidence of type 2 diabetes in young and middle-aged women. *JAMA.* 2004;292:927–934.
35. Palmer JR, Boggs DA, Krishnan S, Hu FB, Singer M, Rosenberg L. Sugar-sweetened beverages and incidence of type 2 diabetes mellitus in African American women. *Arch Intern Med.* 2008;168:1487–1492.
36. Kvaavik E, Meyer HE, Tverdal A. Food habits, physical activity and body mass index in relation to smoking status in 40–42 year old Norwegian women and men. *Prev Med.* 2004;38:1–5.
37. French SA, Jeffery RW, Forster JL, McGovern PG, Kelder SH, Baxter JE. Predictors of weight change over two years among a population of working adults: the Healthy Worker Project. *Int J Obes Relat Metab Disord.* 1994;18:145–154.
38. Bes-Rastrollo M, Sanchez-Villegas A, Gomez-Gracia E, Martinez JA, Pajares RM, Martinez-Gonzalez MA. Predictors of weight gain in a Mediterranean cohort: the Seguimiento Universidad de Navarra Study 1. *Am J Clin Nutr.* 2006;83:362–370.
39. Schulz M, Kroke A, Liese AD, Hoffmann K, Bergmann MM, Boeing H. Food groups as predictors for short-term weight changes in men and women of the EPIC-Potsdam cohort. *J Nutr.* 2002;132:1335–1340.
40. Dhingra R, Sullivan L, Jacques PF, Wang TJ, Fox CS, Meigs JB, D'Agostino RB, Gaziano JM, Vasan RS. Soft drink consumption and risk of developing cardiometabolic risk factors and the metabolic syndrome in middle-aged adults in the community. *Circulation.* 2007;116:480–488.
41. Chen L, Appel LJ, Loria C, Lin PH, Champagne CM, Elmer PJ, Ard JD, Mitchell D, Batch BC, Svetkey LP, Caballero B. Reduction in consumption of sugar-sweetened beverages is associated with weight loss: the PREMIER trial. *Am J Clin Nutr.* 2009;89:1299–1306.
42. Bazzano LA, Li TY, Josphipura KJ, Hu FB. Intake of fruit, vegetables, and fruit juices and risk of diabetes in women. *Diabetes Care.* 2008;31:1311–1317.
43. Paynter NP, Yeh HC, Voutilainen S, Schmidt MI, Heiss G, Folsom AR, Brancati FL, Kao WH. Coffee and sweetened beverage consumption and the risk of type 2 diabetes mellitus: the atherosclerosis risk in communities study. *Am J Epidemiol.* 2006;164:1075–1084.
44. Lutsey PL, Steffen LM, Stevens J. Dietary intake and the development of the metabolic syndrome: the Atherosclerosis Risk in Communities study. *Circulation.* 2008;117:754–761.
45. Nettleton JA, Lutsey PL, Wang Y, Lima JA, Michos ED, Jacobs DR Jr. Diet soda intake and risk of incident metabolic syndrome and type 2 diabetes in the Multi-Ethnic Study of Atherosclerosis (MESA). *Diabetes Care.* 2009;32:688–694.
46. Winkelmayer WC, Stampfer MJ, Willett WC, Curhan GC. Habitual caffeine intake and the risk of hypertension in women. *JAMA.* 2005;294:2330–2335.
47. Nguyen S, Choi HK, Lustig RH, Hsu CY. Sugar-sweetened beverages, serum uric acid, and blood pressure in adolescents. *J Pediatr.* 2009;154:807–813.
48. Sorensen LB, Raben A, Stender S, Astrup A. Effect of sucrose on inflammatory markers in overweight humans. *Am J Clin Nutr.* 2005;82:421–427.
49. Schulze MB, Hoffmann K, Manson JE, Willett WC, Meigs JB, Weikert C, Heidemann C, Colditz GA, Hu FB. Dietary pattern, inflammation, and incidence of type 2 diabetes in women. *Am J Clin Nutr.* 2005;82:675–684.
50. Liu S, Manson JE, Buring JE, Stampfer MJ, Willett WC, Ridker PM. Relation between a diet with a high glycemic load and plasma concentrations of high-sensitivity C-reactive protein in middle-aged women. *Am J Clin Nutr.* 2002;75:492–498.
51. Choi JW, Ford ES, Gao X, Choi HK. Sugar-sweetened soft drinks, diet soft drinks, and serum uric acid level: the Third National Health and Nutrition Examination Survey. *Arthritis Rheum.* 2008;59:109–116.
52. Choi HK, Curhan G. Soft drinks, fructose consumption, and the risk of gout in men: prospective cohort study. *BMJ.* 2008;336:309–312.
53. Fung TT, Malik V, Rexrode KM, Manson JE, Willett WC, Hu FB. Sweetened beverage consumption and risk of coronary heart disease in women. *Am J Clin Nutr.* 2009;89:1037–1042.
54. Wolff E, Dansinger ML. Soft drinks and weight gain: how strong is the link? *Medscape J Med.* 2008;10:189.
55. Raben A, Vasilaras TH, Moller AC, Astrup A. Sucrose compared with artificial sweeteners: different effects on ad libitum food intake and body weight after 10 wk of supplementation in overweight subjects. *Am J Clin Nutr.* 2002;76:721–729.
56. Tordoff MG, Alleva AM. Effect of drinking soda sweetened with aspartame or high-fructose corn syrup on food intake and body weight. *Am J Clin Nutr.* 1990;51:963–969.
57. Popkin BM, Armstrong LE, Bray GM, Caballero B, Frei B, Willett WC. A new proposed guidance system for beverage consumption in the United States. *Am J Clin Nutr.* 2006;83:529–542.
58. Janssens JP, Shapira N, Debeuf P, Michiels L, Putman R, Bruckers L, Renard D, Molenberghs G. Effects of soft drink and table beer consumption on insulin response in normal teenagers and carbohydrate drink in youngsters. *Eur J Cancer Prev.* 1999;8:289–295.
59. Ludwig DS. The glycemic index: physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. *JAMA.* 2002;287:2414–2423.
60. Liu S, Willett WC, Stampfer MJ, Hu FB, Franz M, Sampson L, Hennekens CH, Manson JE. A prospective study of dietary glycemic load, carbohydrate intake, and risk of coronary heart disease in US women. *Am J Clin Nutr.* 2000;71:1455–1461.
61. Vlassara H, Cai W, Crandall J, Goldberg T, Oberstein R, Dardaine V, Peppas M, Rayfield EJ. Inflammatory mediators are induced by dietary glycotoxins, a major risk factor for diabetic angiopathy. *Proc Natl Acad Sci U S A.* 2002;99:15596–15601.

62. Goldberg T, Cai W, Peppas M, Dardaine V, Baliga BS, Uribarri J, Vlassara H. Advanced glycoxidation end products in commonly consumed foods. *J Am Diet Assoc.* 2004;104:1287-1291.
63. Elliott SS, Keim NL, Stern JS, Teff K, Havel PJ. Fructose, weight gain, and the insulin resistance syndrome. *Am J Clin Nutr.* 2002;76:911-922.
64. Havel PJ. Dietary fructose: implications for dysregulation of energy homeostasis and lipid/carbohydrate metabolism. *Nutr Rev.* 2005;63:133-157.
65. Bray GA. How bad is fructose? *Am J Clin Nutr.* 2007;86:895-896.
66. Basciano H, Federico L, Adeli K. Fructose, insulin resistance, and metabolic dyslipidemia. *Nutr Metab (Lond).* 2005;2:5.
67. Stanhope KL, Griffen SC, Bair BR, Swarbrick MM, Keim NL, Havel PJ. Twenty-four-hour endocrine and metabolic profiles following consumption of high-fructose corn syrup-, sucrose-, fructose-, and glucose-sweetened beverages with meals. *Am J Clin Nutr.* 2008;87:1194-1203.
68. Stanhope KL, Havel PJ. Endocrine and metabolic effects of consuming beverages sweetened with fructose, glucose, sucrose, or high-fructose corn syrup. *Am J Clin Nutr.* 2008;88:1733S-1737S.
69. Teff KL, Grudziak J, Townsend RR, Dunn TN, Grant RW, Adams SH, Keim NL, Cummings BP, Stanhope KL, Havel PJ. Endocrine and metabolic effects of consuming fructose- and glucose-sweetened beverages with meals in obese men and women: influence of insulin resistance on plasma triglyceride responses. *J Clin Endocrinol Metab.* 2009;94:1562-1569.
70. Ghanim H, Mohanty P, Pathak R, Chaudhuri A, Sia CL, Dandona P. Orange juice or fructose intake does not induce oxidative and inflammatory response. *Diabetes Care.* 2007;30:1406-1411.
71. Johnson RJ, Segal MS, Sautin Y, Nakagawa T, Feig DI, Kang DH, Gersch MS, Benner S, Sanchez-Lozada LG. Potential role of sugar (fructose) in the epidemic of hypertension, obesity and the metabolic syndrome, diabetes, kidney disease, and cardiovascular disease. *Am J Clin Nutr.* 2007;86:899-906.
72. Nakagawa T, Tuttle KR, Short RA, Johnson RJ. Hypothesis: fructose-induced hyperuricemia as a causal mechanism for the epidemic of the metabolic syndrome. *Nat Clin Pract Nephrol.* 2005;1:80-86.
73. Brown CM, Dulloo AG, Yepuri G, Montani JP. Fructose ingestion acutely elevates blood pressure in healthy young humans. *Am J Physiol Regul Integr Comp Physiol.* 2008;294:R730-R737.
74. Rodin J. Comparative effects of fructose, aspartame, glucose, and water preloads on calorie and macronutrient intake. *Am J Clin Nutr.* 1990;51:428-435.
75. Chan JC, Malik V, Jia W, Kadowaki T, Yajnik CS, Yoon KH, Hu FB. Diabetes in Asia: epidemiology, risk factors, and pathophysiology. *JAMA.* 2009;301:2129-2140.
76. Brownell KD, Farley T, Willett WC, Popkin BM, Chaloupka FJ, Thompson JW, Ludwig DS. The public health and economic benefits of taxing sugar-sweetened beverages. *N Engl J Med.* 2009;361:1599-1605.
77. Panel on Dietary Reference Intakes for Electrolytes and Water, Standing Committee on the Scientific Evaluation of Dietary Reference Intakes, Food and Nutrition Board. Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate. Washington, DC: Institute of Medicine of the National Academies, the National Academies Press; 2005.
78. Jequier E, Constant F. Water as an essential nutrient: the physiological basis of hydration. *Eur J Clin Nutr.* 2010;64:115-123.
79. Popkin BM, Barclay DV, Nielsen SJ. Water and food consumption patterns of U.S. adults from 1999 to 2001. *Obes Res.* 2005;13:2146-2152.
80. Muckelbauer R, Libuda L, Clausen K, Toschke AM, Reinchr T, Kersting M. Promotion and provision of drinking water in schools for overweight prevention: randomized, controlled cluster trial. *Pediatrics.* 2009;123:e661-e667.
81. Stookey JD, Constant F, Gardner CD, Popkin BM. Replacing sweetened caloric beverages with drinking water is associated with lower energy intake. *Obesity (Silver Spring).* 2007;15:3013-3022.
82. Dennis EA, Dengo AL, Comber DL, Flack KD, Savla J, Davy KP, Davy BM. Water consumption increases weight loss during a hypocaloric diet intervention in middle-aged and older adults. *Obesity (Silver Spring).* 2010;18:300-307.
83. Black RM, Leiter LA, Anderson GH. Consuming aspartame with and without taste: differential effects on appetite and food intake of young adult males. *Physiol Behav.* 1993;53:459-466.
84. Almiron-Roig E, Drewnowski A. Hunger, thirst, and energy intakes following consumption of caloric beverages. *Physiol Behav.* 2003;79:767-773.
85. van Dieren S, Uiterwaal CS, van der Schouw YT, van der ADI, Boer JM, Spijkerman A, Grobbee DE, Beulens JW. Coffee and tea consumption and risk of type 2 diabetes. *Diabetologia.* 2009;52:2561-2569.
86. van Dam RM. Coffee consumption and risk of type 2 diabetes, cardiovascular diseases, and cancer. *Appl Physiol Nutr Metab.* 2008;33:1269-1283.
87. Hamack L, Stang J, Story M. Soft drink consumption among US children and adolescents: nutritional consequences. *J Am Diet Assoc.* 1999;99:436-441.
88. Choi HK, Willett WC, Stampfer MJ, Rimm EB, Hu FB. Dairy consumption and risk of type 2 diabetes mellitus in men: a prospective study. *Arch Intern Med.* 2005;165:997-1003.
89. Rajpathak SN, Rimm EB, Rosner B, Willett WC, Hu FB. Calcium and dairy intakes in relation to long-term weight gain in US men. *Am J Clin Nutr.* 2006;83:559-566.
90. Mattes RD, Popkin BM. Nonnutritive sweetener consumption in humans: effects on appetite and food intake and their putative mechanisms. *Am J Clin Nutr.* 2009;89:1-14.
91. Fowler SP, Williams K, Resendez RG, Hunt KJ, Hazuda HP, Stern MP. Fueling the obesity epidemic? Artificially sweetened beverage use and long-term weight gain. *Obesity (Silver Spring).* 2008;16:1894-1900.
92. Mackenzie T, Brooks B, O'Connor G. Beverage intake, diabetes, and glucose control of adults in America. *Ann Epidemiol.* 2006;16:688-691.
93. Sanigorski AM, Bell AC, Swinburn BA. Association of key foods and beverages with obesity in Australian schoolchildren. *Public Health Nutr.* 2007;10:152-157.
94. Fung TT, Willett WC, Stampfer MJ, Manson JE, Hu FB. Dietary patterns and the risk of coronary heart disease in women. *Arch Intern Med.* 2001;161:1857-1862.
95. Kral TV, Stunkard AJ, Berkowitz RI, Stallings VA, Moore RH, Faith MS. Beverage consumption patterns of children born at different risk of obesity. *Obesity (Silver Spring).* 2008;16:1802-1808.
96. Striegel-Moore RH, Thompson D, Affenito SG, Franko DL, Obarzanek E, Barton BA, Schreiber GB, Daniels SR, Schmidt M, Crawford PB. Correlates of beverage intake in adolescent girls: the National Heart, Lung, and Blood Institute Growth and Health Study. *J Pediatr.* 2006;148:183-187.

KEY WORDS: diabetes mellitus ■ epidemiology ■ meta-analysis ■ nutrition ■ prevention

Reduction in Risk Factors for Type 2 Diabetes Mellitus in Response to a Low-Sugar, High-Fiber Dietary Intervention in Overweight Latino Adolescents

Emily Ventura, MPH; Jaimie Davis, PhD, RD; Courtney Byrd-Williams; Katharine Alexander, MS; Arianna McClain; Christianne Joy Lane, MS; Donna Spruijt-Metz, PhD; Marc Weigensberg, MD; Michael Goran, PhD

Objective: To examine if reductions in added sugar intake or increases in fiber intake in response to a 16-week intervention were related to improvements in metabolic outcomes related to type 2 diabetes mellitus risk.

Design: Secondary analysis of a randomized control trial.

Setting: Intervention classes at a lifestyle laboratory and metabolic measures at the General Clinical Research Center.

Participants: Fifty-four overweight Latino adolescents (mean [SD] age, 15.5 [1] years).

Intervention: Sixteen-week study with 3 groups: control, nutrition, or nutrition plus strength training.

Main Outcome Measures: Body composition by dual-energy x-ray absorptiometry; visceral adipose tissue by magnetic resonance imaging; glucose and insulin incremental area under the curve by oral glucose tolerance test; insulin sensitivity, acute insulin response, and disposition index by intravenous glucose tolerance test; and dietary intake by 3-day records.

Results: Fifty-five percent of all participants decreased added sugar intake (mean decrease, 47 g/d) and 59% increased fiber intake (mean increase, 5 g/d), and percentages were similar in all intervention groups, including controls. Those who decreased added sugar intake had an improvement in glucose incremental area under the curve (-15% vs +3%; $P = .049$) and insulin incremental area under the curve (-33% vs -9%; $P = .02$). Those who increased fiber intake had an improvement in body mass index (-2% vs +2%; $P = .01$) and visceral adipose tissue (-10% vs no change; $P = .03$).

Conclusions: Individuals who reduced added sugar intake by the equivalent of 1 can of soda per day or increased fiber intake by the equivalent of a 1/2 cup of beans showed improvements in key risk factors for type 2 diabetes, specifically in insulin secretion and visceral fat. Improvements occurred independent of group assignment and were equally likely to occur in control group participants.

Trial Registration: clinicaltrials.gov Identifier: NCT00697580

Arch Pediatr Adolesc Med. 2009;163(4):320-327

IN 2003-2006, 38.9% OF MEXICAN American adolescents aged 12 to 19 years were at risk of overweight or overweight, as compared with 33.1% of non-Hispanic white adolescents.¹ In addition, independent of body composition, Latino children are more insulin resistant and thus more likely to develop

See also page 400

obesity-related chronic diseases than their white counterparts.² In a convenience sample of overweight Latino children in Los Angeles, California, we previously showed that 30% had a clustering of diabetes mellitus and cardiovascular disease risk factors known as the metabolic syndrome and 32% had prediabetes (ie, impaired fasting or 2-hour glucose intolerance).^{3,4}

Diet is one of the main modifiable risk factors for the development of type 2 dia-

betes. In previous cross-sectional analyses in overweight Latino youth, we showed that dietary fiber consumption is inversely associated with both waist circumference and the metabolic syndrome⁵ and that intake of total and added sugar is associated with poor beta-cell function, independent of adiposity.⁶ Additionally, we showed that in a 12-week pilot intervention study, overweight Latina girls with greater reductions in added sugar intake showed greater reductions in insulin secretion.⁷ To date, only a few studies have examined the effects of a high-fiber, low-sugar diet on metabolic health in overweight youth,^{8,9} and to our knowledge, none have tested the effects of this type of intervention in a mixed-sex group of Latino youth.

This article is a secondary data analysis from a 16-week randomized control trial. The original study assessed the in-

Author Affiliations:

Department of Preventive Medicine, Keck School of Medicine, University of Southern California, Los Angeles (Mss Ventura, Byrd-Williams, Alexander, McClain, and Lane and Drs Davis, Spruijt-Metz, and Goran); and Department of Pediatrics, Los Angeles County-University of Southern California Medical Center (Dr Weigensberg).

cremental effects of the following 3 intervention groups on adiposity and risk factors for type 2 diabetes in overweight Latino adolescents: (1) control, (2) a nutrition education program designed to reduce sugar and increase fiber intake, and (3) same nutrition education program with twice per week strength training. The main outcomes analysis showed no significant overall effects of the intervention on body weight, body composition, or metabolic parameters related to risk for type 2 diabetes, with the exception of an improvement in oral glucose response (6% and 18% reductions in nutrition and combined groups, respectively, compared with a 32% increase in the control group).¹⁰ However, despite the overall lack of intervention effects, there was considerable individual variation in dietary changes and metabolic outcomes within each of the randomized groups. These results prompted the question of whether metabolic outcomes varied by achievement of the dietary goals, regardless of group assignment. The objective of this analysis was, therefore, to test if participants who reduce added sugar intake and/or increase fiber intake will have stronger metabolic improvements related to future diabetes risk, including improvements in insulin/glucose indexes and in adiposity parameters.

METHODS

PARTICIPANTS

Participants were recruited from Los Angeles County and met the following inclusion criteria: body mass index (BMI) (calculated as weight in kilograms divided by height in meters squared) in the 85th percentile or higher,¹¹ Latino ethnicity, and grades 9 through 12. Participants were excluded if they (1) were using medication or were diagnosed with any syndrome or disease that could influence dietary intake, exercise ability, body composition and fat distribution, or insulin action and secretion, (2) were previously diagnosed with any major illness, (3) met diagnostic criteria for diabetes, or (4) participated in a structured exercise, nutrition, or weight loss program in the past 6 months. Informed written consent from parents and assent from the children were obtained. This study was approved by the institutional review board of the University of Southern California, Health Sciences Campus.

RANDOMIZATION

Sixty-six participants were randomized to 1 of 3 groups and allocations were concealed from participants until after pretesting was complete. Of the 66 participants who were randomized, 54 completed the intervention. There were no statistically significant differences in baseline demographics, anthropometrics, or body composition measures between the 12 participants who dropped out of the program and the 54 participants who completed the program.

DESCRIPTION OF INTERVENTIONS

The nutrition-only group received 1 nutrition class per week for 16 weeks. The dietary intervention targeted 2 goals: a decrease in added sugar consumption and an increase in fiber consumption. Participants in the nutrition plus strength training group received the same weekly nutrition classes along with strength training 2 times per week for 16 weeks.

Participants randomized to the control group received no intervention between preintervention and postintervention data collection. Periodically through the 16-week intervention, participants received non-health-related incentives, such as T-shirts, and regular telephone calls to enhance retention. After posttesting, participants were offered a delayed intervention for 1 month.

PROTOCOL AND OUTCOME MEASURES

At both baseline and 16 weeks, participants had both an outpatient and inpatient clinic visit for assessment of insulin and glucose indexes, anthropometrics, body composition, and dietary intake.

Outpatient Visit

Participants arrived at the University of Southern California General Clinical Research Center at approximately 7:30 AM after an overnight fast. A licensed pediatric health care provider conducted a medical history examination and determined Tanner staging using established guidelines.^{12,13} Following the examination, a 3-hour oral glucose tolerance test (OGTT) was conducted. A flexible intravenous catheter was placed in an antecubital vein and subjects then ingested 1.75 g of oral glucose solution per kilogram of body weight (to a maximum 75 g). Blood samples were drawn at baseline and every 10 minutes for 3 hours and were assayed for glucose, insulin, and C-peptide levels. Fasting and 2-hour glucose levels were used to determine normal glucose tolerance (2-hour glucose level, <140 mg/dL) or impaired glucose tolerance (2-hour glucose level, \geq 140 and <200 mg/dL) as defined by the American Diabetes Association.¹⁴ Three-hour insulin and glucose area under the curve (AUC) and incremental area under the curve (IAUC) were calculated from the OGTT data, in milligrams per minute per deciliter for glucose and microunits per minute per milliliter for insulin. Glucose and insulin AUCs are the sum of the area of each time segment by insulin or glucose concentration and IAUCs are the sum of the same area adjusted for the starting point. Insulin AUC and IAUC are approximate measures of insulin secretion in response to a standard oral glucose load.

Anthropometry and Body Composition

Weight and height were measured to the nearest 0.1 kg and 0.1 cm. Body mass index and BMI percentiles for age and sex were determined using EpiInfo 2000, version 1.1 (Centers for Disease Control and Prevention, Atlanta, Georgia). Whole-body fat and soft lean tissue were measured by dual-energy x-ray absorptiometry (DEXA) using a Hologic QDR 4500W (Hologic, Bedford, Massachusetts).

Subcutaneous and visceral fat volumes were obtained by magnetic resonance imaging, using a Siemens Magnetom 1.5-T Symphony Maestro Class Syngo 2004A (Siemens AG, Erlangen, Germany) with a Numaris/4 software at the University of Southern California-Health Consultation Center II imaging center. Patients were positioned supine, and 19 axial images of the abdomen with a thickness of 10 mm were taken. Visceral and subcutaneous abdominal tissue were calculated using image analysis software (SliceOmatic; Tomovision, Montreal, Quebec, Canada) at Image Reading Center (New York, New York).

Inpatient Visit

Approximately 7 to 14 days following the outpatient visit, participants were admitted to the General Clinical Research Center and served a standardized dinner and an evening snack, with

only water permitted after 8 PM. At approximately 7:30 AM the following day, an insulin-modified frequently sampled intravenous glucose tolerance test was performed. At time 0, glucose (25% dextrose, 0.3 g/kg of body weight) was administered intravenously. Blood samples were collected at points -15, -5, 2, 4, 8, 19, 22, 30, 40, 50, 70, 100, and 180 minutes. Insulin (0.02 U/kg of body weight, Humulin R [regular insulin for human injection]; Eli Lilly, Indianapolis, Indiana) was injected intravenously at 20 minutes. Glucose and insulin values were entered into the MINMOD Millennium 2003 computer program (version 5.16; Richard N. Bergman, PhD, University of Southern California) to determine insulin sensitivity (SI), acute insulin response (AIR) (ie, insulin AUC above basal for the first 8 minutes of the frequently sampled intravenous glucose tolerance test), and disposition index (DI) (an index of pancreatic beta-cell function calculated as the product of $SI \times AIR$).

Dietary Intake

At both baseline and 16 weeks, participants were given 3-day diet records to complete. Participants were given a short lesson on how to estimate portion sizes and were given measuring cups and rulers to aid in accurate reporting. Research staff, trained and supervised by a registered dietitian, clarified all dietary records. Nutrition data were analyzed using the Nutrition Data System for Research (NDS-R version 5.0_35), a program developed by the University of Minnesota.

STATISTICAL ANALYSIS

Data Cleaning and Normalization

Of the 54 participants who completed the intervention, 49 had available dietary data. Five of the 49 were missing 1 of the 3 days of diet records for either pretesting or posttesting, and an average of 2 days was used. The DEXA measures were collected for 45 of the 49 subjects because 4 participants were over the 300-lb weight limit and magnetic resonance imaging data, for 40 of the 49 because of logistical problems.

The following outcome variables were nonnormally distributed and analyses were run on the log-transformed values: weight, DEXA fat and lean mass, 2-hour glucose level, glucose AUC and IAUC, insulin AUC and IAUC, fasting insulin level, SI, AIR, and DI. All transformations were log transformations with one exception; BMI percentile used the following transformation: $y_i = \ln(\text{highest value} + 1) - y_i$. Two outliers were identified and removed from models related to glucose and insulin indexes.

Definition of Sugar Intake Decrease and Fiber Intake Increase

Subjects were divided into categories based on whether they decreased sugar intake and/or increased fiber intake. A sugar intake decrease was defined as a decrease in added sugar intake of any magnitude (postintervention - preintervention < 0), as a percentage of total caloric intake, and fiber intake increase was defined as an increase of any magnitude in fiber intake (in grams) per 1000 calories of total energy intake (postintervention - preintervention > 0).

Baseline Comparisons

Baseline characteristics were compared between sugar and fiber intake change categories (decrease vs increase) using χ^2 tests

and independent t tests. Because there were no significant differences in sugar or fiber intake change by randomization group, all participants were combined for subsequent analyses, and randomization group was used as a covariate.

Comparison of Metabolic Change by Sugar and Fiber Intake Change Categories

Preintervention to postintervention changes in adiposity as well as insulin and glucose indexes were analyzed in 2 steps. First, preliminary analysis of raw change scores (postintervention - preintervention) for metabolic outcomes were tested for significance against zero with independent t tests. The grouping variables were sugar intake decrease (yes/no) and fiber intake increase (yes/no). In the second step, repeated-measures analysis of covariance (ANCOVA) was conducted for variables, controlling for covariates with biological significance. The between-subjects factor was sugar or fiber intake change category (increase vs decrease) and the time variable was weeks (0 vs 16). First, all models were run separately with 1 dichotomous between-subjects factor variable (either sugar intake decrease, yes/no, or fiber intake increase, yes or no). Subsequently, a 2-factor model was used by including both between-subjects factors to test for interactions in sugar and fiber intake categories (ie, decreased sugar intake only, increased fiber intake only, both, or neither). In all repeated-measures models, the following a priori covariates were included: sex, randomization group, and baseline sugar and/or fiber intake. Pretest and posttest total fat mass and total lean tissue mass, as well as age, were evaluated as covariates in each model and included only when significant. Pretest and posttest subcutaneous fat was included a priori in all visceral adipose tissue models. Data were analyzed with SPSS version 13.0 (SPSS Inc, Chicago, Illinois), and type I error was set at $\alpha < .05$.

RESULTS

Participants whose sugar and/or fiber intake improved were randomly spread across intervention groups, as is illustrated in **Figure 1**. There were no significant differences in sugar or fiber intake change categories (increase vs decrease) across the 3 different intervention groups (**Table 1** and **Table 2**; $P > .05$). Of the 49 total participants, 55% ($n = 27$) reduced added sugar intake and 59% ($n = 20$) increased fiber intake, and these values were similar across intervention groups.

Baseline characteristics by sugar and fiber intake categories are also shown in Table 1 and Table 2. The sugar intake decrease had a mean (SD) decrease of 47 (42) g/d of added sugar intake and the fiber intake increase had a mean (SD) increase of 5 (8) g/d of total fiber intake. There were no significant differences at baseline in sugar or fiber intake categories for age, sex, Tanner stage, height, measures of adiposity, or glucose/insulin indexes ($P > .05$). There was a trend toward significance for the sugar intake decrease to have a higher BMI at baseline than the sugar intake increase (35.6 vs 32.0; $P = .08$). Though there were no significant differences in macronutrient intake by sugar or fiber intake categories at baseline ($P > .05$), the sugar intake decrease had a higher percentage of calories from added sugar intake at baseline ($P = .003$) and the fiber intake increase had a lower baseline intake of fiber (in grams) per 1000 calories ($P = .001$).

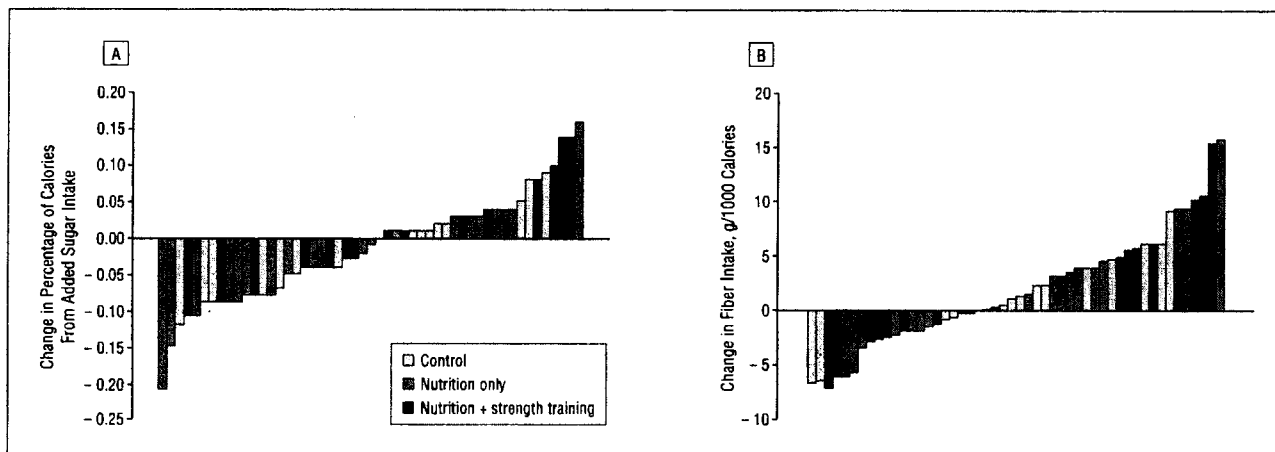


Figure 1. Changes in added sugar (A) and fiber (B) intake displayed by subject, coded by randomization group.

Table 1. Baseline Characteristics in Those Participants Who Decreased or Increased Their Percentage of Calories From Added Sugar Intake^a

	Mean (SD)		P Value
	Decreased Added Sugar Intake (n=27)	Increased Added Sugar Intake (n=22)	
Sex, M/F, %	52/48	50/50	.90
Randomization group, control/nutrition/combo, %	30/44/26	27/36/36	.72
Age, y	15.6 (1.0)	15.2 (1.1)	.20
Height, cm	165.7 (8.2)	165.3 (7.7)	.84
Weight, kg ^b	98.7 (26.9)	87.0 (15.1)	.11
BMI	35.6 (7.6)	32.0 (6.0)	.08
BMI percentile ^b	97.3 (3.7)	95.8 (4.2)	.11
Total fat mass, kg ^b	34.7 (12.3)	31.2 (11.4)	.31
Visceral fat, L	1.7 (0.8)	1.6 (0.8)	.68
Subcutaneous fat, L ^b	10.1 (4.2)	8.0 (3.8)	.12
Total lean tissue mass, kg ^b	55.3 (10.9)	53.3 (7.1)	.63
Fasting glucose level, mg/dL	92.2 (5.8)	92.3 (8.4)	.93
2-h Glucose level, mg/dL ^b	125.9 (24.3)	132.6 (27.0)	.37
Glucose IAUC, mg/min/dL ^b	101.1 (49.8)	103.2 (56.6)	.56
Fasting insulin level, $\mu\text{U/mL}^b$	28.8 (15.7)	26.7 (15.4)	.68
2-h Insulin level, $\mu\text{U/mL}^b$	190.4 (150.9)	179.3 (106.4)	.80
Insulin IAUC, $\mu\text{U/min/mL}^b$	415.7 (304.2)	354.7 (205.4)	.56
Insulin sensitivity, $(\times 10^{-4} \text{min}^{-1})/(\mu\text{U/mL})^b$	1.4 (0.8)	2.1 (1.9)	.20
Acute insulin response, $\mu\text{U/mL} \times 10 \text{ min}^b$	1415.8 (1079.0)	1145.9 (658.0)	.59
Disposition index, $\times 10^{-4} \text{min}^{-1b}$	1501.5 (794.4)	1573.1 (913.7)	.84
Energy, kcal	2032.6 (669.9)	1747.2 (538.1)	.11
Calories from fat, %	31.9 (6.1)	33.1 (6.0)	.52
Calories from protein, %	15.3 (3.2)	16.6 (3.6)	.19
Calories from carbohydrate, %	53.9 (8.1)	51.7 (6.6)	.32
Calories from added sugar, %	17.4 (6.7)	12.2 (4.3)	.003
Fiber, g per 1000 kcal	7.6 (2.8)	9.3 (3.5)	.07

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); IAUC, incremental area under the curve.

SI conversion factors: To convert glucose to millimoles per liter, multiply by 0.0555; insulin to picomoles per liter, multiply by 6.945.

^a χ^2 Tests were used for categorical variables and independent *t* tests, for continuous variables. Sample sizes for dual-energy x-ray absorptiometry were 23 in sugar intake decrease and 22 in sugar intake increase. Sample sizes for magnetic resonance imaging were 22 in sugar intake decrease and 18 in sugar intake increase.

^bVariables were not normally distributed so statistical tests were run with log-transformed data. For BMI percentile, a transformation involving $\ln(\text{highest value} + 1) - y$ was used.

Comparisons of raw change scores by sugar and fiber intake categories are shown in **Table 3**. For the added sugar intake category comparisons, the only significant difference was in insulin IAUC, where the group who decreased sugar intake showed a reduction of 121 $\mu\text{U}/\text{min}/\text{mL}$ as compared with a decrease of 36 $\mu\text{U}/\text{min}/\text{mL}$ in

those who did not decrease sugar intake ($P = .02$). In the dietary fiber intake category comparisons, those who increased fiber intake had a significant decrease in BMI (-0.6 vs $+0.5$; $P = .02$) and in visceral fat (-0.2 vs $+0.006$; $P = .04$) as compared with those who did not, but there were no significant differences in other metabolic outcomes.

Table 2. Baseline Characteristics by Those Individuals Who Decreased or Increased Fiber Intake Relative to Caloric Intake^a

	Mean (SD)		P Value
	Decreased Fiber Intake (n=20)	Increased Fiber Intake (n=29)	
Sex, M/F, %	40/60	59/41	.25
Randomization group, control/nutrition/combo, %	20/40/40	34/41/24	.40
Age, y	15.3 (1.1)	15.5 (1.0)	.44
Height, cm	163.6 (7.0)	166.8 (8.4)	.17
Weight, kg ^b	89.4 (21.4)	96.2 (23.8)	.31
BMI	33.3 (7.2)	34.4 (7.1)	.60
BMI percentile ^b	96.3 (3.9)	96.9 (4.1)	.45
Total fat mass, kg ^b	32.2 (11.8)	33.6 (12.1)	.73
Visceral fat, L	1.7 (0.7)	1.6 (0.8)	.72
Subcutaneous fat, L ^b	8.4 (4.0)	9.5 (4.2)	.47
Total lean tissue mass, kg ^b	51.5 (7.9)	56.3 (9.7)	.10
Fasting glucose level, mg/dL	91.3 (7.8)	92.9 (6.5)	.43
2-h Glucose level, mg/dL ^b	134.0 (28.5)	125.4 (23.0)	.27
Glucose IAUC, mg/min/dL ^b	107.4 (59.4)	98.4 (47.5)	.85
Fasting insulin level, μ U/mL ^b	29.5 (13.7)	27.0 (16.7)	.39
2-h Insulin level, μ U/mL ^b	181.1 (107.0)	188.4 (148.1)	.90
Insulin IAUC, μ U/min/mL ^b	364.4 (211.5)	404.8 (296.9)	.79
Insulin sensitivity, ($\times 10^{-4}$ /min ⁻¹)/(μ U/mL) ^b	2.0 (1.9)	1.6 (1.1)	.28
Acute insulin response, μ U/mL $\times 10$ min ^b	1351.7 (1017.7)	1255.2 (854.7)	.77
Disposition index, $\times 10^{-4}$ min ⁻¹ ^b	1497.7 (686.1)	1558.4 (945.2)	.75
Energy, kcal	1928.6 (696.3)	1887.8 (582.5)	.82
Calories from fat, %	31.0 (6.5)	33.4 (5.5)	.17
Calories from protein, %	16.9 (3.3)	15.2 (3.4)	.09
Calories from carbohydrate, %	53.6 (7.3)	52.4 (7.7)	.57
Calories from added sugar, %	13.7 (5.8)	16.0 (6.5)	.22
Fiber, g per 1000 kcal	10.3 (3.3)	7.1 (2.4)	.001

Abbreviations: See Table 1.

SI conversion factors: See Table 1.

^a χ^2 Tests were used for categorical variables and independent *t* tests, for continuous variables. Sample sizes for dual-energy x-ray absorptiometry were 19 in fiber intake decrease and 26 in fiber intake increase. Sample sizes for magnetic resonance imaging were 14 in fiber intake decrease and 26 in fiber intake increase.

^b Variables were not normally distributed so statistical tests were run with log-transformed data. For BMI percentile, a transformation involving $\ln(\text{highest value} + 1) - y$ was used.

Significant results for the repeated-measures ANCOVA analyses are shown in **Figure 2** and **Figure 3**. In the analyses with sugar intake category as the between-subjects factor, there were significant time \times sugar intake category interactions for both glucose IAUC and insulin IAUC, controlling for sex, randomization group, and baseline added sugar consumption. Those who reduced added sugar intake had a significant reduction in glucose IAUC (-15% vs $+3\%$; $P=.049$) (Figure 2A) and insulin IAUC (-33% vs -9% ; $P=.02$) (Figure 2B) compared with those who increased added sugar intake. Body composition was evaluated as a covariate in both models and was not significant ($P>.05$) and therefore not included in the final models. Changes in adiposity or glucose/insulin index outcomes, including SI, AIR, and DI, were not significantly different in those who reduced added sugar intake vs those who did not.

In the repeated-measures ANCOVA analyses with fiber intake category as the between-subjects factor, there were significant time \times fiber intake category interactions for both BMI and visceral adipose tissue, controlling for sex, randomization group, baseline fiber intake, and subcutaneous adipose tissue (in the visceral adipose tissue model). Those who increased fiber intake had a significant reduction in BMI (-2% vs $+2\%$; $P=.01$)

(Figure 3A) and visceral adipose tissue (-10% vs no change; $P=.03$) (Figure 3B) compared with those who decreased fiber intake. There were no other time \times fiber intake category interactions for other measures of adiposity, such as fat mass or subcutaneous fat, or glucose/insulin index outcomes, including SI, AIR, and DI.

There was considerable overlap in sugar and fiber intake categories: 78% (21 of 27) of those who reduced sugar intake also increased fiber intake, and 72% (21 of 29) of those who increased fiber intake also decreased sugar intake (data not shown). However, when sugar and fiber intake categories were tested together as 2 factors in the same repeated-measures ANCOVA model, there were no significant interactions for any of the adiposity measures or glucose/insulin indexes ($P>.05$).

COMMENT

The main findings from the current analysis show that overweight Latino adolescents who decreased added sugar intake by an average of 47 g/d, equivalent to the sugar in 1 can of soda, had an average 33% decrease in insulin secretion as assessed by IAUC during an OGTT. Additionally, participants who increased fiber intake by an average of 5

Table 3. Unadjusted Changes in Metabolic Outcomes by Dietary Improvement Categories*

	Decreased Added Sugar Intake		Increased Added Sugar Intake		P Value	Decreased Fiber Intake		Increased Fiber Intake		P Value
	Change Score (SD)	Sample Size	Change Score (SD)	Sample Size		Change Score (SD)	Sample Size	Change Score (SD)	Sample Size	
BMI	-0.3 (1.9)	25	0.1 (1.0)	20	.31	0.5 (1.1)	20	-0.6 (1.7)	27	.02
Total fat mass, kg ^b	0.1 (3.0)	22	-0.1 (1.7)	20	.93	0.4 (2.1)	19	-0.2 (2.5)	25	.38
Visceral fat, L	-0.1 (0.3)	20	0.0 (0.1)	17	.15	0.006 (0.1)	14	-0.2 (0.3)	25	.04
Fasting glucose level, mg/dL	-0.5 (6.4)	26	-4.1 (6.9)	21	.08	-1.4 (5.2)	20	-2.7 (7.6)	29	.48
2-h Glucose level, mg/dL ^b	-7.0 (24.8)	26	-12.8 (24.6)	21	.62	-13.8 (28.2)	20	-4.1 (22.9)	29	.27
Glucose IAUC, mg/min/dL ^b	-11.2 (37.3)	26	-0.6 (47.3)	21	.18	-5.8 (42.6)	20	-4.3 (42.0)	29	.46
Fasting insulin level, μ U/mL ^b	-3.1 (11.7)	26	-3.7 (10.0)	21	.79	-2.9 (8.2)	20	-1.8 (15.1)	29	.96
2-h Insulin level, μ U/mL ^b	-46.1 (122.2)	26	-47.1 (112.9)	21	.37	-22.7 (149.5)	20	-23.4 (175.3)	29	.73
Insulin IAUC, μ U/min/mL ^b	-121.1 (179.6)	26	-36.3 (164.4)	21	.02	-19.3 (226.5)	20	-77.3 (225.8)	29	.12
Insulin sensitivity, $(\times 10^{-4} \text{min}^{-1})/(\mu\text{U/mL})^b$	0.3 (0.8)	25	0.02 (0.9)	21	.62	-0.3 (2.0)	20	0.2 (1.0)	28	.06
Acute insulin response, $\mu\text{U/mL} \times 10 \text{ min}^b$	-61.5 (339.7)	25	201.7 (546.6)	21	.18	73.31 (536.9)	20	18.7 (415.9)	28	.08
Disposition index, $\times 10^{-4} \text{min}^{-1} b$	228.9 (872.5)	25	166.8 (910.3)	21	.79	101.0 (828.7)	20	332.2 (915.9)	28	.26

Abbreviations: See Table 1.

SI conversion factors: See Table 1.

*A decrease in added sugar intake is defined as a decrease in the percentage of calories from added sugar (postintervention–preintervention). An increase in fiber intake is defined as an increase in total fiber intake of any magnitude relative to caloric intake. Differences in change scores by category were assessed by independent *t* tests and data are presented as mean (SD).

^bVariables were not normally distributed so statistical tests were run with log-transformed data but untransformed values are reported for ease of interpretation.

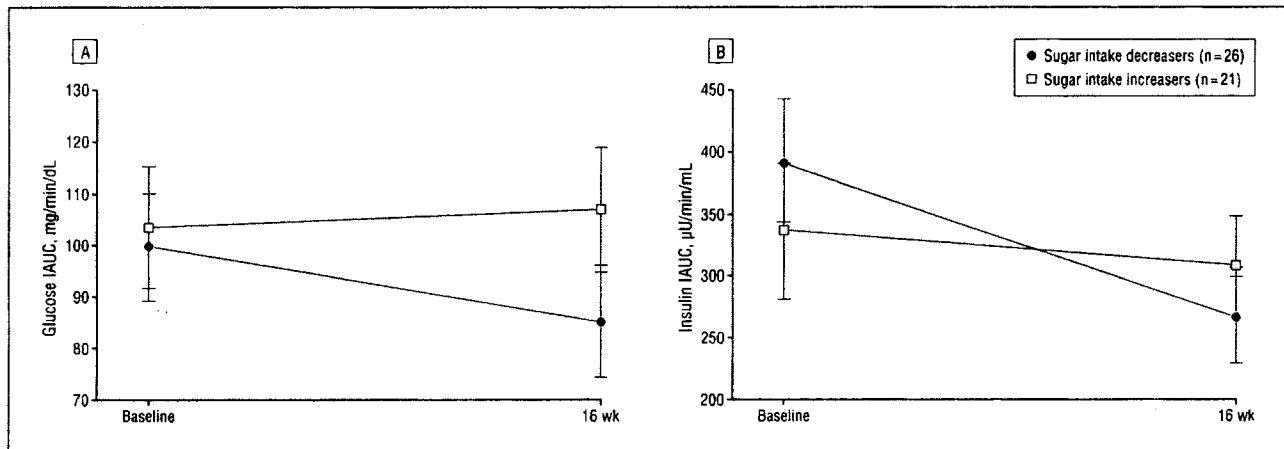


Figure 2. Adjusted changes in postchallenge glucose response (A) and insulin secretion (B) by sugar intake improvement categories. Sugar intake improvement was defined as a decrease of any magnitude in the percentage of calories from added sugar intake. Models are adjusted for sex, randomization group, and baseline added sugar intake. Body composition was evaluated in the model but removed because it was not significant. IAUC indicates incremental area under the curve.

g/d, equivalent to the amount in a 1/2 cup of beans, had an average 10% reduction in visceral adipose tissue volume. Moreover, these dietary changes were independent of intervention group assignment and children assigned to the control group were as likely to make dietary improvements and to show metabolic improvements as those assigned to a rigorous 16-week intervention.

In particular, 57% of the control participants decreased their sugar intake and 71% increased their fiber intake, in the absence of any nutrition intervention. This effect could be attributed to contamination effects because the control participants were not blinded to the purpose of the intervention. The recruitment materials and consent forms for the study explained that the purpose of the intervention was to focus on a decrease in sugar intake and an increase in fiber intake. Furthermore, the change in the control group could also be attributed to

the Hawthorne effect: when participants enrolled in the study, some became motivated to make these dietary changes on their own, knowing that they would be observed. Further analyses are warranted to explore whether intrinsic motivation and other psychosocial variables at baseline predict changes in sugar and/or fiber intake. In addition, these results prompt the question of whether it is necessary to conduct elaborate interventions in people who might already be intrinsically motivated to change.

Regardless of intervention group, participants who were able to reduce sugar intake and/or increase fiber intake showed notable metabolic improvements related to risk reduction for type 2 diabetes. Although there was overlap in sugar and fiber intake improvement, we found that reductions in sugar intake were more related to glucose and insulin indexes whereas increases in fiber intake were more related to adiposity parameters. A reduc-

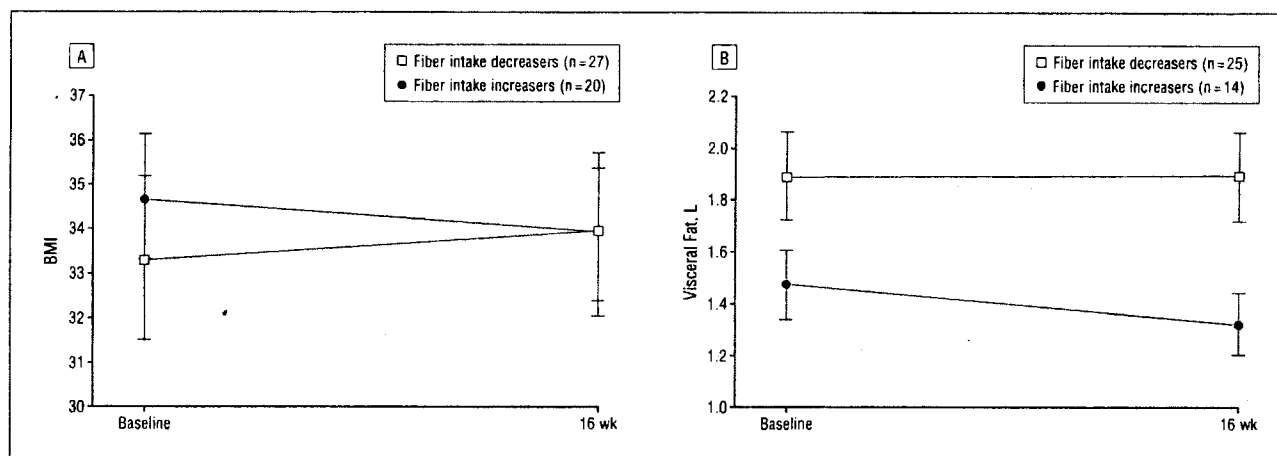


Figure 3. Adjusted changes in body mass index (BMI) (calculated as weight in kilograms divided by height in meters squared) (A) and visceral fat (B) by fiber intake improvement categories. Fiber intake improvement is defined as an increase in fiber intake of any magnitude relative to caloric intake. Models are adjusted for sex, randomization group, and baseline fiber intake. Visceral fat model also included baseline and posttest subcutaneous fat.

tion in visceral fat indicates a reduction in risk for type 2 diabetes, considering that to a greater degree than total body fat, visceral fat has been shown to be negatively associated with SI.¹⁵ In addition, a reduction in insulin response to oral glucose likely indicates a reduction in insulin secretion in response to an increase in SI. If SI increases, less insulin is required and insulin secretion decreases. Accordingly, insulin IAUC has been shown to be an indirect index of SI.¹⁶ Although not significant, we also saw an increase of 0.3 in SI, as measured by intravenous glucose tolerance test, in the participants who reduced their sugar intake as compared with an increase of only 0.02 in those who increased their sugar intake.

It is worthwhile to explore why we saw significant results in outcomes associated with the OGTT, namely glucose and insulin IAUC, but not in indexes from the intravenous glucose tolerance test, namely SI, AIR, and DI, though the directionality of the results was consistent. One explanation for the modest changes in the intravenous glucose tolerance test measures could be the relatively short intervention period of 16 weeks. The body is more responsive to oral delivery of glucose considering that it is a more natural condition, and the oral response includes mechanisms that are not triggered during intravenous delivery, such as the release of gastrointestinal hormones that facilitate insulin secretion from the beta cells after eating.¹⁷

Our results add to the literature in that we are the first, to our knowledge, to test an intensive randomized control intervention focused on quality of carbohydrates in overweight Latino adolescents and to find that reductions in sugar intake and increases in fiber intake have associated metabolic benefits. These findings are consistent with the adult literature, in which prospective studies have shown that added sugar intake is a risk factor for the development of type 2 diabetes while fiber intake is a protective factor. As far as we know, no other interventions besides our pilot study¹⁸ have tested a specific high-fiber intervention with youth, although it has been shown cross-sectionally in youth that whole-grain consumption is associated with lower BMI and increased insulin sensitivity.¹⁹ Other investigators have shown beneficial metabolic re-

sults from interventions targeting sweetened beverages with adolescents of other ethnicities. For example, in a school-based intervention with Zuni adolescents aimed to reduce consumption of soft drinks, Teufel and Ritenbaugh²⁰ found a reduction in fasting and 30-minute insulin levels in students after a 2-year intervention. In a randomized controlled pilot study with an ethnically diverse group of adolescents, Ebbeling et al²¹ found that reductions in sugar-sweetened beverage consumption were associated with a reduction in BMI, specifically in adolescents who had the highest BMI values at baseline. This particular finding parallels what we show in the present analysis, considering that participants who reduced their added sugar intake had a marginally higher BMI at baseline and a significantly higher baseline consumption of added sugar. Perhaps youth with more room for improvement at the time of enrollment have better responses to dietary interventions. In comparison with the findings of Ebbeling et al, we only found a small, nonsignificant decrease in BMI in the participants who reduced their added sugar intake. However, our study was 16 weeks while the Ebbeling et al study was 25 weeks, focused entirely on decreasing sugar-sweetened drink intake, and included the weekly provision of alternative beverages. In the Ebbeling et al study, insulin and glucose indexes were not reported; therefore, we are unable to compare results for these parameters.

In conclusion, through this secondary analysis of response to a 16-week intervention, we found that overweight Latino youth who decreased added sugar intake or increased fiber intake showed stronger improvements in risk factors for type 2 diabetes, specifically in insulin response to an oral glucose challenge or in visceral fat. Modest changes in sugar and fiber consumption, equivalent to omitting 1 can of soda or adding 1 serving of beans daily, could lead to substantial improvements in adiposity and metabolic parameters. Furthermore, given that the control group demonstrated similar dietary changes as the intervention groups, our results suggest that intensive interventions may not be necessary to achieve modifications in sugar and fiber intake. Accordingly, nutritional guidance given in the primary care or community setting may be sufficient to promote the suggested dietary changes in some

individuals. In addition, policies that promote reduced intake of added sugar and increased intake of fiber could be effective public health strategies for the prevention of type 2 diabetes in this high-risk population.

Accepted for Publication: December 17, 2008.

Correspondence: Michael Goran, PhD, 2250 Alcazar St, Los Angeles, CA 90089-9008 (goran@usc.edu).

Author Contributions: Ms Ventura and Dr Goran had full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the data analyses. *Study concept and design:* Ventura, Davis, Spruijt-Metz, Weigensberg, and Goran. *Acquisition of data:* Ventura, Davis, Byrd-Williams, Alexander, Spruijt-Metz, and Goran. *Analysis and interpretation of data:* Ventura, Davis, Byrd-Williams, McClain, Lane, Weigensberg, and Goran. *Drafting of the manuscript:* Ventura, Davis, McClain, Spruijt-Metz, and Goran. *Critical revision of the manuscript for important intellectual content:* Ventura, Davis, Byrd-Williams, Alexander, Lane, Spruijt-Metz, Weigensberg, and Goran. *Statistical analysis:* Ventura, Lane, and Goran. *Obtained funding:* Davis, Spruijt-Metz, and Goran. *Administrative, technical, and material support:* Ventura, Davis, Byrd-Williams, Alexander, and Goran. *Study supervision:* Davis, Spruijt-Metz, Weigensberg, and Goran.

Financial Disclosure: None reported.

Funding/Support: This work was supported by National Cancer Institute Cancer Control and Epidemiology Research Training Grant T32 CA 09492, University of Southern California Center for Transdisciplinary Research on Energetics and Cancer grant U54 CA 116848, National Institute of Child Health and Human Development grant RO1 HD/HL 33064, the Dr Robert C. and Veronica Atkins Foundation, and National Center for Research Resources/National Institutes of Health grant M01 RR 00043.

Role of the Sponsors: None of the funders participated in the design or conduct of the study or the collection, management, analysis, or interpretation of the data.

Additional Contributions: We would like to thank project manager Christina Ayala, MPH, and the SANO LA team as well as the nursing staff at the General Clinical Research Center. In addition, we are grateful to our study participants and their families for their involvement.

REFERENCES

- Ogden CL, Carroll MD, Flegal KM. High body mass index for age among US children and adolescents, 2003-2006. *JAMA*. 2008;299(20):2401-2405.
- Goran MI, Bergman R, Cruz M, Watanabe R. Insulin resistance and associated compensatory responses in African-American and Hispanic children. *Diabetes Care*. 2002;25(12):2184-2190.
- Cruz ML, Weigensberg MJ, Huang T, Ball GDC, Shaibi GQ, Goran MI. The metabolic syndrome in overweight Hispanic youth and the role of insulin sensitivity. *J Clin Endocrinol Metab*. 2004;89(1):108-113.
- Weigensberg MJ, Ball GD, Shaibi GQ, Cruz ML, Goran MI. Decreased beta-cell function in overweight Latino children with impaired fasting glucose. *Diabetes Care*. 2005;28(10):2519-2524.
- Ventura EEDJ, Alexander KE, Shaibi GQ, et al. Dietary intake and the metabolic syndrome in overweight Latino children. *J Am Diet Assoc*. 2008;108(8):1355-1359.
- Davis JN, Ventura E, Weigensberg M, et al. The relation of sugar intake to beta-cell function in overweight Latino children. *Am J Clin Nutr*. 2005;82(5):1004-1010.
- Davis JN, Ventura EE, Shaibi GQ, et al. Reduction in added sugar intake and improvement in insulin secretion in overweight Latina adolescents. *Metab Syndr Relat Disord*. 2007;5(2):183-193.
- Ebbeling CB, Leidig MM, Sinclair KB, Seger-Shippe L, Feldman HA, Ludwig DS. Effects of an ad libitum low-glycemic load diet on cardiovascular disease risk factors in obese young adults. *Am J Clin Nutr*. 2005;81(5):976-982.
- Ebbeling CB, Leidig MM, Sinclair KB, Hangen JP, Ludwig DS. A reduced-glycemic load diet in the treatment of adolescent obesity. *Arch Pediatr Adolesc Med*. 2003;157(8):773-779.
- Davis JN, Kelly LE, Lane CJ, et al. Randomized control trial of a nutrition education and strength training program to prevent obesity related diseases in overweight Latino adolescents. *Obesity*. In press.
- Centers for Disease Control and Prevention. *2000 CDC Growth Charts*. Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics; 2000. US Publication 314.
- Marshall WA, Tanner JM. Variations in pattern of pubertal changes in girls. *Arch Dis Child*. 1969;44(235):291-303.
- Marshall WA, Tanner JM. Variations in the pattern of pubertal changes in boys. *Arch Dis Child*. 1970;45(239):13-23.
- ADA. Type 2 diabetes in children and adolescents. *Pediatrics*. 2000;105(3, pt 1): 671-680.
- Cruz ML, Bergman RN, Goran MI. Unique effect of visceral fat on insulin sensitivity in obese Hispanic children with a family history of type 2 diabetes. *Diabetes Care*. 2002;25(9):1631-1636.
- Weigensberg MJ, Cruz ML, Goran MI. Association between insulin sensitivity and post-glucose challenge plasma insulin values in overweight Latino youth. *Diabetes Care*. 2003;26(7):2094-2099.
- Campioni M, Toffolo G, Shuster LT, Service FJ, Rizza RA, Cobelli C. Incretin effect potentiates beta-cell responsiveness to glucose as well as to its rate of change: OGTT and matched intravenous study. *Am J Physiol Endocrinol Metab*. 2007;292(1):E54-E60.
- Davis JN, Ventura EE, Alexander KE, et al. Feasibility of a home-based versus classroom-based nutrition intervention to reduce obesity and type 2 diabetes in Latino youth. *Int J Pediatr Obes*. 2007;2(1):22-30.
- Steffen LM, Jacobs DR Jr, Murtaugh MA, et al. Whole grain intake is associated with lower body mass and greater insulin sensitivity among adolescents. *Am J Epidemiol*. 2003;158(3):243-250.
- Teufel NI, Ritenbaugh C. Development of a primary prevention program: insight gained in the Zuni Diabetes Prevention Program. *Clin Pediatr (Phila)*. 1998; 37(2):131-141.
- Ebbeling CB, Feldman HA, Osganian SK, Chomitz VR, Ellenbogen SJ, Ludwig DS. Effects of decreasing sugar-sweetened beverage consumption on body weight in adolescents: a randomized, controlled pilot study. *Pediatrics*. 2006;117(3):673-680.

Announcement

Sign Up for Alerts—It's Free! Archives of Pediatrics & Adolescent Medicine offers the ability to automatically receive the table of contents of Archives via e-mail when it is published online. This also allows you to link to individual articles and view the abstract. It makes keeping up-to-date even easier! Go to <http://pubs.ama-assn.org/misc/alerts.dtl> to sign up for this free service.

Metabolic Effects of Fructose and the Worldwide Increase in Obesity

LUC TAPPY AND KIM-ANNE LÊ

Department of Physiology, Faculty of Biology and Medicine, University of Lausanne, Lausanne, Switzerland

I. Introduction	24
A. General context	24
B. Historical perspective	24
C. Fructose consumption	24
II. Fructose Metabolism	26
A. Fructose absorption and metabolism in the gut	26
B. Hepatic metabolism	27
C. Extrahepatic metabolism	28
D. Metabolic fate of an oral fructose load in healthy subjects	28
E. Metabolic fate of an oral load of fructose in diabetic patients	29
F. Fructose and exercise	30
G. Fructose and food intake	30
III. Long-Term Effect of Fructose	31
A. Dyslipidemia	31
B. Ectopic lipid deposition in the liver and skeletal muscle	32
C. Impaired glucose homeostasis and insulin resistance	33
D. Effects of fructose overfeeding versus glucose overfeeding	34
E. Uric acid metabolism	35
F. High blood pressure	36
G. Mineral metabolism	36
IV. Does Free Fructose Exert Different Effects Than Fructose Bound to Sucrose?	36
V. Does Fructose Play a Role in the Pathogenesis of Metabolic Diseases?	37
A. Fructose and energy intake	37
B. Fructose and body weight	37
C. Fructose intake and diabetes	38
D. Fructose intake and cardiovascular risk factors	38
E. Fructose intake and nonalcoholic steatohepatitis	39
F. General conclusions regarding epidemiological studies	39
VI. Perspectives	40

Tappy L, Lê K-A. Metabolic Effects of Fructose and the Worldwide Increase in Obesity. *Physiol Rev* 90: 23–46, 2010; doi:10.1152/physrev.00019.2009.—While virtually absent in our diet a few hundred years ago, fructose has now become a major constituent of our modern diet. Our main sources of fructose are sucrose from beet or cane, high fructose corn syrup, fruits, and honey. Fructose has the same chemical formula as glucose ($C_6H_{12}O_6$), but its metabolism differs markedly from that of glucose due to its almost complete hepatic extraction and rapid hepatic conversion into glucose, glycogen, lactate, and fat. Fructose was initially thought to be advisable for patients with diabetes due to its low glycemic index. However, chronically high consumption of fructose in rodents leads to hepatic and extrahepatic insulin resistance, obesity, type 2 diabetes mellitus, and high blood pressure. The evidence is less compelling in humans, but high fructose intake has indeed been shown to cause dyslipidemia and to impair hepatic insulin sensitivity. Hepatic de novo lipogenesis and lipotoxicity, oxidative stress, and hyperuricemia have all been proposed as mechanisms responsible for these adverse metabolic effects of fructose. Although there is compelling evidence that very high fructose intake can have deleterious metabolic effects in humans as in rodents, the role of fructose in the development of the current epidemic of metabolic disorders remains controversial. Epidemiological studies show growing evidence that consumption of sweetened beverages (containing either sucrose or a mixture of glucose and fructose) is associated with a high energy intake, increased body weight, and the occurrence of metabolic and cardiovascular disorders. There is, however, no unequivocal evidence that fructose

intake at moderate doses is directly related with adverse metabolic effects. There has also been much concern that consumption of free fructose, as provided in high fructose corn syrup, may cause more adverse effects than consumption of fructose consumed with sucrose. There is, however, no direct evidence for more serious metabolic consequences of high fructose corn syrup versus sucrose consumption.

I. INTRODUCTION

A. General Context

Humans, and many mammals, tend to overfeed themselves when presented with a palatable diet, and this trivial observation outlines the importance of sensorial properties of foods in our nutrition. Amongst the factors that make a food palatable, a sweet taste is highly favored by many. This natural attractiveness toward sweetness, which has been translated in many idiomatic expressions (a sweet life, to keep someone sweet, to sweet-talk someone, . . .), is responsible for a substantial consumption of sugars by modern humans.

Sugars are naturally occurring sweeteners, the most common in our nutrition being sucrose, fructose, and glucose. Fructose and glucose are monosaccharides present in small amounts in fruits and honey, while sucrose, a disaccharide formed by one molecule of glucose linked to one molecule of fructose through an α 1–4 glycoside bond, is found in substantial amounts in sugar cane and beets.

Given the substantial participation of fructose in our everyday diet, it appears important to delineate its consequences and metabolic effects. This review therefore focuses on the metabolic effects of dietary fructose and its possible consequences on health. Data collected specifically in humans are addressed, but some studies done on animals are discussed when relevant. Caution should be however called upon their relevance to humans given the very high fructose intake used in many animal studies.

B. Historical Perspective

1. Evolution of fructose consumption through history

Humans have not always been the high sugar-consumers that we are today. Man's ancestors, the Cro-Magnon men during the Paleolithic, obtained their food from hunting and gathering, and their diet was mainly composed of meat. Their nutritional intake was high in protein, moderate in fat, and low in carbohydrates (63). At this time, fruit and berries represented the major source of carbohydrate, while starch consumption was low. It can be speculated that man's natural taste attraction for sweetener dates from these ages, when sugar was scarce.

Honey was the main sweetener, used in limited amounts, until the Crusades, during which time western

Europeans got acquainted with sugar used in the Middle East. Consumption of sugars remained however quite low until the 18th century, when both the development of intercontinental trade with distant countries where sugar cane abounded and technological improvement to extract and refine sugars became available. Sugar was no longer a luxury product and quickly became extremely popular. It was initially mostly extracted and refined from cane and imported to Europe and North America, and later was also prepared from beets. Sugar was first consumed as a sweetener in tea and coffee, the new fashionable drinks, but its use was rapidly extended to be preparation of new tasty and palatable food items such as bakeries and sweets. In England, sugar consumption increased by 1,500% between the 18th and 19th centuries (127), and by the turn of the 20th century, sugars had become one major constituent of our diet.

Sucrose remained the almost exclusive sweetener to be consumed, with only small amounts of glucose and fructose ingested essentially with fruits, until the 1960s when the food industry developed and put into use technologies allowing to extract starch from corn, hydrolyze it to glucose, and convert part of the glucose into fructose through enzymatic isomerization (136). This resulted in the production of corn-derived sweeteners, among which was high fructose corn syrup (HFCS) (90, 241). The high sweetening power of HFCS, its organoleptic properties, its ability to confer a long shelf-life and to maintain a long-lasting moisture in industrial bakeries, together with its low cost, contributed to a very rapid increase in its consumption at the expense of sucrose. HFCS can be produced with various fructose-to-glucose ratios, with the most commonly used being HFCS-55, containing 55% fructose and 45% glucose, i.e., a fructose-to-glucose ratio close to the 1:1 ratio found in sucrose.

C. Fructose Consumption

1. Methods for assessing fructose consumption

Assessing the fructose intake in a population is not an easy task, since fructose intake is not specifically recorded as a variable in most surveys or databases. The two commonly used methods are "per capita disappearance data" and "individual food intake reports."

Per capita disappearance data in the United States have been reported on a yearly basis since 1909. Sweetener disappearance data are available for sucrose, HFCS, and honey. They include both individual consumption and

industrial use for food processing and may thus overestimate real fructose intake due to losses and waste at the consumer level. They nonetheless provide useful estimates of trends in added sugar consumption (<http://www.ers.usda.gov/briefing/sugar/data.htm>).

Individual food intake records are usually performed over a 1- to 3-day period. By combining the recorded intake of specific foods with their fructose content, it is possible to estimate the individual fructose consumption. This method provides a more accurate view of the fructose intake at the individual level, but extrapolation to whole populations is dependent on population sample selection (163).

2. Fructose intake between 1970 and 2007 in the United States

According to United States Department of Agriculture (USDA) reports, per capita added sugar consumption amounted to ~90 g/day in 1970 (225). By this time, HFCS consumption was close to zero. Important changes occurred between 1970 and 1985, when sucrose disappearance progressively declined by almost 50% (Fig. 1A). This decrease in sucrose consumption was mirrored by a sharp

increase in HFCS disappearance. In 2007, sucrose represented 45% and HFCS 41% of the total added sweeteners disappearance, the remaining 14% being accounted for by glucose syrup, pure glucose, and honey. Per capita disappearance of total caloric sweeteners increased by 15% between 1970 and 2007 (<http://www.ers.usda.gov/briefing/sugar/data.htm>).

Analysis by Park et al. (163) of food dietary records obtained in 1977–78 in the USDA Nationwide Food Consumption Survey reported that the average daily fructose intake was 37 g in the United States population. This dietary survey also provided useful information regarding the sources of fructose intake and the differences by age classes and by gender. Sugar-sweetened nonalcoholic beverages, such as soft drinks, appeared as the major source of fructose for all classes of age considered, except for children younger than 6 yr and adults older than 50 yr. The highest consumers were adolescents and young adults (19–22 yr) of both sexes. The third National Health and Nutrition Examination Survey, performed in 1988–94 (NHANES III), allows assessment of the evolution of fructose intake between 1977 and the 1990s. Average daily fructose intake in NHANES III was 54.7 g, corresponding to a 46% increase over a 10- to 16-yr period. Males tended to consume higher absolute amounts of fructose than females, but the difference was not significant when intakes were reported as a percentage of total energy intake. Adolescents and young adults remained the highest fructose consumers, and people with the lowest income consumed more fructose than those with the highest incomes. Soft drinks were the main source of fructose intake for any class of age considered, including this time young children and older adults.

A recent reappraisal of these estimates, based on data collected from the NHANES 1999–2004 study, estimated an average fructose intake of 49 g/day. It also documented that HFCS consumption had continuously increased over the past three decades and accounted for 42% of total caloric sweetener consumption in 1999–2004 versus 16% in 1977–1978. Interestingly, this analysis also documented that total energy intake increased by 18% and total carbohydrate intake by 41% during the same period, while contribution of fructose to carbohydrate intake remained nearly constant (135).

On the basis of both per capita disappearance analysis and individual food records analysis, there is no doubt that fructose consumption has increased over the past four decades in the United States, that teenagers and young adults are the highest consumers, and that the sweetened beverages are the main dietary source of fructose. However, a few points should be considered.

Free fructose consumption dramatically increased between 1970 and 2007, as illustrated by impressive exponential curves (33). However, this rise was merely due to the increased use of HFCS, in which fructose is under

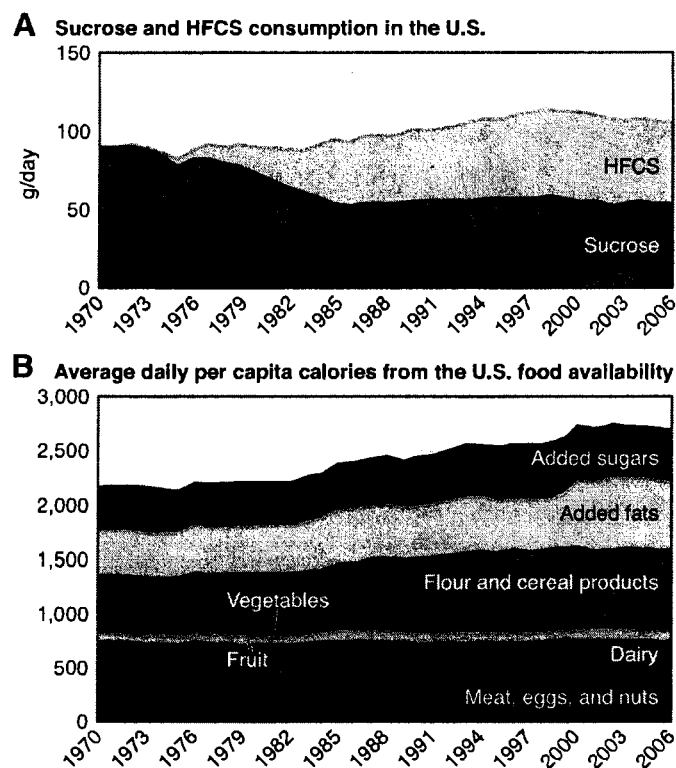


FIG. 1. Evolution of the consumption of high-fructose corn syrup (HFCS) and sucrose in the United States between 1970 and present. HFCS has increased rapidly to replace 50% of the sucrose consumption. Over this period, not only total sugar consumption but also total calorie intake and total fat intake have increased significantly. Source: USDA (<http://www.ers.usda.gov>).

its free form, and was mirrored by a decrease in the consumption of fructose bound to glucose in sucrose. Since there is presently no evidence that the metabolic effects of HFCS-55 (the most widely consumed form of HFCS, containing 55% fructose) differ from those of sucrose (see sect. iv), one should rather consider total fructose, i.e., free plus bound to glucose in sucrose, consumption to assess the nutritional and metabolic impact of fructose.

Over the past decades, there was a general trend toward an increased total energy intake, with all types of foods confounded. From the USDA data (225), total energy intake may have increased by 24%. This includes of course the 15% increase in added sugars discussed above. However, most other nutrients showed the same pattern: fruit consumption also increased by 29%, flour and cereals products increased by 42%, and there was a sharp 55% increase in added fat consumption. Relative proportion of these products remained however comparable (<http://www.ers.usda.gov/>) (Fig. 1B).

3. Fructose intake worldwide

In other parts of the world, data are scarcer than in the United States. The only official source available is the International Sugar Organization, which reports yearly worldwide statistics (104a). Overall, the world average per capita sugar consumption has increased by 16% over the past 20 years, from 56 g/day in 1986 to 65 g/day in 2007. South America and Oceania are the highest sugar consumers, followed by Europe, while low sugar consumption is recorded for Asia and Africa. Sugar consumption recently increased in all part of the world except Oceania, and the most impressive rise was observed in Asia, with a 50% increase (Table 1).

TABLE 1. World per capita consumption of sugar

Continent	Per Capita Consumption of Sugar, g/day	
	1986	2006
Europe	107	124
North America*	83	88
South America	117	143
Asia	30	45
Africa	40	46
Oceania	122	118

Values do not include high fructose corn syrup (HFCS). *Lower values compared with Europe are essentially accounted for by a high consumption of HFCS: 1985, 40 g/day; 2005, 52.4 g/day (<http://www.com.org/percaphcs.htm>). [From the *ISO Sugar Year Book*, 2008 (104a).]

II. FRUCTOSE METABOLISM

A. Fructose Absorption and Metabolism in the Gut

Fructose is a hexose, with a chemical formula $C_6H_{12}O_6$ identical to that of glucose. It differs from glucose by the presence of a keto group in position 2 of its carbon chain, versus an aldehyde group at position 1 of the glucose carbon chain. In solution, it can be present as α - or β -pyranoside and furanoside rings.

Fructose present in the gut, whether issued from ingestion of pure fructose or of HFCS, or from the digestion of sucrose at the brush-border membrane, is transported into the enterocyte through a specific fructose transporter, GLUT5, located at the apical pole of the enterocyte. Contrary to glucose, this process does not require ATP hydrolysis and is independent of sodium absorption. Once inside the enterocyte, fructose diffuses into the blood vessels through a transport mediated by GLUT2 at the basolateral pole of the enterocyte (51, 70).

Compared with glucose, fructose absorption appears to be quantitatively limited. Some individuals may have a low capacity to absorb fructose and develop symptoms of diarrhea and flatulence after fructose loading (116, 172), more particularly when fructose is ingested without glucose (221). In rodents, GLUT5 expression is very low until weaning, but can be stimulated by fructose administration (57). Fructose absorption may also be altered by ageing, since in aged rats, absorption of carbohydrates, including fructose, is decreased (78). Fructose transport is also modulated by noncarbohydrate constituents of the diet. Thus, in rats, a diet high in saturated fatty acids (but not in polyunsaturated fatty acids) enhances intestinal fructose absorption (166).

Once inside the enterocyte, part of the fructose appears to be converted into lactate and released into the portal circulation. This intestinal lactate production appears specific for fructose and was shown, in miniature swine, to account for 12% of the absorbed fructose, versus only 2% with glucose (25). Fructose administration also produced a small rise in intestinal glucose production, suggesting that triose-phosphates were converted into glucose within the enterocyte (25). The presence of glucose-6-phosphatase activity in rodent and human intestine is indeed consistent with a gluconeogenic activity in the gut (170). The functional significance of this intestinal metabolism of fructose remains unknown. It has been suggested that intestinal gluconeogenesis may secondarily exert effects on peripheral metabolism and on food intake through neural reflexes elicited by activation of portal glucose sensors (144).

In the hamster, a high-fructose diet leads to an increase in plasma triglyceride concentrations. These triglycerides, present in the circulation under the form of chylomicrons, were shown to originate from fructose con-

version into fatty acids within the enterocyte (intestinal de novo lipogenesis), with subsequent association with apoB-48 to be released as chylomicrons (88, 126). Whether a similar pathway is active in humans and other mammals remains however unknown.

B. Hepatic Metabolism

After fructose absorption, the fructose present in the portal blood is rapidly and efficiently extracted by the liver (see Fig. 2). Fructose uptake in the liver is thought to be operated by the glucose transporter GLUT2 (43, 49). The bulk of ingested fructose is extracted at first pass in the liver where it is rapidly metabolized into fructose-1-phosphate (P) under the action of the enzyme fructokinase, which is highly specific for fructose. Fructokinase is characterized by a low K_m for fructose [~ 0.5 mM (2, 93) and a high V_{max} (estimated at ~ 3 μ mol/min per gram rat or human liver at 25°C) (2, 92)]. These properties account for a rapid metabolism of fructose in liver cells. Inherited deficiency of fructokinase leads to a rare, benign condition called hereditary fructosuria (95). The loss of fruc-

tose into the urine in this condition illustrates well the fact that fructose having escaped hepatic metabolism is poorly metabolized in extrahepatic tissues.

Subsequent steps of fructose metabolism have been described in full detail elsewhere (138, 150) and are only briefly outlined here. Fructose-1-P is further metabolized into triose-P through the action of aldolase B. Inherited deficiency of aldolase B is a rare condition leading to hereditary fructose intolerance, characterized by the occurrence of hypoglycemia upon exposure to dietary fructose and by the development of liver steatosis and cirrhosis (95).

The hepatic metabolism of fructose differs markedly from that of glucose for several reasons. First, entry of glucose in the glycolytic pathway is under the control of hexokinase IV, or glucokinase. This enzyme is characterized by a high K_m for glucose, and hence, the rate of glucose phosphorylation varies with changes in portal glucose concentration (105). Glucose-6-P is then converted to fructose-6-P, then to fructose-1,6-di-P through a reaction catalyzed by phosphofructokinase. The activity of phosphofructokinase is inhibited by ATP and citrate, which allows regulation of the reaction according to the energy status of the cell (217). Fructose-1,6-di-P is further converted into pyruvate prior to entry into the Krebs cycle. Altogether, conversion of glucose to pyruvate is regulated by insulin, which stimulates glucokinase gene expression and activates glycolytic enzymes, and by the energy status of the cell. In contrast, fructose conversion to triose-P occurs independently of insulin and is a rapid process due to the low K_m of fructokinase for fructose, and absence of negative feedback by ATP or citrate. This leads to a transient depletion of free phosphate and a decrease in ATP in liver cells in response to fructose (35, 52).

Triose-P produced from fructose can subsequently be converted into pyruvate and oxidized into CO_2 and H_2O in the tricarboxylic acid cycle. A portion of the triose-P produced is however converted into lactate to be released into the systemic circulation (27). This probably accounts for the significant increase in plasma lactate concentrations observed after fructose ingestion. This fructose-induced lactate production may be quantitatively important during intravenous fructose administration and has occasionally been associated with lactic acidosis (243). The major portion of triose-Ps produced from fructose metabolism is converted into glucose and glycogen through gluconeogenesis (30, 118). Glucose and lactate production may not be entirely independent processes: in rats, it was documented that the main portion of fructose reaching the portal circulation was taken up by periportal hepatocytes, where nearly half of it was converted into glucose, while lactate release occurred essentially in perivenous hepatocytes. This suggested that fructose-induced lactate production results in periportal conversion of fructose into glucose and the subsequent uptake and glycolysis to lactate in perivenous hepatocytes (36).

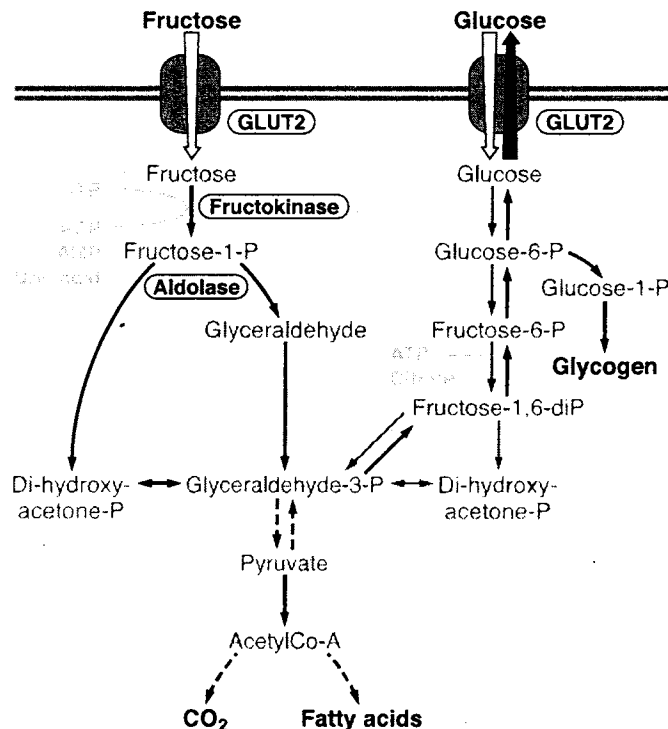


FIG. 2. Fructose metabolism in liver cells. Fructose metabolism (grey arrows) differs from glucose (black arrows) due to 1) a nearly complete hepatic extraction and 2) different enzyme and reactions for its initial metabolic steps. Fructose taken up by the liver can be oxidized to CO_2 and then converted into lactate and glucose; glucose and lactate are subsequently either released into the circulation for extrahepatic metabolism or converted into hepatic glycogen or fat. The massive uptake and phosphorylation of fructose in the liver can lead to a large degradation of ATP to AMP and uric acid.

Finally, part of the carbon atoms of fructose can be converted into fatty acids in hepatocytes through the process of de novo lipogenesis. The existence of this pathway was documented by the observation that, in the rat in vivo (17) and in isolated rat hepatocytes (47, 214), administration of [^{14}C]fructose led to ^{14}C incorporation in liver lipids. Stimulation of hepatic de novo lipogenesis can indeed be documented after acute administration of fructose, or of fructose-glucose mixtures, in humans by monitoring incorporation of infused ^{13}C -labeled acetate into very-low-density lipoprotein (VLDL)-palmitate (165, 190). In vitro data indicated that lactate rather than triose-P is the main lipogenic precursor after fructose administration and that activation of pyruvate dehydrogenase by high-fructose diets is a major regulatory step in this process (41, 59, 162). Simultaneously, fructose inhibits hepatic lipid oxidation, thus favoring fatty acid reesterification and VLDL-triglyceride (TG) synthesis (214). Although not specifically measured with fructose, stimulation of de novo lipogenesis by carbohydrate is likely to take place mainly in perivenous hepatocytes, which are characterized by active lipogenic pathways, whereas periportal hepatocytes are mainly oxidative (87).

Another metabolic effect of acute fructose administration is exerted through an increased intrahepatic fructose-1-P concentration. This rise in fructose-1-P has important indirect effects on hepatic glucose metabolism by modulating glucokinase activity. Hepatic glucokinase is a key regulatory enzyme in hepatic glucose metabolism, since it is required for the formation of glucose-6-P. Decreased activity of glucokinase secondary to heterozygous mutations indeed leads to decreased postprandial hepatic glycogen synthesis (232). Glucokinase also acts as a liver sensor for glycemia and is involved in the inhibition of hepatic glucose release by portal hyperglycemia, a process which is also impaired in patients with glucokinase mutations (203). Glucokinase activity is controlled by the concentration of its substrate glucose and by a regulatory protein, which acts as a competitive inhibitor of glucose for glucokinase. Fructose-1-P, at low concentration, antagonizes glucokinase regulatory protein, thus enhancing glucokinase activity (229). As a consequence, addition of small, so-called "catalytic" doses of fructose to a glucose meal can enhance hepatic glucose disposal (69).

C. Extrahepatic Metabolism

After ingestion of fructose, the increase in plasma fructose concentration remains in the micromolar range, indicating that first-pass hepatic extraction is close to 100%. As a consequence, fructose metabolism does not occur in extrahepatic cells to any significant extent under usual conditions. When fructose is administered parenterally, systemic plasma fructose concentrations increase up

to 1–2 mM (219). Even under such conditions, extrahepatic fructose metabolism can be expected to be small, since extrahepatic cells do not express fructokinase, and the K_m of hexokinase for fructose is high (138). In this regard, the functional significance of the intestinal fructose transporter GLUT5 being expressed in several extrahepatic tissues including the kidney and adipose tissue remains unknown (50, 129). Catheterization studies showed that, during high-dose fructose infusions, which increased plasma fructose up to 3 mM, kidney fructose uptake accounted for ~20% of total fructose metabolism (27). Such an extrahepatic fructose uptake is however unlikely to occur under physiological conditions.

D. Metabolic Fate of an Oral Fructose Load in Healthy Subjects

After ingestion of a fructose load, plasma glucose and insulin showed little changes, and plasma fructose concentrations rose only to ~50–500 μM (133, 205) (see Fig. 3). There was, however, a rapid and sharp increase in net

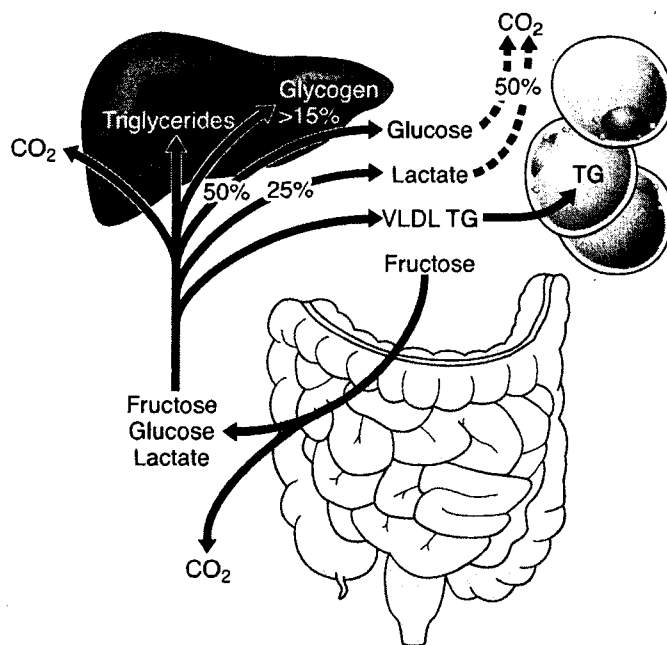


FIG. 3. Metabolic fate of an oral fructose load: after fructose ingestion, fructose metabolism takes place essentially in splanchnic tissues. In the liver, a large proportion of fructose is converted into glucose, which can be either stored as hepatic glycogen or released as plasma glucose. Part of the fructose load is converted into lactate in enterocytes and in the liver, which leads to increased lactacidemia. A small portion of fructose is converted into fatty acids; although quantitatively minor, this pathway may play an important role in the development of fructose-induced hepatic steatosis and dyslipidemia. The estimated contribution of glucose production, glycogen synthesis, lactate production, and total oxidation is indicated based on experimental data described in section 11D. Data for lipogenesis and very-low-density lipoprotein (VLDL)-triglyceride (TG) secretion are not available, but these pathways are quantitatively minor.

carbohydrate oxidation (205). Part of this oxidation is likely to take place in the liver. In addition, when ^{13}C -labeled fructose is administered, one can observe that ~50% of the fructose load recirculates as ^{13}C -labeled glucose in the systemic circulation over the next 6 h (62); this indicates that a substantial portion of ingested fructose is converted into glucose in hepatic cells, to be subsequently oxidized in extrahepatic tissues. Catheterization studies, performed in healthy human subjects fasted for 60 h, also indicated that ~50% of infused fructose was released as glucose in the systemic circulation (26). Infusion of ^{13}C -labeled fructose similarly led to an important release of ^{13}C -labeled glucose into the circulation (218, 219), supporting the view that glucose synthesis is the major pathway of hepatic fructose disposal. Interestingly, stimulation of glucose synthesis by fructose does not lead to an increase in total glucose output (219). Acute stimulation of gluconeogenesis by administration not only of fructose, but also of other gluconeogenic precursors such as lactate (109) or glycerol (106), also fails to increase total glucose output, through a process called autoregulation of glucose production, which involves an inhibition of glycogenolysis (53). It however acutely impairs insulin-induced suppression of glucose production, and hence decreases hepatic insulin sensitivity (67, 189).

A substantial portion of fructose-derived glucose appears to be directly stored as hepatic glycogen. Fructose administration increases even more hepatic glycogen concentrations than administration of an equivalent dose of glucose in both rats (118) and humans (151). In humans, hepatic glycogen synthesis has been shown to account for ~17% of an oral glucose load (167). Although hepatic glycogen synthesis after oral fructose has not been measured in humans, it can therefore be safely estimated to be at least 17%.

Part of the fructose taken up by the liver is also converted into fatty acids through the process of de novo lipogenesis, to be released into the systemic circulation with VLDL. This pathway, although potentially stimulated by fructose, represents only a minor portion of the fructose load (46, 141, 165). Finally, there is an increase in plasma lactate, which strongly suggests that hepatic conversion of fructose to lactate, as observed in animals and in humans during intravenous fructose infusion, is one significant pathway for hepatic fructose disposal (36, 207, 219). Catheterization studies indicated that, in healthy fasted subjects, ~25% of ingested glucose was released as lactate from the splanchnic bed during intravenous fructose infusion (26, 66).

One of the effects of fructose administration is a marked suppression of nonesterified fatty acids in the blood, which indicates an inhibition of adipose tissue lipolysis (205). The integrated postprandial inhibition of plasma nonesterified fatty acids was even of comparable

magnitude after ingestion of equivalent amounts of glucose or fructose (28). Although very modest compared with what is observed after glucose ingestion, the slight increase in plasma insulin elicited by fructose is sufficient to explain this effect due to the extreme sensitivity of adipose cells to insulin (205). It has also been proposed that fructose-induced hyperlactatemia may contribute to the suppression of adipose lipolysis (1).

Fructose administration, as glucose, increases resting energy expenditure. The thermic effect of fructose is, however, significantly higher than with glucose, and this effect is observed with both fructose alone (205) and with fructose added to a meal (191). This is best explained by the high ATP need linked to fructose-induced gluconeogenesis, with possible contribution of de novo lipogenesis (204). It has been shown that an activation of the sympathetic nervous system plays a role in glucose-induced thermogenesis (185, 239). A role of sympathetic nervous system activation is, however, unlikely to be operative with fructose, since fructose infusion does not activate the sympathetic nervous system (234).

E. Metabolic Fate of an Oral Load of Fructose in Diabetic Patients

The glycemic index of fructose is very low compared with glucose (19 and 100, respectively) (82). This property initially elicited a great interest for the use of fructose as a potential beneficial sweetener in patients with diabetes mellitus. One further characteristic of fructose, which suggested that it was well suited for diabetic patients, is that fructose does not require insulin either for its transport into hepatic cells or for the initial steps of its hepatic metabolism. When administered to diabetic patients, fructose indeed produced minor increases in plasma glucose and insulin concentrations compared with glucose (54, 56). The plasma insulin response to fructose was however markedly enhanced in diabetic patients compared with nondiabetic subjects. The stimulation of carbohydrate oxidation and of gluconeogenesis after fructose ingestion appeared globally similar in healthy nondiabetic subjects and in diabetic patients (161, 196). As in healthy subjects, the enhanced gluconeogenesis induced by fructose appeared to be compensated by an autoregulatory process, involving mirror inhibition of glycogenolysis, so that overall glucose output and glycemia did not change to any great extent (161). Of interest, glucose-induced thermogenesis is frequently blunted in insulin-resistant patients, while fructose-induced thermogenesis remains comparable to that observed in controls (196). This is likely explained by the fact that, in insulin-resistant subjects, intracellular glucose metabolism is decreased, leading to lower glucose-induced thermogenesis, while hepatic fructose metabolism is not impaired.

F. Fructose and Exercise

Physical exercise requires a continuous supply of energy to the working muscle, and muscle contraction increases muscle glucose oxidation by severalfold (94). Glucose oxidized by muscle during exercise originates either from blood glucose through exercise-induced translocation of GLUT4 (242), or from muscle glycogen. Muscle fatigue is a complex phenomenon, still incompletely understood, in which a decrease in glycemia and/or exhaustion of muscle glycogen store can play a major role (9). The development of sport drinks and supplements, aimed at preventing a drop in glycemia during exercise and sparing muscle glycogen oxidation, has therefore been the focus of intense research. In this context, fructose has attracted considerable attention.

Fructose can indeed be metabolized during exercise. When infused intravenously during an exercise of moderate intensity, it was shown that ~80% of the dose of fructose administered was metabolized in splanchnic tissues to be released as glucose, pyruvate, and lactate which were subsequently metabolized in working muscle. The remaining 20% were metabolized directly in working and resting skeletal muscle (5). Due to intravenous rather than oral administration, fructose concentration was however very high (up to 6 mM), and it is unlikely that such direct muscle fructose metabolism occurs with the low plasma fructose concentrations elicited by oral fructose. When oxidation of oral glucose or fructose drinks were compared during an exercise of moderate intensity, it was reported that fructose oxidation was comparable to that of glucose (3), or slightly lower (107), and that fructose conversion into glucose accounted for about half of the total glucose production (107). Thus, even though fructose ingestion per se does not increase plasma glucose concentration, it may nonetheless contribute to maintain glycemia by sustaining glucose production during exercise (107).

Sport drinks aim to prevent a drop of glycemia and to provide exogenous glucose to the working muscles. When oral glucose was administered, exogenous glucose metabolism was however limited to a maximum of ~1.0–1.1 g/min, most likely due to saturation of intestinal glucose transport when higher doses are administered (111). When a mixture of glucose and fructose was administered, total carbohydrate oxidation could however be further enhanced by ~40% (110, 235). This may be explained by the different transport systems used for intestinal absorption of glucose and fructose and by their different metabolism, i.e., essentially hepatic for fructose versus primarily within the skeletal muscle for glucose during exercise. It was also reported that moderate doses of fructose reduced the perception of fatigue and stress during exercise (186) and improved exercise performance during a cycling exercise (58).

Regarding the effects of fructose on muscle glycogen synthesis, few contradictory studies were performed. One study showed that fructose was more efficient than glucose to prevent the decrease in muscle glycogen (assessed from a postexercise muscle biopsy) (125), but another study, using similar techniques, observed no difference between fructose and glucose drinks (117). One study compared muscle glycogen recovery after exercise with glucose and fructose feeding. In this study, muscle glycogen repletion, evaluated with ¹³C-NMR spectroscopy, was considerably more efficient with glucose than with fructose (227).

On the basis of these studies, the use of fructose as a supplement in sports drinks may possibly have modest advantages, which however remain to be better documented by larger studies in which performance or endurance are the primary outcome. One concern with the use of fructose during exercise is that it may be incompletely absorbed from the gut and get fermented by intestinal bacteria (145), which may limit the amount that can be administered without adverse gastrointestinal symptoms.

G. Fructose and Food Intake

The effects of fructose on appetite remain controversial. While some studies have shown that ingestion of a fructose load alone reduces subsequent food intake (180, 216), this effect was not observed when fructose was ingested together with a mixed meal (181). There are several reasons to suspect that fructose, based on its known physiological effects, will elicit lower satiation than equivalent doses of glucose or complex carbohydrates. First, the postprandial rise in glycemia plays, directly or indirectly, an important role in the mechanisms controlling satiety and food intake. This effect is likely blunted with fructose, since its glycemic index is about fivefold lower than that of glucose. Second, ingestion of fructose-containing meals elicits a lesser suppression of the appetite-stimulating hormone ghrelin and a lower increase in leptin than meals containing an equivalent amount of glucose (207), which suggests that fructose may be less efficient than glucose to suppress food intake. Although acute fructose ingestion is not expected to stimulate leptin secretion, significant increases in fasting leptin concentrations were observed after 1–4 wk of fructose overfeeding (122); this indicates that fructose overfeeding exerted metabolic effects on adipose cells, which may in the long term contribute to suppress food intake. It was also observed that body weight gain was similar in overweight women subjected to a 10-wk supplementation with either glucose or fructose, suggesting that, in the long term, the effects of fructose and glucose on food intake may not differ in a significant way (199).

In addition to producing a lesser secretion of leptin compared with equivalent doses of glucose, it was observed

that a high fructose intake impairs leptin's actions, thus causing a state of leptin resistance. In fructose-fed rats, the anorectic effects of intraperitoneally administered leptin were nearly abolished; this corresponded to a significant decrease in hypothalamic signal transducer and activator of transcription-3 (STAT-3) phosphorylation in response to fructose (193). It was also observed that, in rats, a high-fructose diet caused hepatic leptin resistance through an enhanced amount of suppressor of cytokine 3 and through decreased serine/threonine phosphorylation of key proteins in leptin signaling. At the level of the liver, where leptin promotes fat mobilization and oxidation, this hepatic leptin resistance may contribute to the pathogenesis of fructose-induced non-alcoholic fatty liver disease (NAFLD) (233).

One intriguing observation has been recently reported: it is well known that glucose is the primary fuel for the brain and that changes in glucose concentrations may act as a signal informing the brain about the metabolic and nutritional state of the organism. Accordingly, administration of glucose in the cerebral ventricles suppressed food intake through an increase in ATP-to-AMP ratio and an increased malonyl-CoA content in specialized hypothalamic areas (97). When fructose was infused intracerebrally instead of glucose, opposite effects were observed, i.e., a drop in ATP-to-AMP ratio, a stimulation of AMPK activity, lowered malonyl-CoA, and increased food intake (42). The physiological significance of this observation remains however unclear, since plasma fructose concentration will never exceed the micromolar range under physiological conditions, and hence fructose ingestion is unlikely to increase fructose concentration in the cerebrospinal fluid.

III. LONG-TERM EFFECT OF FRUCTOSE

Given the low glycemic rise induced by fructose ingestion, and the fact that its metabolism does not strictly require insulin secretion, several studies evaluated the metabolic effects of replacing part of the carbohydrate intake of patients with type 2 diabetes mellitus with fructose. These studies reported conflicting results, in part explained by variations in experimental conditions (duration of treatment, type of carbohydrate replaced by fructose in the diet, etc.). Only about half of them resulted in a significant reduction in blood glucose (10, 14, 16, 55, 56, 85, 139, 154, 210, 211). These studies however pointed out the fact that fructose was associated with a substantial increase in plasma triglyceride and a decrease in high-density lipoprotein (HDL)-cholesterol.

In animal models, numerous studies have addressed the effects of diets enriched with fructose or sucrose. As a whole, they indicated that high-fructose/high-sucrose diets lead to several adverse metabolic and cardiovascular effects, including dyslipidemia, insulin resistance, hypertension, hyperuricemia, and weight gain (24, 91, 123).

A. Dyslipidemia

It has been long recognized that feeding a high-fructose diet for more than 1 wk increases plasma total- and VLDL-triglycerides in healthy volunteers and in patients with insulin resistance or type 2 diabetes. An increase in total cholesterol was also encountered in some of these studies (14, 55, 133). The mechanisms underlying fructose-induced dyslipidemia have been partially elucidated (see Fig. 4). Plasma triglyceride kinetics were measured in rats fed high-sucrose, -glucose, or -fructose diets: it was observed that, compared with glucose, fructose and sucrose both increased triglyceride production and decreased triglyceride clearance (113). Fructose, by providing large amounts of hepatic triose-phosphate as precursors for fatty acid synthesis, is highly lipogenic. It has indeed been observed in several studies that hepatic de novo synthesis is stimulated after acute fructose ingestion, with fructose contributing to the synthesis of both the glycerol- and the fatty-acyl parts of VLDL-triglycerides (46, 165). Fructose may, in addition, increase the expression of key lipogenic enzymes in the liver. It has been shown to induce the expression of the factor of transcription SREBP-1c, the principal inducer of hepatic lipogenesis (137, 194). Furthermore, this effect was independent of changes in insulin concentrations (137, 147). This effect

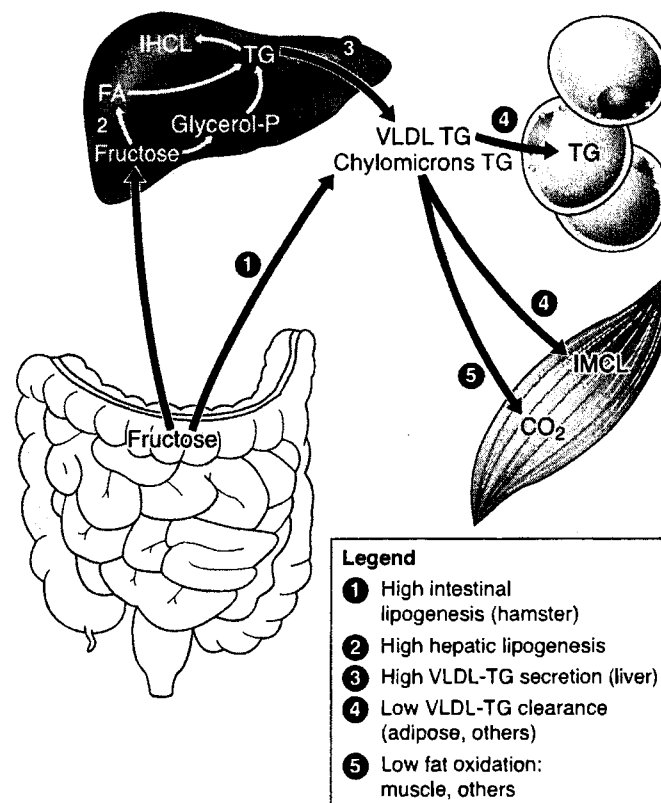


FIG. 4. Possible mechanisms involved in fructose-induced dyslipidemia.

of fructose on SREBP-1c was further shown to require peroxisome proliferator-activated receptor γ coactivator 1 β (PCG-1 β). Fructose also activates the hepatic transcription factors carbohydrate-responsive element binding protein (ChREBP), which upregulates the expression of hepatic fatty acid synthase and acetyl-CoA carboxylase (64, 118). A high-fructose diet increases the expression of the enzyme glucose-6-phosphate dehydrogenase, the first enzyme in the hexose monophosphate pathway, and intermediary substrates of the hexose-monophosphate shunt have been proposed as being responsible for activation of ChREBP (118, 226).

The role played by a stimulation of hepatic de novo lipogenesis in fructose-induced hypertriglyceridemia is supported by 1) the positive correlation observed between fractional hepatic de novo lipogenesis and fasting triglycerides in healthy subjects fed an isocaloric, high-sugar diet (100) or a hypercaloric, high-fructose diet (76) and 2) the fact that a 2-wk supplementation with fish oil reduced both hepatic de novo lipogenesis and fasting triglycerides in healthy subjects overfed with fructose (76). In addition to this increase in fasting plasma triglycerides, acute fructose administration also increased the postprandial rise in plasma triglycerides due to an impaired clearance of triglyceride-rich lipoprotein (46). The same effect was observed with chronic high fructose intake. In overweight women, postprandial triglyceride excursions were enhanced by the consumption of fructose-sweetened beverages over a 10-wk period, indicating that fructose impaired triglyceride clearance (202). This suggests that impaired triglyceride-rich lipoprotein clearance contributes to the hyperlipidemia induced by high-sugar and high-fructose diets (164). This effect of fructose was significantly increased in obese hyperinsulinemic women compared with normal-weight women, suggesting that fructose may produce more severe alterations of lipid homeostasis in insulin-resistant individuals (208). Interestingly, administration of equivalent amounts of pure fructose, sucrose, mixtures of glucose and fructose, or HFCS led to similar increases in postprandial triglyceride; since sucrose, glucose + fructose mixture and HFCS contained approximately half the amount administered with pure glucose, this suggested that coingestion of glucose significantly potentiated the hypertriglyceridemic effect of fructose (198).

Apolipoprotein E is known to be associated with the metabolism of triglyceride-rich lipoproteins. Three common alleles of apoE are encountered in the population: APOE*E2 (E2), APOE*E3 (E3), and APOE*E4 (E4). In population studies, plasma triglycerides are higher in individuals with E2 and E4 alleles (60). It was indeed reported that hypertriglyceridemia was related to sucrose consumption only in individuals with the E2 allele (74). These isolated observations were however not confirmed by an intervention study in which subjects were submitted to an increase in dietary

sucrose intake of 40 g/day: in these subjects, sucrose supplementation failed to alter fasting or postprandial triglycerides, irrespective of the presence or not of the APOE2 allele (75). The possible relationship between apoE polymorphism and the hypertriglyceridemic effect of fructose/sucrose needs therefore to be further documented by larger studies or with higher dietary intakes.

Interestingly, both animal and human studies indicate a gender difference in fructose-induced hypertriglyceridemia: in male rats, chronic high-fructose or high-sucrose diets caused hypertriglyceridemia. In contrast, female rats appeared protected against fructose- or sucrose-induced changes in metabolism (10, 11, 96). This protection was no longer present after oophorectomy, suggesting that female sex hormones may confer protection against the effects of a fructose diet (11). In humans, data are more scarce. Several studies nonetheless reported that the increase in plasma triglyceride induced by fructose feeding was markedly blunted in premenopausal, healthy females compared with age-matched males (12, 15, 198).

The various studies discussed above have addressed the hyperlipidemic effects of fructose, using a large range of dietary fructose/sucrose intake. Since many of the aforementioned studies used a high amount of dietary fructose, the effects of usual fructose intake on plasma triglyceride remain disputed. A meta-analysis (131), compiling the results of all published studies having evaluated the effects of dietary fructose (excluding studies done with HFCS), concluded that a fructose intake >50 g/day (i.e., close to average daily intake in the United States; see sect. II) was associated with increased postprandial triglyceride excursions, while a fructose intake >100 g/day was associated with increased fasting triglycerides.

B. Ectopic Lipid Deposition in the Liver and Skeletal Muscle

In addition to altering plasma lipid profile, fructose may also modulate intracellular lipid deposition (so-called "ectopic lipids," i.e., deposition of triglyceride in the cytoplasm of nonadipose cells, such as hepatocytes, muscle fibers, or endocrine cells; Ref. 224). Such ectopic lipid deposition in the liver and skeletal muscle is closely linked to tissue-specific insulin resistance (224). In rodents, a high-sucrose diet rapidly, within 1 wk, increased intrahepatic fat deposition (159). This effect of fructose may involve both a stimulation of de novo lipogenesis through an enhanced intrahepatic synthesis of triose-phosphate precursors and an increased expression of lipogenic genes (Fig. 5). At the molecular level, it was suggested that mechanisms may involve an inhibition of PPAR α in liver cells, a stimulation of hepatic de novo lipogenesis and a reduced hepatic lipid oxidation (183). This deposition of intrahepatic fat in response to fructose

Mechanisms for fructose-induced hepatic de novo lipogenesis

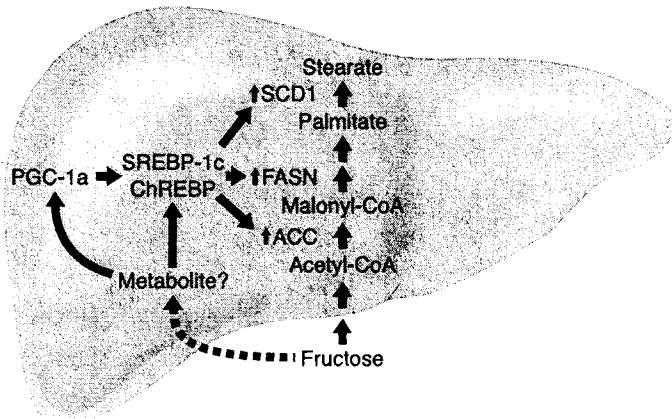


FIG. 5. Mechanisms for fructose-induced de novo lipogenesis: fructose acutely and chronically increases intrahepatic de novo lipogenesis. Stimulation of fatty acid synthesis can be explained by 1) the unregulated provision of trioses-P and acetyl-CoA secondary and 2) an increased expression of key lipogenic genes induced by chronically high fructose intakes. A high-fructose diet stimulates SREBP-1c and ChREBP through unknown mechanisms; stimulation of the hexose-monophosphate pathway and increased concentration of metabolites such as xylulose-5-phosphate have been proposed. Coactivation of SREBP-1c by PGC-1 β appears to be involved.

was shown to require PGC-1 β , which may act as a coactivator of SREBP-1c. Interestingly, inhibition of PGC-1 β in rats prevented both hepatic fat deposition and insulin resistance in response to a high-fructose diet (148).

In the early stage of sucrose overfeeding, rodents thus develop significant alterations of hepatic metabolism and of hepatic insulin sensitivity, with relatively little alterations of glucose homeostasis and no significant alterations of extrahepatic insulin sensitivity. However, when the high-sucrose diet is sustained over a few more weeks, accumulation of intramyocellular lipids and muscle insulin resistance develop (159).

In humans, accumulation of intrahepatic fat following fructose ingestion has been less documented. It has been reported that overfeeding healthy male volunteers with 1.5 g·kg fructose body wt⁻¹·day⁻¹ (corresponding roughly to the content of 2 liters of standard soda beverages) did not significantly alter fat or muscle liver content (122). However, administration of twice as much fructose over only 7 days induced a significant increase in hepatic and intramyocellular fat content (121). The increase in intrahepatic fat positively correlated with the increase in fasting VLDL-TG, suggesting that these two parameters may be driven by a common mechanism, presumably a stimulation of hepatic de novo lipogenesis. Interestingly, the increase in plasma VLDL-TG in intrahepatic fat content was enhanced in nondiabetic offspring of patients with type 2 diabetes mellitus. This suggests that the metabolic effects of fructose may be dependent on the ge-

netic environment. Given the fact that offspring had a lower insulin sensitivity than subjects without a family history of diabetes, this may also indicate that the dyslipidemic effects of fructose are enhanced by the presence of insulin resistance (121).

C. Impaired Glucose Homeostasis and Insulin Resistance

The relationship between disturbed lipid metabolism and insulin resistance has been recognized since the seminal work of Sir Philip Randle in the 1960s (171). While it was initially thought that increased nonesterified fatty acids (NEFA) concentration were the prime actors in lipid-induced insulin resistance, it is now generally admitted that both high NEFA and high plasma triglyceride concentrations are related to insulin resistance (195).

Several studies have pointed to the deleterious effect of fructose on glucose metabolism and insulin sensitivity. Indeed, a high-fructose diet increased glucose and insulin responses to a sucrose load (89), increased fasting glycemia (130), and led to hepatic insulin resistance in healthy men (76). Insulin resistance is closely linked to lipid metabolism disorders; more specifically, insulin-resistant subjects have higher ectopic lipid deposition, which may generate toxic lipid-derived metabolites, such as diacylglycerol, fatty acyl CoA, and ceramides. The presence of these metabolites in the intracellular environment leads to a higher serine/threonine phosphorylation of insulin receptor substrate-1 (IRS-1), which has been shown to reduce insulin signaling (195).

In rodent models, high-fructose or high-sucrose diets were clearly associated with the development of insulin resistance and with disturbed glucose homeostasis. In rats fed a diet in which sucrose was substituted for starch, several alterations of glucose and lipid metabolism developed over time (156). The earliest event was an increase in hepatic triglyceride content, which could be observed already after 1 wk (158, 159, 168); at this stage, fasting hormone and substrate concentrations were not changed, nor was body composition. There was however an impaired suppression of endogenous glucose production, indicating hepatic insulin resistance (158, 159, 168). Between 2 and 5 wk, fasting hyperinsulinemia developed, indicating whole body insulin resistance. The decrease in insulin's actions could indeed be documented by euglycemic, hyperinsulinemic clamps, showing a decreased insulin-mediated glucose disposal after 8 wk. This sucrose-induced insulin resistance was independent of changes in body composition. The mechanism in rodents may involve alteration of postreceptor insulin signaling. Indeed, sucrose did not alter the amount of insulin receptor, IRS-1 or IRS-2, or phosphatidylinositol 3-kinase (PI3K) in hepatocytes; phosphorylation of insulin receptors upon expo-

sition to insulin was not altered, but phosphorylation of IRS-1 and IRS-2 was reduced, indicating that sucrose impaired postreceptor insulin signaling; unexpectedly, PI3K activity was increased, suggesting a possible compensatory mechanism (157). In skeletal muscle of rats, both a high-sucrose diet (73) and a high-fructose diet (73) decreased insulin-induced insulin receptor and IRS-1 phosphorylation. This effect was observed only in living animals but was not reproduced when measuring insulin-mediated glucose disposal of isolated muscles, indicating that the effect of fructose on muscle required the living environment (115).

Although, in most studies, fructose elicited both hepatic insulin resistance and altered hepatic/extrahepatic lipid metabolism, some observations suggest that these two effects may be distinct. Thus, in healthy males, fructose overfeeding increased hepatic de novo lipogenesis and plasma triglycerides and decreased hepatic insulin sensitivity; under such conditions, supplementation with fish oil, which inhibited de novo lipogenesis, efficiently reduced plasma triglycerides but failed to normalize hepatic insulin sensitivity (76). Moreover, a high-fructose diet increased intrahepatic lipid deposition in humans, while hepatic insulin sensitivity remained unchanged (121). In rats, a diet rich in fructose and trans fatty acid also causes hepatic insulin resistance and hepatic steatosis, but here also, fructose appears more related to hepatic insulin resistance while trans fats were more involved in the development of steatohepatitis (209).

It was further observed that sucrose elicited stress responses in hepatocytes, which involved activation of the c-Jun terminal kinase (JNK). Changes in the redox state of the cells upon exposure to sucrose may be responsible for this activation of JNK. Furthermore, normalization of JNK activity in hepatocytes isolated from sucrose-fed rats normalized insulin signaling. In addition, it was documented that the effects of sucrose on JNK activity and insulin sensitivity in the liver were essentially due to the fructose component of sucrose (236–238). Fructose administration was also shown to exert a marked oxidative stress on the organism (37). Providing fructose with honey, which is naturally rich in antioxidant substances, prevented both the oxidative stress induced by fructose and the reduction of insulin sensitivity (38).

Fructose may also possibly decrease insulin sensitivity through changes in the gut microbial flora and/or alterations of intestinal permeability. It is now recognized that insulin resistance in obese patients is associated with markers of inflammation, such as C-reactive protein or proinflammatory cytokines, and with inflammation of adipose tissue (86). Recently, it was observed that a high-fat diet can lead to enhanced intestinal permeability and alterations of intestinal bacterial flora, thus resulting in an increase of the plasma concentration of bacterial lipopolysaccharides, or endotoxin. Low-grade endotoxemia

in turn activates inflammatory pathways and impairs insulin's action, leading to the development of insulin resistance (39, 40). As for a high-fat diet, a high-fructose diet was shown to increase plasma concentrations of endotoxin (212). Furthermore, mice fed a high-fructose diet were protected against both endotoxemia and fatty liver infiltration by an antibiotic treatment, suggesting that part of the metabolic effects of fructose were mediated by changes in the microbial flora (20).

In summary, there is no doubt that high-fructose feeding can cause insulin resistance in rodents. The evidence in humans is less impressive: fructose produces a slight impairment of hepatic insulin's actions, but does not reduce whole body insulin sensitivity. Interactions between fructose and fat or total energy intake remain to be assessed. Regarding the mechanisms possibly linking fructose to insulin resistance (Fig. 6), altered lipid metabolism and lipotoxicity secondary to stimulation of de novo lipogenesis, or fructose-induced oxidative stress may be involved. In addition, fructose may impair endothelial function through increased uric acid production, thus contributing to so-called "preceptor" insulin resistance (see sect. mC).

D. Effects of Fructose Overfeeding Versus Glucose Overfeeding

The intake of naturally occurring free fructose with fruits and honey is relatively low in our western-type diet and accounts for only ~15% of total fructose intake in the United States (135). Under everyday life conditions, fructose is essentially consumed as sucrose, with the corollary that fructose and glucose intake vary in parallel. This

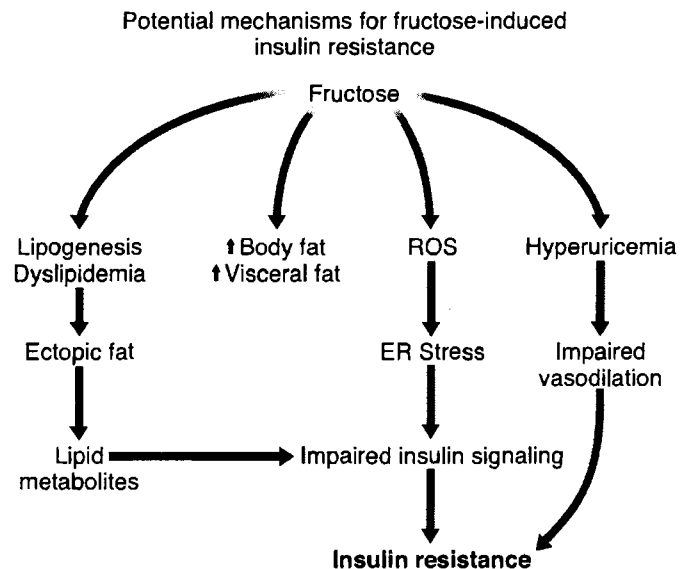


FIG. 6. Summary of the potential mechanisms for fructose-induced insulin resistance.

makes it difficult to sort out the effects of increased fructose intake versus increased glucose or total sugar intake. Several studies have however addressed the effects of short-term glucose versus fructose overfeeding in humans. One study assessed the early (30 and 60 min) response to an acute 60 g glucose load in young women fed a weight-maintaining diet containing 41% of total energy as glucose or sucrose. Plasma glucose responses were comparable with both high-glucose and high-sucrose diets and were not different from a control diet with low sugar intake. Both diets increased plasma insulin responses to the same extent, but the difference reached statistical significance only after the sucrose diet (114). In normal-weight and obese women overfed with 50% glucose, fructose, sucrose, or fat above their energy requirement, fat balance measured by indirect calorimetry was positive and identical under all three conditions; this indicated that fat storage was directly dependent on energy intake and that fructose or sucrose had no specific effect to promote fat deposition (142). De novo lipogenesis was also measured in the women overfed with glucose or sucrose and was found to be identical under both conditions (141). There was also no significant difference in plasma glucose, triacylglycerol, or insulin concentrations. De novo lipogenesis was shown to be stimulated more with acute fructose than glucose ingestion (165). However, increasing the carbohydrate content of weight-maintaining diets by administration of short glucose polymers (98), but not complex carbohydrate (101), was reported to increase fasting hepatic de novo lipogenesis. The stimulation of fasting de novo lipogenesis was of the same magnitude with high-carbohydrate diets based on glucose polymers (98) or on sugar-starch at a 60:40 ratio (98) administered over 2–4 wk. This indicated that stimulation of hepatic de novo lipogenesis may be more related to the carbohydrate load as simple sugars than to the fructose load.

Finally, the effects of a 10-wk supplementation with either glucose or fructose (in amounts corresponding to 30% of total energy requirements) were observed in a group of overweight and obese women (199). In this group of patients, glucose and fructose overfeeding led to similar body weight gains, suggesting that the lower leptin secretion induced by fructose compared with glucose (207) did not result in a larger food intake in the long term. As expected, fructose led to higher postprandial triglyceride concentrations than glucose. Furthermore, fructose, but not glucose, decreased glucose tolerance and increased the plasma concentration of small dense LDL and of oxidized LDL, which are lipid particles associated with a high atherogenic risk. However, and in contrast to the above-mentioned studies (98, 141, 142), only fructose, but not glucose, stimulated hepatic de novo lipogenesis. Of particular concern, fructose increased significantly visceral fat. From these studies, it therefore appears that overfeeding with simple sugars has several

potentially harmful effects and that the effects of fructose are more focused on alterations of hepatic lipid metabolism and of plasma lipid profile, while both sugars may contribute to lipotoxicity by promoting weight gain and increasing hepatic de novo lipogenesis.

E. Uric Acid Metabolism

In the liver, fructose loading, due to its rapid phosphorylation to fructose 1-P, drastically stimulates ATP hydrolysis, with a subsequent increase in AMP. This in turn leads to increased uric acid synthesis (176). It was indeed repeatedly observed that plasma uric acid concentrations were increased by a high dietary fructose intake. The third NANHES report indeed indicates that consumption of sugar-sweetened beverages is significantly associated with plasma uric acid concentrations (45). Furthermore, fructose consumption has been directly related to the occurrence of diseases related to uric acid metabolism, i.e., gout (44) and kidney stones (206).

Hyperuricemia is frequently encountered in patients with the metabolic syndrome and was a minor criterion for the diagnosis of “syndrome X,” or “insulin resistance syndrome” in its initial description by Reaven (174). Although the mechanisms underlying the link between insulin resistance and hyperuricemia remain poorly defined, serum uric acid concentration appears to be a risk factor for the development of type 2 diabetes (61).

Recently, a novel hypothesis was proposed to link fructose intake, hyperuricemia, and insulin resistance. Insulin-induced glucose utilization involves not only the stimulation of key metabolic pathways in insulin-sensitive cells, but also an increase in blood flow and nutritive circulation to the major insulin-sensitive tissue, skeletal muscle (18). This effect of insulin is due to the activation of the endothelial enzyme nitric oxide synthase (eNOS) by insulin (200). In obese subjects, the ability of insulin to produce muscle vasodilation is impaired, and this is thought to contribute to altered glucose homeostasis through “prereceptor” insulin resistance (201). Since eNOS is potently inhibited by uric acid, it was proposed that inhibition of the vascular effects of insulin by uric acid was involved in fructose-induced insulin resistance. In support of this hypothesis it was reported that, in rats fed a high-fructose diet, both hyperuricemia and insulin resistance develop simultaneously. Furthermore, the development of insulin resistance was prevented by lowering uric acid concentrations with an uricosuric agent (149).

Intriguingly, it was recently reported that putative new fructose transporters, SLC2A9 (GLUT9), bear relationships with uricemia. These transporters, expressed in renal tubules, may possibly modulate renal uric acid excretion. Polymorphisms of *SLC2A9* have been shown to be associated with an increased fractional excretion of

uric acid, suggesting that these polymorphisms may effectively modulate uric acid excretion. Furthermore, genetic variations of SLC2A9 appear to be responsible for ~1–2% of the variance of plasma uric acid concentration in males and 5–6% in females (32, 124). Whether the initial expectation that SCL2A9 were fructose carriers, and their role in uric acid metabolism is merely coincidental, or whether these molecules are involved in some yet unidentified link between fructose and uric acid metabolism, remain presently unknown.

F. High Blood Pressure

In rats, high-fructose feeding has been also shown to be associated with the development of hypertension (102, 104). Several putative mechanisms can be proposed for this effect of fructose. As mentioned in the former sections, chronic, high-fructose feeding is associated with the development of insulin resistance. Insulin resistance, and the ensuing hyperinsulinemia, are in turn associated with high blood pressure (173). An increased sympathetic nervous system activity, possibly triggered by hyperinsulinemia, has been invoked as a potential mechanism (103, 175). Hyperinsulinemia may also increase blood pressure by enhancing kidney sodium reabsorption (179). Finally, high-fructose intake leads to a build up of intracellular glyceraldehyde and dihydroxyacetone phosphate, which can be further converted into methylglyoxal, a highly reactive ketoaldehyde. Aldehydes are able to react non-enzymatically with sulfhydryl groups of protein, thus altering their function. Of interest, aldehydes can impair the function of L-type calcium channels, and this may possibly lead to an increased intracellular calcium concentration in vascular smooth muscle, and to an increase of vascular resistance (231). Furthermore, it has been suspected by some investigators that hypertension may rather be related to deficiency in magnesium or copper of experimental high-fructose diets rather than to fructose feeding per se (37, 79).

Although there are numerous reports of fructose-induced hypertension in rodents, the link between fructose intake and high blood pressure in humans is mainly indirect. In healthy normal-weight subjects (122) and in overweight subjects (199), supplementation with fructose in doses amounting to 30% of total energy requirements failed to significantly alter blood pressure. High fructose intake may be linked with high calorie intake and weight gain, and with insulin resistance, and all these factors are themselves associated with high blood pressure. There is, however, little evidence that fructose per se directly increases blood pressure. There is ample evidence that glucose intake acutely stimulates sympathetic activity. This has been shown to be related to the increase in insulin concentration elicited by glucose rather than to

hyperglycemia per se (22, 23, 234). Furthermore, it was demonstrated that, contrary to glucose, acute fructose administration does not elicit an increase in sympathetic activity (234). When the effect of acute oral loads of glucose and fructose were compared, it was observed that fructose, but not glucose, led to a significant, although small increase in blood pressure (34). Both glucose and fructose increased heart rate and cardiac output, but glucose in addition decreased peripheral vascular resistance, which prevented an increase in blood pressure (34). It was also shown that an intravenous infusion of glucose, but not fructose, causes muscle vasodilation (234), through an insulin-mediated nitric oxide release in endothelial cells (200).

The absence of a stimulation of the sympathetic nervous system after acute fructose loading in humans (228, 234) contrasts with numerous reports of increased sympathetic activity in rodents fed a high-fructose diet (192, 246). This is likely due to the fact that chronic high fructose intake in rodents is generally associated with increased adiposity and that body fat mass is a major determinant of sympathetic activity (187).

G. Mineral Metabolism

Fructose readily forms complexes with metal ions and hence may modulate the intestinal absorption and bioavailability of minerals (152). Compared with starch, both sucrose and fructose decrease copper absorption in rats (112). A diet containing up to 20% energy as fructose had, however, no adverse effect on copper balance in humans (177). Fructose also increases iron absorption in rats (177). There was a specific concern that sugar intake may negatively impact calcium balance and bone health (222). When the effects of different types of carbohydrates were assessed in rats, it was observed that glucose and sucrose, but not fructose alone, tended to have adverse effects on bone health. Rats provided with the glucose-sweetened beverages had reduced femur and tibia total phosphate, reduced phosphate and calcium intake, and increased urinary calcium excretion compared with the rats provided the fructose-sweetened beverage. These results suggest that fructose is not directly involved in the negative association that was observed between sugar intake and bone health (223).

IV. DOES FREE FRUCTOSE EXERT DIFFERENT EFFECTS THAN FRUCTOSE BOUND TO SUCROSE?

An increase in fructose consumption has been proposed as a major contributor to the increased prevalence of obesity that was observed over the past decades worldwide. This hypothesis rests on the fact that the increase in

fructose consumption over time roughly parallels the increase in the prevalence of obesity. Much confusion arises from the fact that free fructose, i.e., under the form of HFCS or of pure fructose added as a sweetener, is often considered separate from total fructose, i.e., the sum of free fructose and fructose bound to glucose. As mentioned earlier, total sugar, including sucrose and HFCS, increased by ~15% over the past 30 years in the United States; at the same time, HFCS consumption increased dramatically and replaced a substantial amount of dietary sucrose. It results that consumption of free fructose increased markedly, while at the same time consumption of fructose bound to glucose decreased. This has sometimes led to the speculation that free fructose may have more deleterious effects of its own.

Few studies have specifically addressed the effects of free versus bound fructose. In animals, feeding a diet rich in HFCS elicited all the effects observed after high-fructose or high-sucrose diets, i.e., increased weight, dyslipidemia, and insulin resistance. As for fructose, HFCS feeding elicited an endoplasmic reticulum stress response in hepatocytes. The effects of HFCS appeared therefore qualitatively comparable to those of sucrose, but no direct comparison was made (48, 209). In patients with type 2 diabetes, administration of 35 g of sucrose or equivalent amounts of fructose and glucose as HFCS elicited similar glucose and insulin responses (6). HFCS also produced the same glucose, insulin, ghrelin, and leptin than sucrose in healthy female volunteers (143). In another study, HFCS, sucrose, and equimolar glucose-fructose mixtures elicited similar satiety responses (7) or energy intake at a subsequent meal (197). HFCS also produced an increase in 24-h plasma triglyceride similar to that observed with pure fructose (198). Although the studies comparing HFCS with sucrose remain to be completed with other end points such as lipogenesis, intrahepatic lipid accumulation, stimulation of inflammation, and with longer duration of administration, there is to date no evidence that the effects of free fructose differ from those of fructose bound to glucose.

V. DOES FRUCTOSE PLAY A ROLE IN THE PATHOGENESIS OF METABOLIC DISEASES?

In view of the compelling evidence that high fructose intake can induce, not only in animal models, but also in humans, a whole range of metabolic and cardiovascular alterations, it is legitimate to wonder whether fructose consumption plays a significant role in the pathogenesis of metabolic diseases in our populations.

Verification of this hypothesis however requires 1) that the fructose intake in the population be quantitatively evaluated, 2) that epidemiological data support a link between dietary fructose intake and disease (by showing

an increased odds of developing the disease at high fructose intake), and/or 3) that intervention studies are consistent with a pathogenic role of fructose, either by showing that increasing fructose intake increases the disease or markers of the disease, or by showing that reducing fructose intake improves the disease or risk factors for the disease.

Although data on fructose consumption are available and reliable in some countries, accurate information is lacking in most parts of the world. Furthermore, many epidemiological studies did not assess directly the effects of total fructose consumption, but of "sugars" or sweet beverages. As a consequence, the information required is only partially available but is nonetheless useful to evaluate the link between fructose and diseases.

A. Fructose and Energy Intake

To evaluate the relationship between fructose consumption on one hand, and obesity and metabolic disorders on the other hand, the effect of fructose on total energy intake is an important issue. On the basis of small studies, it can be expected that fructose does not elicit satiating signals to the same extent as glucose, and hence that it may lead to uncontrolled, excessive energy intake (see sect. II G). Several studies that assessed the relationship between soft drink consumption and energy expenditure were included in a recent meta-analysis. The conclusion was that soft drink intake was clearly associated with increased energy intake. Soft drink intake also was associated with lower intakes of milk and calcium (230).

B. Fructose and Body Weight

Several cross-sectional studies have assessed the relationship between consumption of sugar-sweetened beverages and body weight and were reviewed recently (71). Many of these studies were performed on children and adolescents. Most of these studies (13, 21, 84, 128, 132, 188, 220, 240) showed a positive association between sugar-containing drink consumption and body weight, but others failed to show such association (29, 81, 120, 182). These studies have to be interpreted with caution, however, because soft drink consumption is influenced by several factors, such as socioeconomic status, education, etc. Furthermore, soft drink intake can be associated with a different pattern of physical activity, or a different pattern of feeding. Several cross-sectional studies even showed an inverse relation between total sucrose consumption (from all sources) and body weight (31, 134), which certainly cannot be held as an indicator that sugar consumption promotes weight loss, but is rather explained by other uncontrolled variables; among a pediatric population, it was shown that high-sugar consumers

ate less fat and meat than low-sugar consumers (77). In addition, consumption of sugar-sweetened beverages may be associated with alteration of the consumption of other beverages, such as tea, coffee, or milk, with possible health consequence. For instance, replacing milk with soft drinks may have deleterious effects on calcium metabolism and bone health (8, 119, 230).

Meta-analyses linking body weight and soft drink consumption also yield conflicting results. One such meta-analysis of 88 published studies reported a significant positive association between soft drink consumption and body weight (230), while another meta-analysis of 12 studies showed no such association (80).

Intervention studies provide a clearer view of the relationship between sugar-containing beverages and body weight. In a few experimental studies, sugar-containing diets were added to the usual, ad libitum, diet. In one study, addition of beverages sweetened with HFCS or aspartame, a non-calorie-containing sweetener, resulted in a significant weight gain with HFCS-sweetened beverages only (215). In another study, overweight subjects receiving sugar-containing beverages increased significantly their energy intake and gained weight, while subjects who received non-caloric-sweetened drinks as a control did not change weight (169). Conversely, several studies, mostly performed on children and adolescents, reduced the daily intake of sugar-sweetened beverages; they all showed a significant reduction in energy intake and/or body weight (11, 68, 72, 184, 244).

C. Fructose Intake and Diabetes

Few studies have specifically evaluated the relationship between sugar intake and the risk of developing diabetes. The Women's Health Study is a prospective study in which 39,345 women aged >45 yr were enrolled and followed prospectively, while receiving either low-dose aspirin and vitamin E or placebo. Although the primary aim of the study was to evaluate the incidence of cancer and cardiovascular diseases, each participant provided detailed dietary information which allowed the evaluation of the impact of sugar intake on the subsequent risk to develop type 2 diabetes. The relative risk of diabetes was not different when the lowest and highest quintiles of sugar intake were compared. Furthermore, this absence of increased relative risk was also observed when the analysis was restricted to fructose intake (108). The Nurse's Health Study includes 121,700 registered nurses aged 30–35 yr at inclusion, who provided detailed information by questionnaires regarding diet, life-style, and medical history. Of these, 71,346 were nondiabetic at inclusion and had provided all information required to evaluate the relationship between fruit and fruit juice consumption and subsequent incidence of diabetes. The

results indicate that fruit (and vegetable) intake was associated with a lower incidence of diabetes, while consumption of fruit juice tended to be associated with a higher incidence (19). The Finnish Mobile Clinic Health Examination Survey included 51,522 nondiabetic men and women, aged 40–60 yr, from several regions of Finland and collected dietary and life-style information by interviews and questionnaires. Combined intake of glucose and fructose was associated with an increased risk of diabetes, as was consumption of sweetened fruit juices and soft drinks (146). In another study including 59,000 Afro-American women, the incidence of diabetes was significantly associated with sweetened beverage consumption, but this association was almost entirely mediated by effects of drink consumption on body weight (160). In the Nurses' Health Study II, 51,603 women free of diabetes were included, and a complete dietary assessment was obtained. The risk of gaining weight and of developing type 2 diabetes over an 8-yr follow-up period was significantly increased in women who consumed one or more sugar-sweetened beverages per day (188).

Another study examined, in 2,500 subjects of the fifth Framingham Offspring study (1991–1995), the relationship between sweetened beverage intake and surrogate markers of insulin resistance. Consumption of sweetened drinks was positively associated with fasting insulin concentrations, but not with fasting glucose concentration or with an insulin sensitivity index calculated from fasting glucose and insulin concentrations (245).

D. Fructose Intake and Cardiovascular Risk Factors

In the Framingham Heart Study, the relationship between soft drink consumption and cardiovascular risk factors was evaluated in 6,039 participants. Consumption of more than one can of soft drink per day was significantly associated with the prevalence of the metabolic syndrome, defined by three or more of the following: high blood pressure, waist circumference >35 inches (females) or 40 inches (males), high fasting plasma glucose, high plasma triglyceride, and low HDL-cholesterol. Furthermore, upon prospective follow-up of individuals without the metabolic syndrome at inclusion, consumption of more than one soft drink per day was associated with an increased risk of developing the metabolic syndrome (65).

In a study of 74 6- to 14-yr-old Swiss children, it was observed that overweight children had a similar total fructose intake as normal-weight children, but consumed a significantly higher percentage of fructose from sweets and sweetened drinks. In this population, fructose intake was associated with an increased concentration of small, dense LDL particles known to be associated with a high atherosclerotic risk (4).

Finally, the relationship between sweetened drink intake and the occurrence of coronary heart disease was assessed in 88,520 women enrolled in the Nurse Health Study. Sweetened beverage consumption was significantly associated with an increased incidence of heart disease. A major portion of the relationship was, however, mediated by effects on body weight. The relationship between sweetened beverage intake and incidence of coronary disease remained significant after adjusting for body weight and could be ascribed either to the higher glycemic index or to the high fructose content of sweetened beverages (83).

Over the past decades, several "novel markers" of cardiovascular risk have been identified. These include, amongst others, inflammatory mediators or cytokines, factors related to coagulation and fibrinolysis [such as plasminogen, tissue plasminogen-activator inhibitor-1 (tPAI-1), thrombomodulin], markers of oxidative stress, and markers of endothelial dysfunction (140, 178). In one study including 12 patients with nonalcoholic fatty liver disease and 6 healthy controls, tPAI-1 was positively correlated with total carbohydrate intake, with sucrose intake, and with fructose intake (212). Another study assessed, in 207 men and women aged 18–39 yr, the prevalence of increased novel risk markers (adhesion molecules such as vascular cell adhesion molecule-1, intercellular adhesion molecule-1, cytokines such as tumor necrosis factor- α or interleukin-6, markers of oxidative stress, adipokines, and many others). Several of these markers were positively associated with sucrose intake (213).

E. Fructose Intake and Nonalcoholic Steatohepatitis

Few studies evaluated the relationship between fructose or sucrose consumption and hepatic fat deposition. One study evaluated whether fructose, at levels of intake usually encountered in the population, may play a role in the deposition of intrahepatic lipids. It was observed that fructose intake was nearly twice as high (~90 g/day) in patients with NAFLD than in patients without hepatic steatosis (~45 g) (155). In another study, the consumption of sweetened beverages was found to be increased in patients with nonalcoholic fatty liver disease compared with healthy controls (12). In this group of subjects, consumption of sweetened beverage was the best predictor of intrahepatic fat estimated with ultrasonography.

F. General Conclusions Regarding Epidemiological Studies

Altogether, epidemiological studies at this stage provide an incomplete, sometimes discordant appraisal of

the relationship between fructose or sugar intake and metabolic/cardiovascular diseases. Part of the discordances may be explained by the fact that intakes of sugar, fructose, fruit juices, or sweetened beverages were often not recorded individually, which precludes an accurate calculation of total fructose intake. In addition, fructose is essentially consumed as either sucrose or HFCS, with the consequence that glucose intakes essentially varies with fructose intake. Confounding factors (i.e., interrelationship between sugar intake and intake of other nutrients, association with physical activity and life-style) are important and difficult to control for. At present, there appears to be strong evidence that consumption of sweetened beverages is associated with obesity, at least in children and adolescents. There is at present not the single hint the HFCS may have more deleterious effect on body weight than other sources of sugar. Regarding the relationship between fructose or sucrose intake and cardiovascular risk factors or type 2 diabetes, the evidence is even sparser. Given the number of confounding variables, there is clearly a need for intervention studies in which the fructose intake of high fructose consumers is reduced to better delineate the possible pathogenic role of fructose. At present, short-term intervention studies however suggest that a high-fructose intake consisting of soft drinks, sweetened juices, or bakery products can increase the risk of metabolic and cardiovascular diseases. There is, however, no objective ground to support that moderate intake of fructose, or of fructose consumed with fruits or honey, is unsafe.

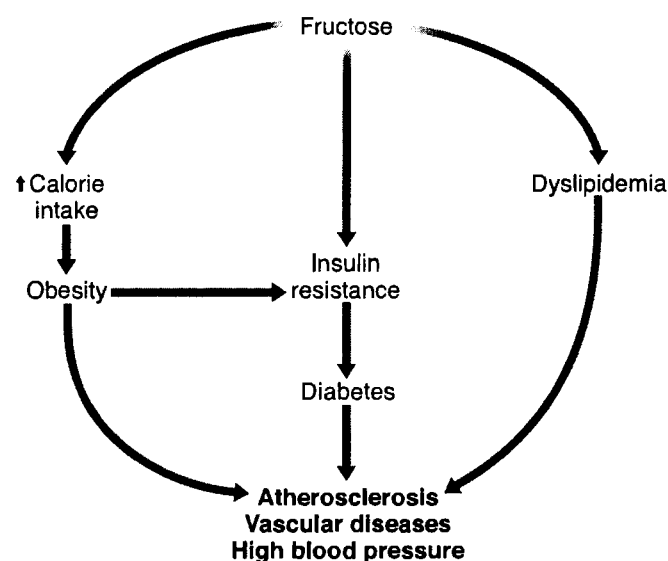


FIG. 7. Potential relationships of high fructose intake with human diseases.

VI. PERSPECTIVES

The potential danger of fructose consumption and its links to various metabolic disorders have been widely documented. Deleterious effects of high fructose intake on body weight, insulin sensitivity/glucose homeostasis, dyslipidemia, and atherosclerotic disease have been identified, and potential mechanisms have been proposed (Fig. 7). These effects, in humans, were often documented at very high levels of fructose intake, however, and some important questions remain to be addressed. Among the numerous deleterious effects of fructose, which ones are directly relevant for human daily nutrition? Most human studies addressing specifically the effects of fructose have administered large doses, often as a supplementation to an isocaloric diet. Nevertheless, there is solid evidence that fructose, even at moderate doses, can cause hypertriglyceridemia. Moreover, although data are scarcer, the fact that fructose may increase intrahepatic lipids and lead to insulin resistance in experimental settings raises some concern. Studies aimed at delineating the dose threshold at which fructose starts to chronically exert such effects remain to be performed. In addition to that, in everyday life, fructose cannot be blamed as the only culprit for all metabolic disorders. Indeed, a high fructose consumption most of the time clusters with additional "risky" behaviors, such as a hypercaloric diet, a diet rich in saturated fat, or low physical activity. Thus which part of metabolic disorders can be attributed to fructose and which results from interactions with other risk factors? Long-term intervention and longitudinal studies may help bring some clues to these issues.

ACKNOWLEDGMENTS

Address for reprint requests and other correspondence: L. Tappy, Dept. of Physiology, Univ. of Lausanne, 7 rue du Bugnon, CH-1005 Lausanne, Switzerland (e-mail: luc.tappy@unil.ch).

GRANTS

The work performed on this topic in the authors' laboratory has been supported by Swiss National Foundation for Science Grants 067787, 109737, and 121995.

REFERENCES

1. Abdel-Sayed A, Binnert C, Le KA, Bortolotti M, Schneiter P, Tappy L. A high-fructose diet impairs basal and stress-mediated lipid metabolism in healthy male subjects. *Br J Nutr* 1–7, 2008.
2. Adelman RC, Ballard FJ, Weinhouse S. Purification and properties of rat liver fructokinase. *J Biol Chem* 242: 3360–3365, 1967.
3. Adopo E, Peronnet F, Massicotte D, Brisson GR, Hillaire-Marcel C. Respective oxidation of exogenous glucose and fructose given in the same drink during exercise. *J Appl Physiol* 76: 1014–1019, 1994.
4. Aeberli I, Zimmermann MB, Molinari L, Lehmann R, l'Allemand D, Spinaz GA, Berneis K. Fructose intake is a predictor of LDL particle size in overweight schoolchildren. *Am J Clin Nutr* 86: 1174–1178, 2007.
5. Ahlborg G, Bjorkman O. Splanchnic and muscle fructose metabolism during and after exercise. *J Appl Physiol* 69: 1244–1251, 1990.
6. Akgun S, Ertel NH. The effects of sucrose, fructose, and high-fructose corn syrup meals on plasma glucose and insulin in non-insulin-dependent diabetic subjects. *Diabetes Care* 8: 279–283, 1985.
7. Akhavan T, Anderson GH. Effects of glucose-to-fructose ratios in solutions on subjective satiety, food intake, and satiety hormones in young men. *Am J Clin Nutr* 86: 1354–1363, 2007.
8. Albala C, Ebbeling CB, Cifuentes M, Lera L, Bustos N, Ludwig DS. Effects of replacing the habitual consumption of sugar-sweetened beverages with milk in Chilean children. *Am J Clin Nutr* 88: 605–611, 2008.
9. Allen DG, Lamb GD, Westerblad H. Skeletal muscle fatigue: cellular mechanisms. *Physiol Rev* 88: 287–332, 2008.
10. Anderson JW, Story LJ, Zettwoch NC, Gustafson NJ, Jefferson BS. Metabolic effects of fructose supplementation in diabetic individuals. *Diabetes Care* 12: 337–344, 1989.
11. Ashley JM, St Jeor ST, Perumean-Chaney S, Schrage J, Bovee V. Meal replacements in weight intervention. *Obes Res* 9 Suppl 4: 312S–320S, 2001.
12. Assy N, Nasser G, Kamayse I, Nseir W, Beniashvili Z, Djibre A, Grosovski M. Soft drink consumption linked with fatty liver in the absence of traditional risk factors. *Can J Gastroenterol* 22: 811–816, 2008.
13. Bandini LG, Vu D, Must A, Cyr H, Goldberg A, Dietz WH. Comparison of high-calorie, low-nutrient-dense food consumption among obese and non-obese adolescents. *Obes Res* 7: 438–443, 1999.
14. Bantle JP, Laine DC, Thomas JW. Metabolic effects of dietary fructose and sucrose in types I and II diabetic subjects. *JAMA* 256: 3241–3246, 1986.
15. Bantle JP, Raatz SK, Thomas W, Georgopoulos A. Effects of dietary fructose on plasma lipids in healthy subjects. *Am J Clin Nutr* 72: 1128–1134, 2000.
16. Bantle JP, Swanson JE, Thomas W, Laine DC. Metabolic effects of dietary fructose in diabetic subjects. *Diabetes Care* 15: 1468–1476, 1992.
17. Bar-On H, Stein Y. Effect of glucose and fructose administration on lipid metabolism in the rat. *J Nutr* 94: 95–105, 1968.
18. Baron AD, Steinberg HO, Chaker H, Leaming R, Johnson A, Brechtel G. Insulin-mediated skeletal muscle vasodilation contributes to both insulin sensitivity and responsiveness in lean humans. *J Clin Invest* 96: 786–792, 1995.
19. Bazzano LA, Li TY, Joshipura KJ, Hu FB. Intake of fruit, vegetables, and fruit juices and risk of diabetes in women. *Diabetes Care* 31: 1311–1317, 2008.
20. Bergheim I, Weber S, Vos M, Kramer S, Volynets V, Kaserouni S, McClain CJ, Bischoff SC. Antibiotics protect against fructose-induced hepatic lipid accumulation in mice: role of endotoxin. *J Hepatol* 48: 983–992, 2008.
21. Berkey CS, Rockett HR, Field AE, Gillman MW, Colditz GA. Sugar-added beverages and adolescent weight change. *Obes Res* 12: 778–788, 2004.
22. Berne C, Fagius J, Niklasson F. Sympathetic response to oral carbohydrate administration. Evidence from microelectrode nerve recordings. *J Clin Invest* 84: 1403–1409, 1989.
23. Berne C, Fagius J, Pollare T, Hjemdahl P. The sympathetic response to euglycaemic hyperinsulinaemia. Evidence from microelectrode nerve recordings in healthy subjects. *Diabetologia* 35: 873–879, 1992.
24. Bizeau ME, Pagliassotti MJ. Hepatic adaptations to sucrose and fructose. *Metab Clin Exp* 54: 1189–1201, 2005.
25. Bjorkman O, Crump M, Phillips RW. Intestinal metabolism of orally administered glucose and fructose in Yucatan miniature swine. *J Nutr* 114: 1413–1420, 1984.
26. Bjorkman O, Felig P. Role of the kidney in the metabolism of fructose in 60-hour fasted humans. *Diabetes* 31: 516–520, 1982.
27. Bjorkman O, Gunnarsson R, Hagström E, Felig P, Wahren J. Splanchnic and renal exchange of infused fructose in insulin-deficient type 1 diabetic patients and healthy controls. *J Clin Invest* 83: 52–59, 1989.

28. **Blaak EE, Saris WH.** Postprandial thermogenesis and substrate utilization after ingestion of different dietary carbohydrates. *Metabolism* 45: 1235–1242, 1996.
29. **Blum JW, Jacobsen DJ, Donnelly JE.** Beverage consumption patterns in elementary school aged children across a two-year period. *J Am Coll Nutr* 24: 93–98, 2005.
30. **Bode C, Durr HK, Bode JC.** Effect of fructose feeding on the activity of enzymes of glycolysis, gluconeogenesis, and the pentose phosphate shunt in the liver and jejunal mucosa of rats. *Horm Metab Res* 13: 379–383, 1981.
31. **Bolton-Smith C, Woodward M.** Dietary composition and fat to sugar ratios in relation to obesity. *Int J Obes Relat Metab Disord* 18: 820–828, 1994.
32. **Brandstatter A, Kiechl S, Kollerits B, Hunt SC, Heid IM, Coassin S, Willeit J, Adams TD, Illig T, Hopkins PN, Kronenberg F.** Sex-specific association of the putative fructose transporter SLC2A9 variants with uric acid levels is modified by BMI. *Diabetes Care* 31: 1662–1667, 2008.
33. **Bray GA, Nielsen SJ, Popkin BM.** Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. *Am J Clin Nutr* 79: 537–543, 2004.
34. **Brown CM, Dulloo AG, Yepuri G, Montani JP.** Fructose ingestion acutely elevates blood pressure in healthy young humans. *Am J Physiol Regul Integr Comp Physiol* 294: R730–R737, 2008.
35. **Bruynseels K, Bergans N, Gillis N, van Dorpen F, Van Hecke P, Stalmans W, Vanstapel F.** On the inhibition of hepatic glycogenolysis by fructose. A ³¹P-NMR study in perfused rat liver using the fructose analogue 2,5-anhydro-D-mannitol. *NMR Biomed* 12: 145–156, 1999.
36. **Burns SP, Murphy HC, Iles RA, Bailey RA, Cohen RD.** Hepatic intralobular mapping of fructose metabolism in the rat liver. *Biochem J* 349: 539–545, 2000.
37. **Busserolles J, Gueux E, Rock E, Mazur A, Rayssiguier Y.** High fructose feeding of magnesium deficient rats is associated with increased plasma triglyceride concentration and increased oxidative stress. *Magnesium Res* 16: 7–12, 2003.
38. **Busserolles J, Gueux E, Rock E, Mazur A, Rayssiguier Y.** Substituting honey for refined carbohydrates protects rats from hypertriglyceridemic and prooxidative effects of fructose. *J Nutr* 132: 3379–3382, 2002.
39. **Cani PD, Amar J, Iglesias MA, Poggi M, Knauf C, Bastelica D, Neyrinck AM, Fava F, Tuohy KM, Chabo C, Waget A, Delmee E, Cousin B, Sulpice T, Chamontin B, Ferrieres J, Tanti JF, Gibson GR, Casteilla L, Delzenne NM, Alessi MC, Burcelin R.** Metabolic endotoxemia initiates obesity and insulin resistance. *Diabetes* 56: 1761–1772, 2007.
40. **Cani PD, Bibiloni R, Knauf C, Waget A, Neyrinck AM, Delzenne NM, Burcelin R.** Changes in gut microbiota control metabolic endotoxemia-induced inflammation in high-fat diet-induced obesity and diabetes in mice. *Diabetes* 57: 1470–1481, 2008.
41. **Carmona A, Freedland RA.** Comparison among the lipogenic potential of various substrates in rat hepatocytes: the differential effects of fructose-containing diets on hepatic lipogenesis. *J Nutr* 119: 1304–1310, 1989.
42. **Cha SH, Wolfgang M, Tokutake Y, Chohnan S, Lane MD.** Differential effects of central fructose and glucose on hypothalamic malonyl-CoA and food intake. *Proc Natl Acad Sci USA* 105: 16871–16875, 2008.
43. **Cheeseman CI.** GLUT2 is the transporter for fructose across the rat intestinal basolateral membrane. *Gastroenterology* 105: 1050–1056, 1993.
44. **Choi HK, Curhan G.** Soft drinks, fructose consumption, and the risk of gout in men: prospective cohort study. *BMJ* 336: 309–312, 2008.
45. **Choi JW, Ford ES, Gao X, Choi HK.** Sugar-sweetened soft drinks, diet soft drinks, and serum uric acid level: the Third National Health and Nutrition Examination Survey. *Arthritis Rheum* 59: 109–116, 2008.
46. **Chong MF, Fielding BA, Frayn KN.** Mechanisms for the acute effect of fructose on postprandial lipemia. *Am J Clin Nutr* 85: 1511–1520, 2007.
47. **Clark DG, Rognstad R, Katz J.** Lipogenesis in rat hepatocytes. *J Biol Chem* 249: 2028–2036, 1974.
48. **Collison KS, Saleh SM, Bakheet RH, Al-Rabiah RK, Inglis AL, Makhoul NJ, Maqbool ZM, Zaidi MZ, Al-Johi MA, Al-Mohanna FA.** Diabetes of the liver: the link between nonalcoholic fatty liver disease and HFCS-55. *Obesity*. In press.
49. **Colville CA, Seatter MJ, Jess TJ, Gould GW, Thomas HM.** Kinetic analysis of the liver-type (GLUT2) and brain-type (GLUT3) glucose transporters in *Xenopus* oocytes: substrate specificities and effects of transport inhibitors. *Biochem J* 290: 701–706, 1993.
50. **Corpe CP, Bovelander FJ, Munoz CM, Hoekstra JH, Simpson IA, Kwon O, Levine M, Burant CF.** Cloning and functional characterization of the mouse fructose transporter, GLUT5. *Biochim Biophys Acta* 1576: 191–197, 2002.
51. **Corpe CP, Burant CF, Hoekstra JH.** Intestinal fructose absorption: clinical and molecular aspects. *J Pediatr Gastroenterol Nutr* 28: 364–374, 1999.
52. **Cortez-Pinto H, Chatham J, Chacko VP, Arnold C, Rashid A, Diehl AM.** Alterations in liver ATP homeostasis in human nonalcoholic steatohepatitis: a pilot study. *JAMA* 282: 1659–1664, 1999.
53. **Courtney Moore M, Connolly CC, Cherrington AD.** Autoregulation of hepatic glucose production. *Eur J Endocr* 138: 240–248, 1998.
54. **Crapo PA, Insel JI, Sperling M, Kolterman OG.** Comparison of serum glucose, insulin and glycogen responses to different types of complex carbohydrates in non insulin dependent diabetic patients. *Am J Clin Nutr* 34: 184–190, 1981.
55. **Crapo PA, Kolterman OG.** The metabolic effects of 2-week fructose feeding in normal subjects. *Am J Clin Nutr* 39: 525–534, 1984.
56. **Crapo PA, Kolterman OG, Olefsky JM.** Effects of oral fructose in normal, diabetic, and impaired glucose tolerance subjects. *Diabetes Care* 3: 575–582, 1980.
57. **Cui XL, Schlesier AM, Fisher EL, Cerqueira C, Ferraris RP.** Fructose-induced increases in neonatal rat intestinal fructose transport involve the PI3-kinase/Akt signaling pathway. *Am J Physiol Gastrointest Liver Physiol* 288: G1310–G1320, 2005.
58. **Currell K, Jeukendrup AE.** Superior endurance performance with ingestion of multiple transportable carbohydrates. *Med Sci Sports Exerc* 40: 275–281, 2008.
59. **Da Silva LA, De Marcucci OL, Carmona A.** Adaptive changes in total pyruvate dehydrogenase activity in lipogenic tissues of rats fed high-sucrose or high-fat diets. *Comp Biochem Physiol B Comp Physiol* 103: 407–411, 1992.
60. **Dallongeville J, Lussier-Cacan S, Davignon J.** Modulation of plasma triglyceride levels by apoE phenotype: a meta-analysis. *J Lipid Res* 33: 447–454, 1992.
61. **Dehghan A, van Hoek M, Sijbrands EJ, Hofman A, Witteman JC.** High serum uric acid as a novel risk factor for type 2 diabetes. *Diabetes Care* 31: 361–362, 2008.
62. **Delarue J, Normand S, Pachiaudi C, Beylot M, Lamisse F, Riou JP.** The contribution of naturally labelled ¹³C fructose to glucose appearance in humans. *Diabetologia* 36: 338–345, 1993.
63. **Delluc G, Delluc B, Roques editors M.** *La nutrition préhistorique. Périgueux*. ISBN2-9501983-8-4: Pilote 24, 1995, p. 223.
64. **Denechaud PD, Dentin R, Girard J, Postic C.** Role of ChREBP in hepatic steatosis and insulin resistance. *FEBS Lett* 582: 68–73, 2008.
65. **Dhingra R, Sullivan L, Jacques PF, Wang TJ, Fox CS, Meigs JB, D'Agostino RB, Gaziano JM, Vasan RS.** Soft drink consumption and risk of developing cardiometabolic risk factors and the metabolic syndrome in middle-aged adults in the community. *Circulation* 116: 480–488, 2007.
66. **Dietze G, Wicklmayr M, Grunst J, Stiegler S, Mehert H.** Utilization of glucose and fructose in human liver and muscle. *Int Z Vitam Ernahrungsforsch Beih* 15: 31–43, 1976.
67. **Dirlewanger M, Schneiter P, Jéquier E, Tappy L.** Effects of fructose on hepatic glucose metabolism in humans. *Am J Physiol Endocrinol Metab* 279: E907–E911, 2000.
68. **Ditschuneit HH, Flechtner-Mors M, Johnson TD, Adler G.** Metabolic and weight-loss effects of a long-term dietary intervention in obese patients. *Am J Clin Nutr* 69: 198–204, 1999.
69. **Donmoyer CM, Ejiogor J, Lacy DB, Chen SS, McGuinness OP.** Fructose augments infection-impaired net hepatic glucose uptake during TPN administration. *Am J Physiol Endocrinol Metab* 280: E703–E711, 2001.

70. **Douard V, Ferraris RP.** Regulation of the fructose transporter GLUT5 in health and disease. *Am J Physiol Endocrinol Metab* 295: E227–E237, 2008.
71. **Drewnowski ABF.** Liquid calories, sugar, and body weight. *Am J Clin Nutr* 85: 651–661, 2007.
72. **Ebbeling CB, Feldman HA, Osganian SK, Chomitz VR, Ellenbogen SJ, Ludwig DS.** Effects of decreasing sugar-sweetened beverage consumption on body weight in adolescents: a randomized, controlled pilot study. *Pediatrics* 117: 673–680, 2006.
73. **Eiffert KC, McDonald RB, Stern JS.** High sucrose diet and exercise: effects on insulin-receptor function of 12- and 24-mo-old Sprague-Dawley rats. *J Nutr* 121: 1081–1089, 1991.
74. **Erkkila AT, Sarkkinen ES, Lindi V, Lehto S, Laakso M, Uusitupa MI.** APOE polymorphism and the hypertriglyceridemic effect of dietary sucrose. *Am J Clin Nutr* 73: 746–752, 2001.
75. **Erkkila AT, Schwab US, Agren JJ, Hallikainen M, Gylling H, Uusitupa MI.** Moderate increase in dietary sucrose does not influence fasting or postprandial serum lipids regardless of the presence of apolipoprotein E2 allele in healthy subjects. *Eur J Clin Nutr* 61: 1094–1101, 2007.
76. **Faeh D, Minehira K, Schwarz J, Periasami R, Seongus P, Tappy L.** Effect of fructose overfeeding and fish oil administration on hepatic de novo lipogenesis and insulin sensitivity in healthy males. *Diabetes* 54: 1907–1913, 2005.
77. **Farris RP, Nicklas TA, Myers L, Berenson GS.** Nutrient intake and food group consumption of 10-year-olds by sugar intake level: the Bogalusa Heart Study. *J Am Coll Nutr* 17: 579–585, 1998.
78. **Ferraris RP, Hsiao J, Hernandez R, Hirayama B.** Site density of mouse intestinal glucose transporters declines with age. *Am J Physiol Gastrointest Liver Physiol* 264: G285–G293, 1993.
79. **Fields M, Lewis CG.** Dietary fructose but not starch is responsible for hyperlipidemia associated with copper deficiency in rats: effect of high-fat diet. *J Am Coll Nutr* 18: 83–87, 1999.
80. **Forshee RA, Anderson PA, Storey ML.** Sugar-sweetened beverages and body mass index in children and adolescents: a meta-analysis. *Am J Clin Nutr* 87: 1662–1671, 2008.
81. **Forshee RA, Storey ML.** Total beverage consumption and beverage choices among children and adolescents. *Int J Food Sci Nutr* 54: 297–307, 2003.
82. **Foster-Powell K, Miller JB.** International tables of glycemic index. *Am J Clin Nutr* 62: 871S–890S, 1995.
83. **Fung TT, Malik V, Rexrode KM, Manson JE, Willett WC, Hu FB.** Sweetened beverage consumption and risk of coronary heart disease in women. *Am J Clin Nutr* 89: 1037–1042, 2009.
84. **Giammattei J, Blix G, Marshak HH, Wollitzer AO, Pettitt DJ.** Television watching and soft drink consumption: associations with obesity in 11- to 13-year-old school children. *Arch Pediatr Adolesc Med* 157: 882–886, 2003.
85. **Grigoresco C, Rizkalla SW, Halfon P, Bornet F, Fontvieille AM, Bros M, Dauchy F, Tchobroutsky G, Slama G.** Lack of detectable deleterious effects on metabolic control of daily fructose ingestion for 2 mo in NIDDM patients. *Diabetes Care* 11: 546–550, 1988.
86. **Grimble RF.** Inflammatory status and insulin resistance. *Curr Opin Clin Nutr Metab Care* 5: 551–559, 2002.
87. **Guzman M, Castro J.** Zonation of fatty acid metabolism in rat liver. *Biochem J* 264: 107–113, 1989.
88. **Haidari M, Leung N, Mahbub F, Uffelman KD, Kohen-Avramoglu R, Lewis GF, Adeli K.** Fasting and postprandial overproduction of intestinally derived lipoproteins in an animal model of insulin resistance. Evidence that chronic fructose feeding in the hamster is accompanied by enhanced intestinal de novo lipogenesis and ApoB48-containing lipoprotein overproduction. *J Biol Chem* 277: 31646–31655, 2002.
89. **Hallfrisch J, Ellwood KC, Michaelis OEt Reiser S, O'Doriso TM, Prather ES.** Effects of dietary fructose on plasma glucose and hormone responses in normal and hyperinsulinemic men. *J Nutr* 113: 1819–1826, 1983.
90. **Hanover LM, White JS.** Manufacturing, composition, and applications of fructose. *Am J Clin Nutr* 58: 724S–732S, 1993.
91. **Havel PJ.** Dietary fructose: implications for dysregulation of energy homeostasis and lipid/carbohydrate metabolism. *Nutr Rev* 63: 133–157, 2005.
92. **Heinz F, Lamprecht W, Kirsch J.** Enzymes of fructose metabolism in human liver. *J Clin Invest* 47: 1826–1832, 1968.
93. **Hers HG, Kusaka T.** The metabolism of fructose-1-phosphate in the liver. *Biochim Biophys Acta* 11: 427–437, 1953.
94. **Holloszy JO.** Stimulation of glucose transport by muscle contractions: role of calcium. In: *Diabetes Mellitus and Exercise*, edited by Devlin JT, Horton ES, Vranic M. Nishimura: Smith-Gordon, 1992, p. 91–98.
95. **Hommes FA.** Inborn errors of fructose metabolism. *Am J Clin Nutr* 58: 788S–795S, 1993.
96. **Horton TJ, Gayles EC, Prach PA, Koppenhafer TA, Pagliasotti MJ.** Female rats do not develop sucrose-induced insulin resistance. *Am J Physiol Regul Integr Comp Physiol* 272: R1571–R1576, 1997.
97. **Hu Z, Cha SH, Chohnan S, Lane MD.** Hypothalamic malonyl-CoA as a mediator of feeding behavior. *Proc Natl Acad Sci USA* 100: 12624–12629, 2003.
98. **Hudgins LC, Hellerstein M, Seidman C, Neese R, Diakun J, Hirsch J.** Human fatty acid synthesis is stimulated by a eucaloric low fat, high carbohydrate diet. *J Clin Invest* 97: 2081–2091, 1996.
99. **Hudgins LC, Hellerstein MK, Seidman CE, Neese RA, Tremaroli JD, Hirsch J.** Relationship between carbohydrate-induced hypertriglyceridemia and fatty acid synthesis in lean and obese subjects. *J Lipid Res* 41: 595–604, 2000.
100. **Hudgins LC, Seidman CE, Diakun J, Hirsch J.** Human fatty acid synthesis is reduced after the substitution of dietary starch for sugar. *Am J Clin Nutr* 67: 631–639, 1998.
101. **Hwang IS, Ho H, Hoffman BB, Reaven GM.** Fructose-induced insulin resistance and hypertension in rats. *Hypertension* 10: 512–516, 1987.
102. **Hwang IS, Ho H, Hoffman BB, Reaven GM.** Fructose-induced insulin resistance and hypertension in rats. *Hypertension* 10: 512–516, 1987.
103. **Hwang IS, Huang WC, Wu JN, Shian LR, Reaven GM.** Effect of fructose-induced hypertension on the renin-angiotensin-aldosterone system and atrial natriuretic factor. *Am J Hypertens* 2: 424–427, 1989.
- 104a. **International Sugar Organization.** *Sugar Year Book 2008*. London: ISO, 2008.
105. **Iynedjian PB.** Mammalian glucokinase and its gene. *BMJ* 293: 1–13, 1993.
106. **Jahoor F, Peters EJ, Wolfe RR.** The relationship between gluconeogenic substrate supply and glucose production in humans. *Am J Physiol Endocrinol Metab* 258: E288–E296, 1990.
107. **Jandrain BJ, Pallikarakis N, Normand S, Pirnay F, Lacroix M, Mosora F, Pachiandi C, Gautier JF, Scheen AJ, Riou JP.** Fructose utilization during exercise in men: rapid conversion of ingested fructose to circulating glucose. *J Appl Physiol* 74: 2146–2154, 1993.
108. **Janket SJ, Manson JE, Sesso H, Buring JE, Liu S.** A prospective study of sugar intake and risk of type 2 diabetes in women. *Diabetes Care* 26: 1008–1015, 2003.
109. **Jenssen T, Nurjhan N, Consoli A, Gerich JE.** Failure of substrate-induced gluconeogenesis to increase overall glucose appearance in normal humans. *J Clin Invest* 86: 489–497, 1990.
110. **Jentjens RL, Moseley L, Waring RH, Harding LK, Jeukendrup AE.** Oxidation of combined ingestion of glucose and fructose during exercise. *J Appl Physiol* 96: 1277–1284, 2004.
111. **Jeukendrup AE.** Carbohydrate intake during exercise and performance. *Nutrition* 20: 669–677, 2004.
112. **Johnson MA.** Interaction of dietary carbohydrate, ascorbic acid and copper with the development of copper deficiency in rats. *J Nutr* 116: 802–815, 1986.
113. **Kazumi T, Vranic M, Steiner G.** Triglyceride kinetics: effects of dietary glucose, sucrose, or fructose alone or with hyperinsulinemia. *Am J Physiol Endocrinol Metab* 250: E325–E330, 1986.
114. **Kelsay JL, Behall KM, Holden JM, Prather ES.** Diets high in glucose or sucrose and young women. *Am J Clin Nutr* 27: 926–936, 1974.
115. **Kim JY, Nolte LA, Hansen PA, Han DH, Kawanaka K, Holloszy JO.** Insulin resistance of muscle glucose transport in male and female rats fed a high-sucrose diet. *Am J Physiol Regul Integr Comp Physiol* 276: R665–R672, 1999.

116. **Kneepkens CM, Vonk RJ, Fernandes J.** Incomplete intestinal absorption of fructose. *Arch Dis Child* 59: 735–738, 1984.
117. **Koivisto VA, Harkonen M, Karonen SL, Groop PH, Elovainio R, Ferrannini E, Sacca L, DeFronzo RA.** Glycogen depletion during prolonged exercise: influence of glucose, fructose, or placebo. *J Appl Physiol* 58: 731–737, 1985.
118. **Koo HY, Wallig MA, Chung BH, Nara TY, Cho BH, Nakamura MT.** Dietary fructose induces a wide range of genes with distinct shift in carbohydrate and lipid metabolism in fed and fasted rat liver. *Biochim Biophys Acta* 1782: 341–348, 2008.
119. **Kristensen M, Jensen M, Kudsk J, Henriksen M, Molgaard C.** Short-term effects on bone turnover of replacing milk with cola beverages: a 10-day interventional study in young men. *Osteoporos Int* 16: 1803–1808, 2005.
120. **Kvaavik E, Andersen LF, Klepp KI.** The stability of soft drinks intake from adolescence to adult age and the association between long-term consumption of soft drinks and lifestyle factors and body weight. *Public Health Nutr* 8: 149–157, 2005.
121. **Lê KA, Ith M, Kreis R, Faeh D, Bortolotti M, Tran C, Boesch C, Tappy L.** Fructose over consumption causes dyslipidemia and ectopic lipid deposition in healthy subjects with and without a family history of type 2 diabetes. *Am J Clin Nutr* 89: 1760–1765, 2009.
122. **Lê KA, Faeh D, Stettler R, Ith M, Kreis R, Vermathen P, Boesch C, Ravussin E, Tappy L.** A 4-wk high-fructose diet alters lipid metabolism without affecting insulin sensitivity or ectopic lipids in healthy humans. *Am J Clin Nutr* 84: 1374–1379, 2006.
123. **Lê KA, Tappy L.** Metabolic effects of fructose. *Curr Opin Clin Nutr Metab Care* 10: 210–214, 2007.
124. **Le MT, Shafiu M, Mu W, Johnson RJ.** SLC2A9—a fructose transporter identified as a novel uric acid transporter. *Nephrol Dial Transplant* 23: 2746–2749, 2008.
125. **Levine L, Evans WJ, Cadarette BS, Fisher EC, Bullen BA.** Fructose and glucose ingestion and muscle glycogen use during submaximal exercise. *J Appl Physiol* 55: 1767–1771, 1983.
126. **Lewis GF, Uffelman K, Naples M, Szeto L, Haidari M, Adeli K.** Intestinal lipoprotein overproduction, a newly recognized component of insulin resistance, is ameliorated by the insulin sensitizer rosiglitazone: studies in the fructose-fed Syrian golden hamster. *Endocrinology* 146: 247–255, 2005.
127. **Licht FO (Editor).** *International Sugar Economic Yearbook and Directory*. Turnbridge Wells, UK: Agna Informa, 2005.
128. **Liebman M, Pelican S, Moore SA, Holmes B, Wardlaw MK, Melcher LM, Liddil AC, Paul LC, Dunnagan T, Haynes GW.** Dietary intake, eating behavior, and physical activity-related determinants of high body mass index in rural communities in Wyoming, Montana, Idaho. *Int J Obes Relat Metab Disord* 27: 684–692, 2003.
129. **Litherland GJ, Hajdich E, Gould GW, Hundal HS.** Fructose transport and metabolism in adipose tissue of Zucker rats: diminished GLUT5 activity during obesity and insulin resistance. *Mol Cell Biochem* 261: 23–33, 2004.
130. **Liu J, Grundy SM, Wang W, Smith SC Jr, Vega GL, Wu Z, Zeng Z, Wang W, Zhao D.** Ethnic-specific criteria for the metabolic syndrome: evidence from China. *Diabetes Care* 29: 1414–1416, 2006.
131. **Livesey G, Taylor R.** Fructose consumption and consequences for glycation, plasma triacylglycerol, and body weight: meta-analyses and meta-regression models of intervention studies. *Am J Clin Nutr* 88: 1419–1437, 2008.
132. **Ludwig DS, Peterson KE, Gortmaker SL.** Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. *Lancet* 357: 505–508, 2001.
133. **Macdonald I.** Influence of fructose and glucose on serum lipid levels in men and pre- and postmenopausal women. *Am J Clin Nutr* 18: 369–372, 1966.
134. **Maillard G, Charles MA, Lafay L, Thibult N, Vray M, Borys JM, Basdevant A, Eschwege E, Romon M.** Macronutrient energy intake and adiposity in nonobese prepubertal children aged 5–11 yr (the Fleurbaix Laventie Ville Sante Study). *Int J Obes Relat Metab Disord* 24: 1608–1617, 2000.
135. **Marriott BP, Cole N, Lee E.** National estimates of dietary fructose intake increased from 1977 to 2004 in the United States. *J Nutr* 139: 1228–1235, 2009.
136. **Marshall RO, Kooi ER.** Enzymatic conversion of D-glucose to D-fructose. *Science* 125: 648–649, 1957.
137. **Matsuzaka T, Shimano H, Yahagi N, Amemiya-Kudo M, Okazaki H, Tamura Y, Iizuka Y, Ohashi K, Tomita S, Sekiya M, Hasty A, Nakagawa Y, Sone H, Toyoshima H, Ishibashi S, Osuga J, Yamada N.** Insulin-independent induction of sterol regulatory element-binding protein-1c expression in the livers of streptozotocin-treated mice. *Diabetes* 53: 560–569, 2004.
138. **Mayes PA.** Intermediary metabolism of fructose. *Am J Clin Nutr* 58, Suppl: 754S–765S, 1993.
139. **McAteer EJ, O'Reilly G, Hadden DR.** The effects of one month high fructose intake on plasma glucose and lipid levels in non-insulin-dependent diabetes. *Diabet Med* 4: 62–64, 1987.
140. **McDermott MM, Liu K, Ferrucci L, Tian L, Guralnik JM, Green D, Tan J, Liao Y, Pearce WH, Schneider JR, McCue K, Ridker P, Rifai N, Criqui MH.** Circulating blood markers and functional impairment in peripheral arterial disease. *J Am Geriatr Soc* 56: 1504–1510, 2008.
141. **McDevitt RM, Bott SJ, Harding M, Coward WA, Bluck LJ, Prentice AM.** De novo lipogenesis during controlled overfeeding with sucrose or glucose in lean and obese women. *Am J Clin Nutr* 74: 737–746, 2001.
142. **McDevitt RM, Poppitt SD, Murgatroyd PR, Prentice AM.** Macronutrient disposal during controlled overfeeding with glucose, fructose, sucrose, or fat in lean and obese women. *Am J Clin Nutr* 72: 369–377, 2000.
143. **Melanson KJ, Zukley L, Lowndes J, Nguyen V, Angelopoulos TJ, Rippe JM.** Effects of high-fructose corn syrup and sucrose consumption on circulating glucose, insulin, leptin, and ghrelin and on appetite in normal-weight women. *Nutrition* 23: 103–112, 2007.
144. **Mithieux G, Misery P, Magnan C, Pillot B, Gautier-Stein A, Bernard C, Rajas F, Zitoun C.** Portal sensing of intestinal gluconeogenesis is a mechanistic link in the diminution of food intake induced by diet protein. *Cell Metab* 2: 321–329, 2005.
145. **Mitsui T, Shimaoka K, Kanao Y, Kondo T.** Colonic fermentation after ingestion of fructose-containing sports drink. *J Sports Med Phys Fitness* 41: 121–123, 2001.
146. **Montonen J, Jarvinen R, Knekt P, Heliövaara M, Reunanen A.** Consumption of sweetened beverages and intakes of fructose and glucose predict type 2 diabetes occurrence. *J Nutr* 137: 1447–1454, 2007.
147. **Nagai Y, Nishio Y, Nakamura T, Maegawa H, Kikkawa R, Kashiwagi A.** Amelioration of high fructose-induced metabolic derangements by activation of PPAR α . *Am J Physiol Endocrinol Metab* 282: E1180–E1190, 2002.
148. **Nagai Y, Yonemitsu S, Erion DM, Iwasaki T, Stark R, Weismann D, Dong J, Zhang D, Jurczak MJ, Loffler MG, Cresswell J, Yu XX, Murray SF, Bhanot S, Monia BP, Bogan JS, Samuel V, Shulman GI.** The role of peroxisome proliferator-activated receptor gamma coactivator-1 beta in the pathogenesis of fructose-induced insulin resistance. *Cell Metab* 9: 252–264, 2009.
149. **Nakagawa T, Hu H, Zharikov S, Tuttle KR, Short RA, Glushakova O, Ouyang X, Feig DI, Block ER, Herrera-Acosta J, Patel JM, Johnson RJ.** A causal role for uric acid in fructose-induced metabolic syndrome. *Am J Physiol Renal Physiol* 290: F625–F631, 2006.
150. **Newsholme EA, Leech AR.** *Biochemistry for the Medical Sciences*. Chichester, UK: Wiley, 1983.
151. **Nilsson LH, Hultman E.** Liver and muscle glycogen in man after glucose and fructose infusion. *Scand J Clin Lab Invest* 33: 5–10, 1974.
152. **O'Dell BL.** Fructose and mineral metabolism. *Am J Clin Nutr* 58: 771S–778S, 1993.
154. **Osei K, Falko J, Bossetti BM, Holland GC.** Metabolic effects of fructose as a natural sweetener in the physiologic meals of ambulatory obese patients with type II diabetes. *Am J Med* 83: 249–255, 1987.
155. **Ouyang X, Cirillo P, Sautin Y, McCall S, Bruchette JL, Diehl AM, Johnson RJ, Abdelmalek MF.** Fructose consumption as a risk factor for non-alcoholic fatty liver disease. *J Hepatol* 48: 993–999, 2008.
156. **Pagliassotti MJ, Horton TJ.** Sucrose, insulin action and biologic complexity. *Recent Res Dev Physiol* 2: 337–353, 2004.

157. **Pagliassotti MJ, Kang J, Thresher JS, Sung CK, Bizeau ME.** Elevated basal PI 3-kinase activity and reduced insulin signaling in sucrose-induced hepatic insulin resistance. *Am J Physiol Endocrinol Metab* 282: E170–E176, 2002.
158. **Pagliassotti MJ, Prach PA.** Quantity of sucrose alters the tissue pattern and time course of insulin resistance in young rats. *Am J Physiol Regul Integr Comp Physiol* 269: R641–R646, 1995.
159. **Pagliassotti MJ, Prach PA, Koppenhafer TA, Pan DA.** Changes in insulin action, triglycerides, and lipid composition during sucrose feeding in rats. *Am J Physiol Regul Integr Comp Physiol* 271: R1319–R1326, 1996.
160. **Palmer JR, Boggs DA, Krishnan S, Hu FB, Singer M, Rosenberg L.** Sugar-sweetened beverages and incidence of type 2 diabetes mellitus in African American women. *Arch Intern Med* 168: 1487–1492, 2008.
161. **Paquot N, Schneiter P, Jéquier E, Gaillard R, Lefèbvre PJ, Scheen A, Tappy L.** Effects of ingested fructose and infused glucagon on endogenous glucose production in obese NIDDM patients, obese non-diabetic subjects, and healthy subjects. *Diabetologia* 39: 580–586, 1996.
162. **Park OJ, Cesar D, Faix D, Wu K, Shackleton CH, Hellerstein MK.** Mechanisms of fructose-induced hypertriglyceridaemia in the rat. Activation of hepatic pyruvate dehydrogenase through inhibition of pyruvate dehydrogenase kinase. *Biochem J* 282: 753–757, 1992.
163. **Park YK, Yetley EA.** Intakes and food sources of fructose in the United States. *Am J Clin Nutr* 58, Suppl: 737S–747S, 1993.
164. **Parks EJ, Hellerstein MK.** Carbohydrate-induced hypertriglycerolemia: historical perspective and review of biological mechanisms. *Am J Clin Nutr* 71: 412–433, 2000.
165. **Parks EJ, Skokan LE, Timlin MT, Dingfelder CS.** Dietary sugars stimulate fatty acid synthesis in adults. *J Nutr* 138: 1039–1046, 2008.
166. **Perin N, Keelan M, Jarocka-Cyrta E, Clandinin MT, Thomson AB.** Ontogeny of intestinal adaptation in rats in response to isocaloric changes in dietary lipids. *Am J Physiol Gastrointest Liver Physiol* 273: G713–G720, 1997.
167. **Petersen KF, Cline GW, Gerard DP, Magnusson I, Rothman DL, Shulman GI.** Contribution of net hepatic glycogen synthesis to disposal of an oral glucose load in humans. *Metabolism* 50: 598–601, 2001.
168. **Podolin DA, Sutherland E, Iwahashi M, Simon FR, Pagliassotti MJ.** A high-sucrose diet alters the lipid composition and fluidity of liver sinusoidal membranes. *Horm Metab Res* 30: 195–199, 1998.
169. **Raben A, Vasilaras TH, Moller AC, Astrup A.** Sucrose compared with artificial sweeteners: different effects on ad libitum food intake and body weight after 10 wk of supplementation in overweight subjects. *Am J Clin Nutr* 76: 721–729, 2002.
170. **Rajas F, Bruni N, Montano S, Zitoun C, Mithieux G.** The glucose-6 phosphatase gene is expressed in human and rat small intestine: regulation of expression in fasted and diabetic rats. *Gastroenterology* 117: 132–139, 1999.
171. **Randle P, Garland P, Hales C, Newsholme E.** The glucose fatty-acid cycle. Its role in insulin sensitivity and the metabolic disturbances of diabetes mellitus. *Lancet* 1: 785–789, 1963.
172. **Ravich WJ, Bayless TM, Thomas M.** Fructose: incomplete intestinal absorption in humans. *Gastroenterology* 84: 26–29, 1983.
173. **Reaven GM.** Insulin resistance, hyperinsulinemia, and hypertriglyceridemia in the etiology and clinical course of hypertension. *Am J Med* 90: 7S–12S, 1991.
174. **Reaven GM.** Pathophysiology of insulin resistance in human disease. *Physiol Rev* 75: 473–486, 1995.
175. **Reaven GM, Lithell H, Landsberg L.** Hypertension and associated metabolic abnormalities: the role of insulin resistance and the sympathoadrenal system. *New Engl J Med* 334: 374–381, 1996.
176. **Reiser S.** Effect of dietary sugars on metabolic risk factors associated with heart disease. *Nutr Health* 3: 203–216, 1985.
177. **Reiser S, Smith JC Jr, Mertz W, Holbrook JT, Scholfeld DJ, Powell AS, Canfield WK, Canary JJ.** Indices of copper status in humans consuming a typical American diet containing either fructose or starch. *Am J Clin Nutr* 42: 242–251, 1985.
178. **Ridker PM.** Fibrinolytic and inflammatory markers for arterial occlusion: the evolving epidemiology of thrombosis and hemostasis. *Thromb Haemost* 78: 53–59, 1997.
179. **Rocchini AP, Katch V, Kveselis D, Moorehead C, Martin M, Lampman R, Gregory M.** Insulin and renal sodium retention in obese adolescents. *Hypertension* 14: 367–374, 1989.
180. **Rodin J.** Comparative effects of fructose, aspartame, glucose, and water preloads on calorie and macronutrient intake. *Am J Clin Nutr* 51: 428–435, 1990.
181. **Rodin J.** Effects of pure sugar vs. mixed starch fructose loads on food intake. *Appetite* 17: 213–219, 1991.
182. **Rodriguez-Artalejo F, Garcia EL, Gorgojo L, Garces C, Royo MA, Martin Moreno JM, Benavente M, Macias A, De Oya M.** Consumption of bakery products, sweetened soft drinks and yogurt among children aged 6–7 years: association with nutrient intake and overall diet quality. *Br J Nutr* 89: 419–429, 2003.
183. **Rogians N, Vila L, Farre M, Alegret M, Sanchez RM, Vazquez-Carrera M, Laguna JC.** Impairment of hepatic Stat-3 activation and reduction of PPARalpha activity in fructose-fed rats. *Hepatology* 45: 778–788, 2007.
184. **Rothacker DQ, Staniszewski BA, Ellis PK.** Liquid meal replacement vs traditional food: a potential model for women who cannot maintain eating habit change. *J Am Diet Assoc* 101: 345–347, 2001.
185. **Rowe JW, Young JB, Minaker KL, Stevens AL, Pallota J, Landsberg L.** Effects of insulin and glucose infusions on sympathetic nervous system activity in normal man. *Diabetes* 30: 219–225, 1981.
186. **Rowlands DS, Thorburn MS, Thorp RM, Broadbent S, Shi X.** Effect of graded fructose coingestion with maltodextrin on exogenous ¹⁴C-fructose and ¹³C-glucose oxidation efficiency and high-intensity cycling performance. *J Appl Physiol* 104: 1709–1719, 2008.
187. **Scherrer U, Randin D, Tappy L, Vollenweider P, Jéquier E, Nicod P.** Body fat and sympathetic nerve activity in healthy subjects. *Circulation* 89: 2634–2640, 1994.
188. **Schulze MB, Manson JE, Ludwig DS, Colditz GA, Stampfer MJ, Willett WC, Hu FB.** Sugar-sweetened beverages, weight gain, and incidence of type 2 diabetes in young and middle-aged women. *JAMA* 292: 927–934, 2004.
189. **Schwarz JM, Acheson KJ, Tappy L, Piolino V, Müller MJ, Felber JP, Jéquier E.** Thermogenesis and fructose metabolism in humans. *Am J Physiol Endocrinol Metab* 262: E591–E598, 1992.
190. **Schwarz JM, Neese R, Shackleton C, Hellerstein MK.** De novo lipogenesis during fasting and oral fructose in lean and obese hyperinsulinemic subjects (Abstract). *Diabetes* 42, Suppl 1: 39A, 1993.
191. **Schwarz JM, Schutz Y, Froidevaux F, Acheson K, Jeanprêtre N, Schneider H, Felber JP, Jéquier E.** Thermogenesis in men and women induced by fructose vs glucose added to a meal. *Am J Clin Nutr* 49: 667–674, 1989.
192. **Sechi LA.** Mechanisms of insulin resistance in rat models of hypertension and their relationships with salt sensitivity. *J Hypertens* 17: 1229–1237, 1999.
193. **Shapiro A, Mu W, Roncal C, Cheng KY, Johnson RJ, Scarpace PJ.** Fructose-induced leptin resistance exacerbates weight gain in response to subsequent high-fat feeding. *Am J Physiol Regul Integr Comp Physiol* 295: R1370–R1375, 2008.
194. **Shimomura I, Bashmakov Y, Horton JD.** Increased levels of nuclear SREBP-1c associated with fatty livers in two mouse models of diabetes mellitus. *J Biol Chem* 274: 30028–30032, 1999.
195. **Shulman GI.** Cellular mechanisms of insulin resistance. *J Clin Invest* 106: 171–176, 2000.
196. **Simonson DC, Tappy L, Jéquier E, Felber JP, DeFronzo RA.** Normalization of carbohydrate-induced thermogenesis by fructose in insulin-resistant states. *Am J Physiol Endocrinol Metab* 254: E201–E207, 1988.
197. **Soenen S, Westerterp-Plantenga MS.** No differences in satiety or energy intake after high-fructose corn syrup, sucrose, or milk preloads. *Am J Clin Nutr* 86: 1586–1594, 2007.
198. **Stanhope KL, Griffen SC, Bair BR, Swarbrick MM, Keim NL, Havel PJ.** Twenty-four-hour endocrine and metabolic profiles following consumption of high-fructose corn syrup, sucrose, fructose-, and glucose-sweetened beverages with meals. *Am J Clin Nutr* 87: 1194–1203, 2008.
199. **Stanhope KL, Schwarz JM, Keim NL, Griffen SC, Bremer AA, Graham JL, Hatcher B, Cox CL, Dyachenko A, Zhang W, McGahan JP, Seibert A, Krauss RM, Chiu S, Schaefer EJ, Ai M, Otokozawa S, Nakajima K, Nakano T, Beysen C, Heller-**

- stein MK, Berglund L, Havel PJ. Consuming fructose-sweetened, not glucose-sweetened, beverages increases visceral adiposity and lipids and decreases insulin sensitivity in overweight/obese humans. *J Clin Invest* 119: 1322–1334, 2009.
200. Steinberg HO, Brechtel G, Johnson A, Fineberg N, Baron AD. Insulin-mediated skeletal muscle vasodilation is nitric oxide dependent. A novel action of insulin to increase nitric oxide release. *J Clin Invest* 94: 1172–1179, 1994.
201. Steinberg HO, Chaker H, Learning R, Johnson A, Brechtel G, Baron AD. Obesity/insulin resistance is associated with endothelial dysfunction. Implications for the syndrome of insulin resistance. *J Clin Invest* 97: 2601–2610, 1996.
202. Swarbrick MM, Stanhope KL, Elliott SS, Graham JL, Krauss RM, Christiansen MP, Griffen SC, Keim NL, Havel PJ. Consumption of fructose-sweetened beverages for 10 weeks increases postprandial triacylglycerol and apolipoprotein-B concentrations in overweight and obese women. *Br J Nutr* 100: 947–952, 2008.
203. Tappy L, Dussoix P, Iynedjian P, Henry S, Schneiter P, Zahnd G, Jéquier E, Philippe J. Abnormal regulation of hepatic glucose output in maturity onset diabetes of the young caused by a specific mutation of the glucokinase gene. *Diabetes* 46: 204–208, 1997.
204. Tappy L, Jéquier E. Fructose and dietary thermogenesis. *Am J Clin Nutr* 58, Suppl: 766S–770S, 1993.
205. Tappy L, Randin JP, Felber JP, Chioloro R, Simonson DC, Jéquier E, DeFronzo RA. Comparison of thermogenic effect of fructose and glucose in normal humans. *Am J Physiol Endocrinol Metab* 250: E718–E724, 1986.
206. Taylor EN, Curhan GC. Fructose consumption and the risk of kidney stones. *Kidney Int* 73: 207–212, 2008.
207. Teff KL, Elliott SS, Tschöp M, Kieffer TJ, Rader D, Heiman M, Townsend RR, Keim NL, D'Alessio DA, Havel PJ. Dietary fructose reduces circulating insulin and leptin, attenuates postprandial suppression of ghrelin, and increases triglycerides in women. *J Clin Endocrinol Metab* 89: 2963–2972, 2004.
208. Teff KL, Grudziak J, Townsend RR, Dunn TN, Grant RW, Adams SH, Keim NL, Cummings BP, Stanhope KL, Havel PJ. Endocrine and metabolic effects of consuming fructose- and glucose-sweetened beverages with meals in obese men and women: influence of insulin resistance on plasma triglyceride responses. *J Clin Endocrinol Metab* 94: 1562–1569, 2009.
209. Tetri LH, Basaranoglu M, Brunt EM, Yerian LM, Neuschwander-Tetri BA. Severe NAFLD with hepatic neuroinflammatory changes in mice fed trans fats and a high-fructose corn syrup equivalent. *Am J Physiol Gastrointest Liver Physiol* 295: G987–G995, 2008.
210. Thorburn AW, Crapo PA, Beltz WF, Wallace P, Witztum JL, Henry RR. Lipid metabolism in non-insulin-dependent diabetes: effects of long-term treatment with fructose-supplemented mixed meals. *Am J Clin Nutr* 50: 1015–1022, 1989.
211. Thorburn AW, Crapo PA, Griver K, Wallace P, Henry RR. Long-term effects of dietary fructose on carbohydrate metabolism in non-insulin-dependent diabetes mellitus. *Metabolism* 39: 58–63, 1990.
212. Thuy S, Ladurner R, Volynets V, Wagner S, Strahl S, Konigsrainer A, Maier KP, Bischoff SC, Bergheim I. Nonalcoholic fatty liver disease in humans is associated with increased plasma endotoxin and plasminogen activator inhibitor 1 concentrations and with fructose intake. *J Nutr* 138: 1452–1455, 2008.
213. Tonstad S, Thorsrud H, Torjesen PA, Seljeflot I. Do novel risk factors differ between men and women aged 18 to 39 years with a high risk of coronary heart disease? *Metabolism* 56: 260–266, 2007.
214. Topping DL, Mayes PA. The immediate effects of insulin and fructose on the metabolism of the perfused liver. Changes in lipoprotein secretion, fatty acid oxidation and esterification, and lipogenesis and carbohydrate metabolism. *Biochem J* 126: 295–311, 1972.
215. Tordoff MG, Alleva AM. Effect of drinking soda sweetened with aspartame or high-fructose corn syrup on food intake and body weight. *Am J Clin Nutr* 51: 963–969, 1990.
216. Tordoff MG, Friedman MI. Hepatic control of feeding: effect of glucose, fructose, and mannitol infusion. *Am J Physiol Regul Integr Comp Physiol* 254: R969–R976, 1988.
217. Tornheim K, Lowenstein JM. Control of phosphofructokinase from rat skeletal muscle. Effects of fructose diphosphate, AMP, ATP, and citrate. *J Biol Chem* 251: 7322–7328, 1976.
218. Tounian P, Schneiter P, Henry S, Delarue J, Tappy L. Effects of dexamethasone on hepatic glucose production and fructose metabolism in healthy humans. *Am J Physiol Endocrinol Metab* 273: E315–E320, 1997.
219. Tounian P, Schneiter P, Henry S, Jéquier E, Tappy L. Effects of infused fructose on endogenous glucose production, gluconeogenesis and glycogen metabolism in healthy humans. *Am J Physiol Endocrinol Metab* 267: E710–E717, 1994.
220. Troiano RP, Briefel RR, Carroll MD, Bialostosky K. Energy and fat intakes of children and adolescents in the United States: data from the national health and nutrition examination surveys. *Am J Clin Nutr* 72: 1343S–1353S, 2000.
221. Truswell AS, Seach JM, Thorburn AW. Incomplete absorption of pure fructose in healthy subjects and the facilitating effect of glucose. *Am J Clin Nutr* 48: 1424–1430, 1988.
222. Tsanzi E, Fitch CW, Tou JC. Effect of consuming different caloric sweeteners on bone health and possible mechanisms. *Nutr Rev* 66: 301–309, 2008.
223. Tsanzi E, Light HR, Tou JC. The effect of feeding different sugar-sweetened beverages to growing female Sprague-Dawley rats on bone mass and strength. *Bone* 42: 960–968, 2008.
224. Unger RH. Minireview: weapons of lean body mass destruction: the role of ectopic lipids in the metabolic syndrome. *Endocrinology* 144: 5159–5165, 2003.
225. United States Department of Agriculture. *Sugar and Sweeteners Yearbook Tables. Other Recommended Data Products.* <http://www.ers.usda.gov/briefing/sugar/data.htm>. (03.02.2009).
226. Uyeda K, Repa JJ. Carbohydrate response element binding protein, ChREBP, a transcription factor coupling hepatic glucose utilization and lipid synthesis. *Cell Metab* 4: 107–110, 2006.
227. Van Den Bergh AJ, Houtman S, Heerschap A, Rehrer NJ, Van Den Boogert HJ, Oeseburg B, Hopman MT. Muscle glycogen recovery after exercise during glucose and fructose intake monitored by ¹³C-NMR. *J Appl Physiol* 81: 1495–1500, 1996.
228. Van Gaal L, Mertens I, Vansant G, De Leeuw I. Carbohydrate-induced thermogenesis in obese women. Effect of insulin and catecholamines. *J Endocrinol Invest* 22: 109–114, 1999.
229. Van Schaftingen E, Detheux M, Veiga da Cunha M. Short-term control of glucokinase activity: role of a regulatory protein. *FASEB J* 8: 414–419, 1994.
230. Vartanian LR, Schwartz MB, Brownell KD. Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis. *Am J Public Health* 97: 667–675, 2007.
231. Vasdev S, Longereich L, Gill V. Prevention of fructose-induced hypertension by dietary vitamins. *Clin Biochem* 37: 1–9, 2004.
232. Velho G, Petersen KF, Perseghin G, Hwang JH, Rothman DL, Pueyo ME, Cline GW, Froguel P, Shulman GI. Impaired hepatic glycogen synthesis in glucokinase-deficient (MODY-2) subjects. *J Clin Invest* 98: 1755–1761, 1996.
233. Vila L, Roglans N, Alegret M, Sanchez RM, Vazquez-Carrera M, Laguna JC. Suppressor of cytokine signaling-3 (SOCS-3) and a deficit of serine/threonine (Ser/Thr) phosphoproteins involved in leptin transduction mediate the effect of fructose on rat liver lipid metabolism. *Hepatology* 48: 1506–1516, 2008.
234. Vollenweider P, Tappy L, Randin D, Schneiter P, Jéquier E, Nicod P, Scherrer U. Differential effects of hyperinsulinemia and carbohydrate metabolism on sympathetic nerve activity and muscle blood flow in humans. *J Clin Invest* 92: 147–154, 1993.
235. Wallis GA, Rowlands DS, Shaw C, Jentjens RL, Jeukendrup AE. Oxidation of combined ingestion of maltodextrins and fructose during exercise. *Med Sci Sports Exercise* 37: 426–432, 2005.
236. Wei Y, Pagliassotti MJ. Hepatospecific effects of fructose on c-jun NH₂-terminal kinase: implications for hepatic insulin resistance. *Am J Physiol Endocrinol Metab* 287: E926–E933, 2004.
237. Wei Y, Wang D, Pagliassotti MJ. Fructose selectively modulates c-jun N-terminal kinase activity and insulin signaling in rat primary hepatocytes. *J Nutr* 135: 1642–1646, 2005.
238. Wei Y, Wang D, Topczewski F, Pagliassotti MJ. Fructose-mediated stress signaling in the liver: implications for hepatic insulin resistance. *J Nutr Biochem* 18: 1–9, 2007.
239. Welle S, Schwartz RG, Statt M. Reduced metabolic rate during β -adrenergic blockade in humans. *Metabolism* 40: 619–622, 1991.

240. **Welsh JA, Cogswell ME, Rogers S, Rockett H, Mei Z, Grummer-Strawn LM.** Overweight among low-income preschool children associated with the consumption of sweet drinks: Missouri, 1999–2002. *Pediatrics* 115: e223–229, 2005.
241. **White JS.** Straight talk about high-fructose corn syrup: what it is and what it ain't. *Am J Clin Nutr* 88: 1716S–1721S, 2008.
242. **Winder WW, Taylor EB, Thomson DM.** Role of AMP-activated protein kinase in the molecular adaptation to endurance exercise. *Med Sci Sports Exerc* 38: 1945–1949, 2006.
243. **Woods HF, Alberti KG.** Dangers of intravenous fructose. *Lancet* 2: 1354–1357, 1972.
244. **Yip I, Go VL, DeShields S, Salzman P, Bellman M, Thames G, Murray S, Wang HJ, Elashoff R, Heber D.** Liquid meal replacements and glycemic control in obese type 2 diabetes patients. *Obes Res* 9, Suppl 4: 341S–347S, 2001.
245. **Yoshida M, McKeown NM, Rogers G, Meigs JB, Saltzman E, D'Agostino R, Jacques PF.** Surrogate markers of insulin resistance are associated with consumption of sugar-sweetened drinks and fruit juice in middle and older-aged adults. *J Nutr* 137: 2121–2127, 2007.
246. **Young JB, Weiss J, Boufath N.** Effects of dietary monosaccharides on sympathetic nervous system activity in adipose tissues of male rats. *Diabetes* 53: 1271–1278, 2004.

Luc Tappy and Kim-Anne Lê

Physiol Rev 90:23-46, 2010. doi:10.1152/physrev.00019.2009

You might find this additional information useful...

This article cites 237 articles, 132 of which you can access free at:

<http://physrev.physiology.org/cgi/content/full/90/1/23#BIBL>

Medline items on this article's topics can be found at <http://highwire.stanford.edu/lists/artbytopic.dtl> on the following topics:

Agriculture .. Corn
Biochemistry .. Fructose
Biochemistry .. Sucrose
Medicine .. Diet
Medicine .. Obesity
Medicine .. Body Weight

Updated information and services including high-resolution figures, can be found at:

<http://physrev.physiology.org/cgi/content/full/90/1/23>

Additional material and information about *Physiological Reviews* can be found at:

<http://www.the-aps.org/publications/prv>

This information is current as of April 12, 2010 .

Sweetened Beverage Marketing

Sugar Water Gets a Facelift: What Marketing Does for Soda

SEPTEMBER 2009

arbonated water. High fructose corn syrup. Sucrose. Sugar. Caramel color. Phosphoric acid. Artificial flavors. Natural flavors. Caffeine. Citric acid. Potassium benzoate. Sodium benzoate. Sodium citrate.

Without marketing, sodas would be known only for the ingredients listed on their bottles and cans. Instead, they are known for their elaborate campaigns and catchy jingles. The three companies that produce the majority of the industry's 450 soft drinks—Coca-Cola, Pepsi, and Cadbury Schweppes*—make sure of that.¹ You can walk on “The Coke Side of Life” or “Drink Pepsi, Get Stuff” (or buy both and get double the amount of branded T-shirts and other “stuff”).

If marketing didn't work, the Coca-Cola Company wouldn't pay \$35 million a year to co-sponsor American Idol, and Pepsi wouldn't have invested \$1.2 billion in 2008 just to revamp its logo. Much of the cost for this change has gone toward replacing the old Pepsi logo with the new one everywhere it appears around the world: trucks, vending machines, stadium signs, and point-of-sale materials.²

The marketing blitz is more than just business as usual; it's part of the soda industry's response to the country's declining consumption of full-calorie soda, which has been sliding for the past decade. As obesity rates rise and type 2 diabetes—once unheard of in children—becomes commonplace, more people than ever are drinking diet sodas or switching to other beverages.

In recent years, the public health establishment dealt a powerful blow to the soda industry when it demanded the removal of soda vending machines in schools. In 2006, under threat of lawsuits and regulation, soda executives from the three top companies conceded. They also promised to insert healthy diet or lifestyle messages into at least half of their advertising to children under 12 years old.

Now the \$72 billion carbonated soft drink industry³ is doing everything it can to keep its current customers and attract new ones. “We've got to recruit new users and hold on to users as they age,” Bill Elmore, president and chief operating officer of Coca-Cola Bottling Company, Consolidated, told the *Wall Street Journal*.⁴

* On May 7, 2008, Cadbury Schweppes spun off its soft drink business which is now known as Dr Pepper Snapple Group. We refer to Cadbury Schweppes in this brief as all relevant figures are from before May 7, 2008.

So far, the industry's amped up marketing efforts seem to be working: In spite of increased demand for diet drinks and an industry-wide bruising from the public health establishment, full-calorie soda—delivering 13 teaspoons of sugar per can—is still the most popular drink in the United States, dominating over 70% of the non-alcoholic beverage market.⁵

What marketing tactics are soda companies using to distinguish their particular combination of carbonated water, sugar, flavor, and other chemicals? Who is their target audience? In this framing brief, we find out.

The 4 P's of Marketing

Soda may be on "The Most Wanted List" of public health advocates and policy makers, but its marketers present the opposite picture. Consider the four "P's" of marketing—product, promotion, place and price. Together, they represent strategies to target specific demographic groups. The object is to maintain and increase consumption of existing customers, attract new ones, compete for customers of other brands, and create a positive public image. Analyzing the four "P's" of soda marketing is one way to understand the industry's tactics and develop effective responses.

PRODUCT: This is the item being marketed and the package in which it is sold. Sometimes, this marketing involves inventing a product to attract consumers who are not already using a product. Take Coke Zero ("Real Coke Taste, Zero Calories"), created because many men do not like ordering "diet" drinks, which they perceive to be for women who are watching their weight. Coke Zero's no-frills black-and-red bottle has been branded with a large "Z" to evoke masculine taste. In 2007, according to *SportsBusiness Journal*, the company spent \$13 million during the NCAA basketball tournament to boost the then-new product.⁶⁷

PLACE: This is the location of sale, service and consumption, and industry practices used in making it work. No companies are better at "place" than Coca-Cola and Pepsi. For those who think ethnic targeting regarding place is new, consider the history of Pepsi: As a result of segregated regiments in WWII, Pepsi-Cola reports that it was the only soft drink available to African-American soldiers. By the end of that war, it was the soft drink of choice among that overseas group. For decades, Pepsi had bragging rights to being first choice of African-Americans. Walter Mack, Pepsi's president during the 1940s, hired a former executive of the National Urban League to develop a program to increase its sales to the Black community. Edward Boyd, credited by many with being the first to use "target marketing," hired a team of 10 African-American

Top soda brands

In 2007, the three top carbonated soft drink companies spent a total of \$608.5 million on domestic advertising⁶⁸—more than \$1 million a day in the United States alone. The top 10 selling carbonated soft drinks haven't changed much in the last decade. In order of sales (with their companies in parentheses), top brands include:⁷

1. Coke Classic
2. Pepsi-Cola
3. Diet Coke
4. Mountain Dew (Pepsi Cola)

can salesmen who traveled the country spreading the Pepsi story of equality. At age 7, Ron Brown, Bill Clinton's Secretary of Commerce, was featured in Pepsi's first ad aimed at the Black community that demanded its stores carry Pepsi.⁶⁹ The "place" Pepsi captured with its marketing was the African American community.

PRICE: One way the soda industry is responding to the slowdown in soda sales is to change the product's size and price. During the summer of 2008, some of Coca-Cola's and Pepsi's biggest bottlers replaced the 20-ounce that sold in convenience stores for \$1.49 with a 24-ounce bottle that cost 20 cents more. Others replaced the 20-ounce with 16-ounce bottles priced at 99 cents, less expensive than the 20-ounce bottles.⁶⁷ The companies' goal is to price the product attractively so every demographic group will find a perfect fit.

PROMOTION: Promotion conveys the industry's message about the attributes of the product and producer. It informs the consumer of the product's benefits and improves the producer's public image. Soda marketers are on the cutting edge of promotion, using digital marketing to send coupons directly to kids' cell phones, in addition to traditional promotions like TV ads, billboards, point-of-sale advertising, and sponsorships. Download a text message from one of the top brands and get a code for a free soda at your nearest fast food restaurant. Also included in soda promotion is sponsorship of sports and other events, philanthropic donations for health research, and product placement such as the Coke glasses raised by the judges on *American Idol*.

Public health advocates can think of the 4 P's for prevention. Using the 4 P's of marketing to promote health, advocates could raise the price of soda with fees or taxes, insist the product is sold in smaller portion sizes, restrict where it is consumed (place), and limit the advertising seen by children and youth (promotion). All of these restrictions in marketing would decrease consumption and the harms that come from it.

5. Diet Pepsi
6. Dr Pepper (Cadbury Schweppes)
7. Sprite (Coca-Cola Company)
8. Fanta (Coca-Cola Company)
9. Diet Mountain Dew (Pepsi Cola)
10. Diet Dr Pepper (Cadbury Schweppes)

What has changed is how the industry spends its marketing dollars.

Where do soda companies spend their marketing dollar?

TV advertising is expensive, and most soda marketing dollars still go there (Table 1). But that is changing. TNS Media Intelligence reports that the three dominant soda companies spent less in 2007 on television than in 2006. According to John Sicher, editor and publisher of *Beverage Digest*, soft drink industry spending on measured media advertising—broadcast, billboards and print—is down “because they are spending on different kinds of marketing—promotions, email, handing out samples, and the like.”⁸

The latest figures come from *Marketing Food to Children and Adolescents*, a Federal Trade Commission study of expenditures and activities by 44 food and beverage companies, including the big three, released in July of 2008.⁹ Ordered by Congress, the analysis covers only 2006, the year before soda companies announced self-regulatory agreements. Among its findings:

- Carbonated beverages was the highest category in terms of marketing expenditure directed at children (ages 2-11) and adolescents (ages 12-17) (\$492 million, compared to \$294 million for restaurant foods, the next highest category);
- Of the \$492 million, 96% was directed at marketing to adolescents;
- Carbonated beverage companies spent \$21 million on advertising using Web sites, Internet, digital ads, word-of-mouth, and viral marketing. Carbonated beverage companies spent more on “new media” than did any other food or beverage category.
- The 44 companies spent \$91 million on in-store marketing and packaging of carbonated beverages, almost all of it directed toward teenagers;
- They spent \$117 million marketing carbonated beverages using traditional promotional activities such as product placement ads appearing before or within a video game; ads preceding a home video or theatrical movie feature, including license fees paid to use a third-party animated character in advertising or for cross-promotional arrangements; sponsorships of sports teams and athletes; fees paid for celebrity endorsements; or product branding in conjunction with philanthropic endeavors.

Each of these marketing categories uses research and special firms to help the soda manufacturers figure out

TABLE 1.
Measured media expenditures for all audiences for soda, 2006 and 2007 (in millions)

Type of Advertising	Coca-Cola		PepsiCo		Cadbury	
	2006	2007	2006	2007	2006	2007
Television	227.0	197.0	225.0	172.0	112.0	68.0
Outdoor Ads	25.0	25.0	4.0	11.0	6.5	6.5
Magazine	26.0	35.0	15.0	3.0	15.0	2.0
Newspaper	3.0	3.4	0.7	0.9	0.3	0.4
Radio	32.0	22.0	31.0	30.8	4.5	7.3
Online Display	9.0	4.0	7.0	15.0	1.7	1.7

Source: TNS Media Intelligence

how to reach consumers, including children and teens. The fastest growing marketing techniques are digital.

Soda's digital future is now

The future of soda advertising is being shaped mostly overseas—and under the radar of most American consumers—by means of digital media. Here and abroad, soda companies are marketing on the Internet, via cell phones or other mobile devices, and through video games, integrating their digital campaigns with traditional media like TV or billboards.

Through digital marketing, soda companies can fine-tune their target markets, especially for young consumers, in the U.S. and around the world.

Call the new target Generation P for “programmers.” Tim Rosta, executive vice president of integrated marketing at MTV Networks, came up with that moniker while partnering with Pepsi Cola on a futuristic project. Their audience, he says, is “people aged 12 to 34 who are programming their own world and creating content around our shows.”¹⁰ Comfortable in the digital world, young people create identities for themselves online, connected to sites or programs designed especially for them. Users create online characters or alter egos called avatars that interact in the often heavily branded “virtual” world.

In 2007, MTV included its prime-time hit series *The Hills* online in its virtual world. Users create an avatar (a visual representation of the user that can appear two- or three-dimensional) to interact with others in that world, where they can chat, play games, and watch episodes of *The Hills*. Pepsi joined as a sponsor, creating what *Ad-Week* referred to as a category-exclusive, branded content program where characters could pump their virtual coins to buy a drink to quench their virtual thirst. Avatars could also acquire Pepsi-themed clothing.

In May of 2008, MTV unveiled a case study claiming that linking its TV shows to Internet sites can sustain the interest viewers aged 12 to 34 have in the advertising as well as the entertainment. Among the study's findings was that “Pepsi's positive brand image traits increased dramatically among fans who not only watch the show, but browse *The Hills* content online, where Pepsi runs 30-second spots and banners. Positive brand image increased even more among fans who played in *The Hills* virtual world as well ... Pepsi's products were a hit with partic-

ipating consumers in MTV's virtual world. Pepsi was the top-selling [virtual] product in 2007, moving more than 110,000 cans that were virtually recycled and used more than 650,000 times. ...”¹¹

In January of 2008, *BetaNews* reported that “virtual reality” (an immersive computer-generated environment that seems real to the user) is making a comeback from the 1990s. But this time, it's as an advertising tool.¹²

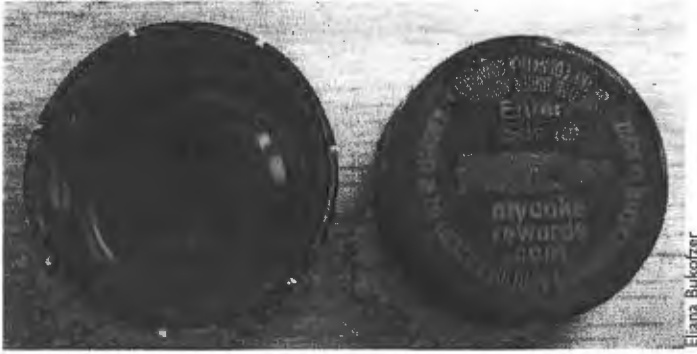
Reporter Jacqueline Emigh wrote: “Some people might be shocked by the use of kids' Web sites for ‘immersive advertising,’ but others might argue that kids have long been the targets of ads and celebrity promotional campaigns anyhow, through vehicles ranging from Beatles cards in bubble gum packs in the 1960s, to cereal ads on TV cartoon shows, since the 1950s.”¹³

What's different now is the intense, immersive, and incessant nature of the marketing. Consider one campaign from Coca-Cola, in which the company joined Nike on reportedly the most popular mobile site in Japan, dubbed *mobagetown*. By clicking on ads and registering with or shopping on affiliate sites, a user could pocket “virtual” money and use it to play Coca-Cola-branded games and “buy” exclusive Coca-Cola items for the avatar. More than 1 million users signed up with Coca-Cola Mobile, as many as 350,000 users became “friends” with the Coke avatar, and 190,000 comments were left on the character's blog. In March of 2008, nearly a year later, users still sported the brand's virtual clothing online.¹⁴

The immersive nature of digital marketing is significant first because users spend far more time engaged with the brand than in earlier marketing like the 30-second commercial on TV. The engagement is highly personal since the users create their own characters which are designed to be online extensions of themselves. And, perhaps most important from the soda companies' perspective, the marketer can collect data on every move—every click—the user makes, feeding the companies' ability to direct ever more targeted marketing back to the users.

It's no surprise, then, that digital marketing expenditures are going up. “For the first time ever,” reports Christopher Billich of Infinita, a Japanese firm delivering market intelligence and research, “online advertising expenditures (\$4.1 billion) exceeded combined radio and magazine advertising expenditures.”¹⁵

What's different now is the intense, immersive, and incessant nature of the marketing.



Special codes under Sprite bottle caps for MyCokeRewards.com

In 2006, Coca-Cola spent a total of \$1.9 billion on global marketing.¹⁶ In the summer of 2007, the company developed in China and brought stateside its “Sprite Yard,” a real-time digital community for teenagers. To accomplish this, the soda giant built its own global mobile network. Users chat, send messages, upload and share digital pictures, and download free content such as ring tones. Unlike other mobile social networks, the point of entry is Sprite’s single-serving bottles, whose caps contain a printed code that can trigger a text message when the user signs up and enters it. With that code, users can enter the Sprite digital world and customize their online personas, just as with other social networks like Facebook or MySpace.¹⁷

Another universe with music downloads, blogs and its own currency resides at MyCoke.com. While the company helps the user associate personality with brand identity, the teen is asked, “Ready to reinvent yourself?” The users can remake themselves by creating an avatar that can hang out in Coke Studios, where they can meet and chat with other avatars, play games, and download music. “You’ve just made millions of new friends!” blinks the message after registration. “People are cool. We’ll help you meet more of them.”¹⁸

Engagement in these sites is intense. MyCokeRewards.com customizes the experience for users based on 400 pieces of information the company captures on each user. The company can capture and record every click, every music download, every movement of every avatar. According to Promo Magazine, “That data is crunched, then spit back out in highly individualized messaging, reward recommendations, partner information and pro-

motions.” Members of MyCokeRewards.com average over nine minutes per visit on the site. Nearly six million rewards have been redeemed by the more than nine million members since the site first launched in 2006.¹⁹ Coca-Cola CEO Muhtar Kent said, globally, the soda giant has “19 million consumers, of which over 40% are under the age of 25” registered in their databases.²⁰

Soda sponsorship

Sports sponsorships proliferated during the late 1990s and early 2000s. Today, say soda market watchers, the big companies go deeper with fewer ventures. Coca-Cola’s worldwide sports sponsorship is estimated at between \$800 million and \$1 billion annually on the National Association for Stock Car Auto Racing, National Collegiate Athletic Association (NCAA), National Hot Rod Association, Professional Golfers’ Association of America, the U.S. Olympic Committee, and others.²¹

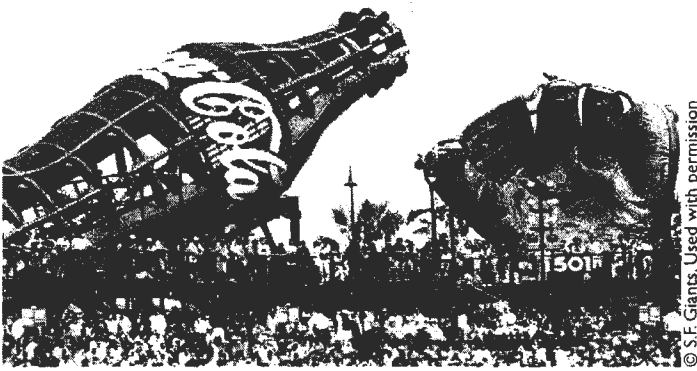
According to the weekly *SportsBusiness Journal*, Coke spent \$11 million in sports media in 2006. In 2007, the *Journal* reports, the company spent \$13 million during the NCAA basketball tournament to boost its new Coke Zero, targeted to male soda drinkers who have historically considered diet drinks for females.²²

The *Journal* also reports that “Pepsi, whose sports spending has dropped consistently for a number of years, doled out \$47.9 million for sports and entertainment sponsorships in 2006, per Nielsen. Cadbury Schweppes spent \$26.9 million.”²³ Carbonated beverage companies spent \$21.1 million in 2006 on athletic sponsorships targeted just to children under 18.²⁴

Along with AT&T and VISA, The Coca-Cola Company is a major sponsor of the \$350-million San Francisco Giants’ ballpark in San Francisco, named best sports facility in the country by the *SportsBusiness*

Journal in 2008.²⁵ The Coca-Cola brand dominates left field, giving it a carnival twist when lights come on at night games. The Coca-Cola Fan Lot was designed as an outlet where parents can watch the game while keeping an eye on the kids. On non-game days, the community can enjoy the area for free. Shaped as a giant Coca-Cola glass bottle, the main attraction is the Coca-Cola Superslide—located 465 feet from home plate, with two 56-foot-long curving slides (the “Guzzler”) and two 20-foot-long twisting slides (the “Twist-Off”). The bot-

Members of MyCokeRewards.com
average over nine minutes
per visit on the site.



Coca-Cola paid \$20 million to erect the huge bottle play structure in the Giant's baseball stadium.

tle weighs 130,000 pounds, rests at a 25-degree angle and is 47 feet tall at its highest point.

According to Stacey Slaughter, vice president of communications for the team, kids from 3 to 11 years of age gravitate toward the slide. Other attractions in this corner of Coke world include a giant baseball glove, Little Giants Park and a “fantasy photo booth.”

Few San Franciscans objected when the Coke bottle was proposed in 1998. Children’s advocate Margaret Brodtkin, concerned about the message it sent to children, couldn’t dissuade the Giants from erecting the humongous bottle—and collecting \$20 million from Coca Cola.²⁶

Brodtkin’s objection a decade ago to the giant Coke bottle in San Francisco’s baseball stadium fell on deaf ears. But that was before the rise in childhood obesity was evident. Nobody would build a Coke-bottle-shaped play structure now, Brodtkin says. Still, challenging the marketers isn’t easy. Even for a seasoned advocate like Brodtkin, going up against one of the nation’s largest marketers can be intimidating. “When you’re pushing the envelope, it’s scary and upsetting,” Brodtkin says. “I was stunned how alone I was when I objected to the Coke bottle. Everyone, including the superintendent of schools, was supporting it. They’d never do that today.”²⁷

Soda captures cultural icons: from Santa to American Idol

Childhood dreams were the stuff of Coke’s most ubiquitous, long-term ad campaign—Coca-Cola and Santa Claus. According to urban legend, the jolly, old St. Nick image we know today originated from annual Coke ads in which he wears the corporate colors. Santa didn’t al-

ways wear just red and white. The ruddy, sack-carrying Santa made the switch from the green, blue and other colors he was known to wear in the 19th century to the red suit and flowing white whiskers, which became the standard image by the 1920s.

“It was Coca-Cola’s magazine advertisements, billboards, and point-of-sale store displays that exposed nearly everyone in America to the modern Santa Claus image,” reports Snopes.com, the Web site that debunks urban myths. Though they didn’t invent him, “Coca-Cola certainly helped make Santa Claus one of the most popular men in America.”²⁸

Today, Coke is embedded in one of the biggest commercial fantasies of the 21st Century—*American Idol*. The Coca-Cola Company pays \$35 million to sponsor, along with Ford and AT&T, the most popular show on American television.²⁹ Being an *American Idol* sponsor means airing commercials during the show, posting on-line content about the show and their sponsorship, and running co-branded marketing programs off-air. The judges drink from red cups bearing the Coke logo, which also flashes behind performers on an on-stage billboard.

Soda companies also use cultural symbols and icons to target racial and ethnic groups. In July of 2008, Pepsi launched its Sierra Mist campaign with the tag line “Refresh your mind” and used Latino themes to create advertisements. With Latino actor Efrén Ramirez, commercials focused on humorous situations, in which a marriage-obsessed woman uses karate moves on other women to ensure she catches a wedding bouquet, or a man does anything to get fashionable clothing for free.³⁰ Pepsi has also targeted the Latino community through the creation of PepsiMusica, a bilingual entertainment program, and their “Blue Carpet Bash,” a VIP-style party for young Latinos. “It’s important for us to reach young Latinos with messaging that is relevant and authentic because obviously they are the future for us,” explained Martha Bermudez, senior manager of multicultural marketing at Pepsi-Cola North America.^{31,32}

To target African-Americans, in 2007 and 2008 Coca-Cola announced partnerships with two popular hip-hop artists, Jay-Z and Big Boi, for re-launching Cherry Coke and Full Throttle Fury. Jay-Z played a role in creating the look of the new Cherry Coke can. Full-Throttle Fury was a good product to target at African-American males, as “the orange flavor is one that resonates... specifically with African-American males.”^{33,34} Coke’s Full Throttle brand was also targeted at Latinos in Los Ange-

les, as the brand sponsored a Dodger baseball ticket giveaway at local grocery stores. Nearly half of those attending Dodger games are Latino.³⁵ In Houston, Texas, where Hispanic consumers make up 40% of consumers and are “getting wealthier and spending more on food and beverages than the average consumer,” according to *Beverage Digest*, Coca-Cola is targeting them with Mexican Coke, a product imported from Mexico, and with in-store materials promoting Coke as a product consumed in the home by families eating together.³⁶

One of the most successful examples of target marketing is Miles Thirst—a pitchman with a Chris Rock-like attitude who appeared on a series of Sprite ads starting in 2004. With his afro, gold chains, baggy jeans, and fur-trimmed coats, Thirst (“The Sprite Guy”) ended each commercial with, “Show ‘em my motto.” The motto—“Obey Your Thirst”—was the slogan for Sprite, a Coca-Cola product. Thirst toured NBA rookie star LeBron James’ crib (apartment) and became so popular that a 10-inch vinyl doll with his likeness became a collectors’ item.³⁷

The target marketing seems to be working, as people of color tend to drink more soda than other groups.³⁸

Soda marketed as health food

The latest culture in which soda is looking to embed itself is health. Nutrition professor and author Marion Nestle, who has chaired New York University’s nutrition department and helped develop U.S. Dietary Guidelines, is a voice for stopping the industry’s return to its 19th century roots of claiming soda can be a health boost.

Soda companies are marketing products infused with vitamins and minerals, when there is no evidence of these deficiencies among Americans, Nestle says.³⁹ One example: Coca-Cola’s Diet Coke Plus contains vitamins B₄, B₆, and B₁₂, along with zinc and magnesium. Only people who are sick and really poor (and sometimes iron-deficient, pregnant women) need supplements, according to Nestle. This is “misleading marketing” and is “deluding the public into thinking these things are healthier, when they’re not,” she says.

Nestle points to “a structural change in society” over the past 25 years as responsible for soda moving from what was once an occasional treat sold in 6-ounce bottles

to an every-day beverage sold in 20-ouncers and consumed in large amounts that threaten health. 7-Eleven’s Double Gulp, a 64-ounce soda, is 10 times the size of a Coca-Cola when it was first introduced to the market. With more than 800 calories, the Double Gulp is about one-third of the daily caloric requirement for the average person.⁴⁰

Gatorade and other electrolyte beverages are one way soda companies have bridged the gap between soda and so-called healthy beverages. Though infused with electrolytes, such beverages are still filled with sugar. Soda companies are taking advantage of concerns about health by marketing so-called “smart waters,” vitamin-infused bottled water. According to *Beverage Digest*, sales volume grew less than 1% for regular bottled water in the first half of 2008 after nearly a decade of triple- and double-digit growth.⁴¹ But the introduction of “functional” waters enhanced with vitamins has proven successful for many companies. An early 2008 survey found that nearly half of respondents reported purchasing a functional food or beverage in the previous three months, compared to about one-third of respondents in 2006.⁴² Vitamin waters appear to be a place where soda marketers are playing up health benefits to recover revenues from declining soda sales. With fortified products, soda companies are trying to cast a healthy glow across all their brands.

Some advocates say the companies have gone too far. In January 2009, Center for Science in the Public Interest (CSPI) filed a class-action lawsuit against Coca-Cola for

making deceptive and unsubstantiated claims on its Vitamin Water line of beverages. CSPI’s litigation director Steve Gardner says, “Vitamin Water is Coke’s attempt to dress up soda in a physician’s white coat. Underneath, it’s still sugar water, albeit sugar water that costs about ten bucks a gallon.”⁴³

In addition to health, soda companies have jumped on the “green” bandwagon and are marketing themselves as environmentally friendly. A February, 2008 article in *Advertising Age* reported a \$10-million marketing effort by Coca-Cola promoting “sustainability.” According to Coke’s president-general manager Hendrik Steckhan, the environmentalist frame has the advantage over the traditional health-and-wellness frame in that it allows Coke to

*“Vitamin Water is Coke’s attempt
to dress up soda in a physician’s
white coat. Underneath,
it’s still sugar water,
albeit sugar water that costs
about ten bucks a gallon.”*

Steve Gardner, CSPI

“focus on what it support[s],” rather than what it stands against. In other words, the health message puts the soda company on the defensive, while the environmental message puts it on the offensive.⁴⁴ (See our Framing Brief, *Food Marketers Greenwash Junk Food* for more on this tactic.)

Soda marketing as philanthropy

From the industry’s point of view, marketing in today’s health climate means countering criticism by showcasing its corporate good-guy self celebrating different cultures, joining health campaigns and being philanthropic.

Known for its historical emphasis on recruiting African-Americans and running successful campaigns to that market, PepsiCo recently accepted *Latina Style* magazine’s award as the number one of 50 top companies for Latinas. The company had participated in events for the National Society of Hispanic MBAs and National Council of La Raza, and created a Latino/Hispanic Advisory Board.⁴⁵

In 2003, The Coca-Cola Company Foundation awarded \$1 million to the American Association of Pediatric Dentists Foundation (AAPD).

“We approached them,” says John Rutkauskas, AAPD executive director. “The first grant we funded was for research on Xylitol, a sugar substitute found in gum that is thought to reduce bacteria that cause cavities.”⁴⁶

According to the director, the AAPD foundation board agreed that seeking and accepting big money from the world’s top soda maker conforms to its policies of “serving the best interest of children’s oral health, offering no actual or implied endorsement of products, and supporting AAPD’s mission and goals.”⁴⁷

In October of 2007, Coca-Cola opened The Coca-Cola Research Center for Chinese Medicine at the China Academy of Chinese Medical Sciences in Beijing, where the soda giant was a major sponsor of the 2008 Olympics.⁴⁸

Both Coke and Pepsi are on a three-year business partnership contract with the American Dietetic Association (ADA). Connie Diekman, ADA president, says that each of the organization’s six sponsors (including pharmaceutical giant GlaxoSmithKline) contribute financially at different

levels that are not considered public information. Sponsoring nutrition fact sheets under the association’s letterhead are among the supported activities.

“We shape their messages,” says Diekman. “They do not shape ours. By partnering, we can influence the influencers. We tell soda companies in our guidelines that we will not endorse their brand or promotion. We just want to get the right nutrition messages out and we have to partner everywhere to do this. The money allows us to do more of what we do well.”⁴⁹

Regarding the partnership, Marion Nestle blogs: “As long as your organization partners with makers of food and beverage products, its opinions about diet and health will never be believed independent (translation—based on science, not politics)...”⁵⁰

It is through philanthropy that Pepsi might at last top Coke. In the fall of 2007, the PepsiCo Foundation gave \$5.2 million to the Oxford Health Alliance, a global coalition aiming to prevent chronic disease. The grant supplements a three-year research and intervention project in England, China, India, and Mexico, to prevent further spread of obesity, tobacco use, and related illnesses.⁵¹

In Mexico, where both companies are active in schools, Coca-Cola is the object of consumer group *El Poder del Consumidor*’s protest for allegedly portraying the drink as one of several beverages that school children can use for hydration after physical activity. The Mexican group has joined the Global Dump Soft Drinks Campaign, organized by Center for Science in the Public Interest. Bruce Silverglade, CSPI legal director, says he has communicated with Coca-Cola’s representative about the hydration campaign.

“The Coca-Cola Company says it is going to look into it,” says Silverglade. “It says that the program was aimed at parents, not children and by the end of 2009, it wants a global policy that promotes physical activity in schools without promoting its brand.” According to Silverglade, the soda giant stood by its message that Coke “can be a source of hydration, but they’d be willing to

reconsider” that message.⁵²

Meanwhile, PepsiCo is working with the education ministry in Mexico on “Live Healthily”—a computer-centered program the soda company designed to help

“As long as your organization partners with makers of food and beverage products, its opinions about diet and health will never be believed independent (translation—based on science, not politics)...”
Marion Nestle

children learn how to make everyday decisions such as buying food and exercising.⁵³

According to Jo Tuckman of *The Guardian*, a 2006 national survey reveals that 72% of Mexican adults are “overweight or obese” and a quarter of Mexican children between the ages of 5 and 11 are “too heavy”—an increase of 40% since 2000. The reporter says that Mexican officials refuse to comment “on how major players in the junk food industry became the highest profile motors behind the fight against childhood obesity.”⁵⁴

At the end of May, 2008, the major soft drink companies announced that they would extend to the rest of the world their American pledges to stop targeting advertising to children under the age of 12.

Instead of mimicking the U.S. policy worldwide, says Silverglade, the companies should have agreed to the stricter curbs demanded by the British government and to an international code of marketing of foods and beverages to children that has been proposed by world-wide consumer organizations.

“Coke and Pepsi are proving that it’s hard to adopt a strong anti-obesity policy when your core products are major causes of obesity,” says Silverglade.⁵⁵

Soda self-regulation

The soda industry has always self-regulated its advertising, but by 2006, 43 states had enacted or introduced legislation to improve child nutrition in schools and the soda industry felt the pressure.⁵⁶ A national consortium of public health groups and lawyers was in negotiations with the companies when a one-time soda slugger, former President Bill Clinton, emerged as dealmaker.

At first, according to Ira Magaziner, a Clinton aide working in his foundation, soda companies fought against restrictions in high school beverages. The industry asked why students who were nearly old enough to fight in Iraq should be refused their soda of choice, said Magaziner.⁵⁷ But in the end, the companies decided to acquiesce. Even then, Magaziner reported that industry resistance was so strong that they had to negotiate “drink by drink” before reaching agreement.⁵⁸

The big three soda companies, along with the American Beverage Association (formerly the National Soft

Drinks Association), agreed that by 2009–10, all full-calorie soda would be removed from elementary and middle schools and replaced by bottled water, unsweetened fruit juices and low-fat milk. High schools could sell diet drinks, unsweetened tea, lower-calorie sports drinks, and flavored water.

The American Beverage Association announced a \$10 million ad campaign to “educate the country” about the new school beverage guidelines and the Alliance for a Healthier Generation was born, sponsored by Clinton’s Foundation and the American Heart Association, to make sure the goals were met.

Tricia Garrison, marketing and communications director for the Alliance for a Healthier Generation, reports that schools have been on track during the first of the three-year phase.

“Calories from beverages shipped to schools dropped 41% across America,” she says. “There has been a 45% reduction in shipments of full-calorie soft drinks to the schools. And the average high school student consumed less than half a can of full-calorie soft drinks a week in school (5.9 ounces), compared with a little more than a full can a week (12.5 ounces) in 2004. Shipments of water are also up by 23% since 2004.”⁵⁹

In November of 2006, nearly a year after the school drinks deal, the Council of Better Business Bureaus (CBBB) put together the Children’s Food and Beverage Advertising Initiative with the companies that accounted for two-thirds of children’s food and beverage TV advertising expenditures in 2004. Today,

15 companies, including the big three soda companies, have signed on to the voluntary, self-regulation program. Each has made a pledge to devote at least 50% of its advertising directed to children under 12 years of age to promote healthier dietary choices and/or to messages encouraging good nutrition and/or healthy lifestyles. PepsiCo’s pledge differs from The Coca-Cola Company’s in

that Pepsi will advertise Gatorade, baked Cheetos and crackers, as long as the ads show kids engaged in physical activity.

“Coke and Pepsi are in compliance as far as I can tell now,” says Elaine Kolish, director of the Initiative and a former Federal Trade Commission regulator for 25 years, who is in charge of assuring industry compliance.⁶⁰

“Coke and Pepsi are proving that it’s hard to adopt a strong anti-obesity policy when your core products are major causes of obesity.”

Bruce Silverglade, CSPJ

Dale Kunkel, professor of communications at the University of Arizona and one of the nation's leading researchers on children and media, is analyzing food and beverage industry compliance from February to May of 2008.

"Given the stakes involved, the industry clearly needs to pick up the pace of its reform efforts," Kunkel reports. "Thus far, the data reflect only a modest improvement in the nutritional quality of foods advertised to children. Advertising for unhealthy foods still predominates in the most recent studies examining food marketing to children."⁶¹ (Kunkel has not separated soda producers from industry participants that produce food and soda.)

Nutrition expert Nestle doubts Clinton's school deal, the CBBB's initiative or any self-regulation can protect children's health.

"There's so much evidence that they're only giving lip service to this," she says. "They can't do what they say, because they won't sell products if they do. They're not a public health agency. Either they have to go into another business or figure out some other way. They're not going to sell healthy products to kids."⁶²

To Nestle, the Clinton alliance was "a way for soda companies to keep vending machines in schools." Why are they selling water to kids in cities where water is free and good quality, she asks. "They've convinced people that the water from fountains is bad. Gatorade is still a soft drink with sugar that has nothing to do with sports and gives kids the idea that they have to eat and drink all the time." The vending machines keep the brand in front of children and generate good feelings about the company.

What do the experts think?

In 2004, an American Psychological Association task force led by researcher Kunkel recommended that advertising targeting children under the age of 8 be restricted. After several years of research review, the team found that children under that age lack the cognitive development to understand the persuasive intent of television advertising and are uniquely susceptible to advertising's influence. Children recall content from ads to which they've been exposed, according to the research, and preference for a product has been shown to occur with as little as a single commercial exposure and strengthened with repeated exposures.⁶³

Almost twice as many American children (2-11 years of age) are watching American Idol than SpongeBob SquarePants.

"*American Idol* is a Coke ad," says Kunkel.

The Coca-Cola Company says it does not advertise to children under the age of 12 when they are 50% or more of the TV viewing audience. But according to Kunkel, that assertion is "grossly oversimplified"

He says that the soda giant "is trying to say, 'We're not targeting ads in programs made exclusively for children.' But they're implying that their advertising is not seen by substantial numbers of children, and millions of children see Coke ads every day."

The way soda companies reach children and teenagers is through family entertainment. Just ask Diana Garza-Ciarlante, communications director for Coca-Cola, North America.

"Coca-Cola respects the sanctity of childhood," she says. "With *American Idol*, the issue becomes a question of programming. This is family programming, on the air 8 p.m. or later. Even at its height of *Idol* popularity, children under the age of 12 were 7 or 8% of the audience. Children are not alone. They're not in a bubble. We need to be realistic ... That said, we have a responsibility to present [the product] in a place appropriate to the brand. Family environments are appropriate. The expectation is that the parent or caregiver is making the decision whether or not it's appropriate to be exposed to the programming."⁶⁴

Garza-Ciarlante is correct about the percent of children in the *Idol* audience on average. But when accounting for the millions of children the percentage represents, almost twice as many American children (2-11 years of age) are watching *American Idol* than *SpongeBob Square Pants*.

According to Anne Elliot, vice president of communications for The Nielsen Company, *American Idol* averaged 29.4 million viewers during the 2007 season.⁶⁵ The age distribution shows that:

- *American Idol* averaged 2.3 million kids (2-11), which is 5.7% of all kids in TV homes.
- The show averaged 1.9 million teens (12-17), which is 7.5% of the teens in TV homes.
- It averaged 14.2 million adults (18-49), which is 11% of adults of those ages in TV homes.

Elliot looked at *SpongeBob* during the week of April 21, 2008, when there were 56 telecasts of the program:

- *SpongeBob's* average kids (2-11) audience was 1.5 mil-

lion—or 3.7%—of kids in TV homes.

- The top-rated *SpongeBob* telecast had 2 million kids in the audience, which is 5.3% of kids in TV homes.

“If you compare the average number of children viewing an episode of *American Idol* to the average number watching *SpongeBob*, *American Idol* will win every time,” says Kunkel.

To Nestle, it is “thinkable” that soda advertising on a show with a child audience like that of *American Idol*, could be regulated. “Other countries do this,” she says. (The United Kingdom, for instance, forbids soft drink broadcast advertising to youngsters under the age of 16.)

Conclusion

Research shows TV advertising influences children’s preferences and purchase requests. With soda companies’ foray into digital marketing, where children spend not 30 seconds as with TV commercials but sometimes hours on end, the situation is more urgent. And with soda companies intensively targeting racial and ethnic communities and co-opting the language and arguments of their critics to position themselves on the side of health and the environment, it is more important than ever for public health advocates to keep the industry in check. In spite of self-regulation, sugary sodas remain the top non-alcoholic beverage in America. It’s no wonder, given the major spending of soda marketers and the near ubiquity of their ads.

The large sums of cash thrown at popular TV shows like *American Idol* belie soda industry claims that they are serious about children’s health or eager to cut back on advertising. Says Andrew Kaplan, editor of *Beverage World*, referring to soda companies’ online marketing tactics and Coca-Cola’s sponsorship of *Idol*: “I don’t think soda companies are cutting down on anything that communicates to teenagers.”⁶⁶

References

- 1 West, Larry. What is the Problem with Soft Drinks? *The Environmental Magazine*, 2007. Available at: http://environment.about.com/od/health/a/soft_drinks.htm, accessed on January 21, 2009.
- 2 Zimuda, Natalie. Pepsi Upends Brands With \$1.2 Billion Shake-Up. *Advertising Age*, October 20, 2008, page 6.
- 3 Interview with John Sicher, editor and publisher, *Beverage Digest*, conducted by Hilary Abramson in May, 2008.
- 4 McKay, Betsy. Downsize This! After Years of Supersizing, Food Makers Shrink Portions (and Fatten Profit Margins). *Wall Street Journal*, January 27, 2004, page B1.
- 5 Sicher interview.
- 6 TNS Media Intelligence, Report From 1/1/06–12/31/07.

- 7 *Beverage Digest*. Special Issue: Top-10 CSD Results for 2007. March 12, 2008, 52(5).
- 8 Sicher interview.
- 9 Federal Trade Commission. Marketing Food to Children and Adolescents: A Review of Industry Expenditures, Activities, and Self-Regulation. Federal Trade Commission, July, 2008.
- 10 McClellan, Steve. ‘The Hills’ Is Alive; MTV research links cross-platform marketing to brand affinity among Web users. *Adweek.com*, May 5, 2008. Available at: www.adweek.com/aw/content_display/research/e3i26f1bfd408799a207c5f2ec5f3a350ae, accessed on November 14, 2008.
- 11 McClellan, 2008.
- 12 Emigh, Jacqueline. ‘Branded’: Kids’ Web sites Sell Virtual Pepsi, Disney. Avatar. *BetaNews*, January 30, 2008. Available at: www.betanews.com/article/Branded_Kids_Web_sites_sell_virtual_Pepsi_Disney_avatar_gear/1201709774, accessed on November 14, 2008.
- 13 Emigh, 2008.
- 14 Billich, Christopher. 21st Century Mobile Marketing Global Insights into the World’s Most Advanced Mobile Market: Japan. *Infinita Inc.*, March 19, 2008. Available at: <http://mmaglobal.com/?q=node/2292>, accessed on November 14, 2008.
- 15 Billich, 2008.
- 16 *Advertising Age*. Top 100 Global Marketers. *Advertising Age’s* 21st Annual Global Marketers Part 1: Global Ad Spending by Marketer, November 19, 2007, page 4.
- 17 The Coca-Cola Co. News Release: Sprite Pioneers New Consumer Connections Through Consumer Technologies. TheCoca-ColaCompany.com, June 6, 2007. Available at: www.thecoca-colacompany.com/presscenter/nr_20070606_sprite_yard.html, accessed on November 17, 2008.
- 18 MyCoke.com. Welcome to My Coke. Available at: <http://mycoke.com>, accessed on November 17, 2008.
- 19 *Promo Magazine*. Rewarding Redemption. May 1, 2008. Available at: http://promomagazine.com/incentives/cocacola_redemption/, accessed on January 21, 2009.
- 20 World Advertising Research Center. Coca-Cola focuses on digital. July 23, 2009. Available at www.warc.com/News/TopNews.asp?ID=25443, accessed August 29, 2009.
- 21 Stanley TL. Seeking more pop from sponsorship: Why soft drink companies are taking a hard look at the money they spend in sports. *Sports-Business Journal*, May 14, 2007, page 17.
- 22 Stanley, 2007.
- 23 Stanley, 2007.
- 24 Federal Trade Commission, July, 2008.
- 25 AT&T Park Recognized as the 2008 Sports Facility of the Year. *Sports-Business Journal*, May 26, 2008, page 60.
- 26 Levy D. Giants prevail in tussle over big coke bottle. *San Francisco Chronicle*, October 23, 1998.
- 27 Interview with Margaret Brodtkin, conducted by Lori Dorfman in March, 2009.
- 28 Snopes.com. The Claus That Refreshes. Available at: www.snopes.com/cokelore/santa.asp, accessed on November 14, 2008.
- 29 Gunelius, Susan. Ford, Coke & AT&T Pay More to Sponsor American Idol. *Brandcurve.com*, January 18, 2008. Available at: www.brandcurve.com/ford-coke-att-pay-more-to-sponsor-american-idol/, accessed on November 14, 2008.
- 30 De Lafuente, Della. Pepsi’s Sierra Mist Aims Ads at Multicultural Consumers. *MarketingMedios.com*, July 14, 2008. Available at: www.marketingmedios.com/marketingmedios/noticias/article_display.jsp?vnu_content_id=1003827156, accessed on November 14, 2008.

- 31 De Lafuente, Della. Research: Report: McD's, Pepsi Score Best With Young Hispanics. *Brandweek.com*, April 21, 2008. Available at: www.brandweek.com/bw/magazine/current/article_display.jsp?vnu_content_id=1003791582, accessed on November 14, 2008.
- 32 De Lafuente, Della. Pepsi Gives Gen Y Latinos VIP Party Pass. *Brandweek.com*, January 17, 2008. Available at: http://login.vnuemedia.com/bw/eseach/article_display.jsp?vnu_content_id=1003697496, accessed on November 14, 2008.
- 33 Hein, Kenneth. Strategy: Cherry Coke Gets Fresh Jay-Z Remix. *Brandweek.com*, January 29, 2007. Available at: www.brandweek.com/bw/magazine/current/article_display.jsp?vnu_content_id=1003538550&&imw=Y, accessed on November 14, 2008.
- 34 Maul, Kimberly. Coke relaunches energy drink with hip-hop flair. *PR Week*, October 6, 2008, page 2.
- 35 Ayala, Nancy. Sports Toss More Than Soft Pitches for Latinos. *AdWeek.com*, June 11, 2007. Available at: www.adweek.com/aw/research/article_display.jsp?vnu_content_id=1003596731, accessed on November 14, 2008.
- 36 *Beverage Digest*. Coke Undertaking Several Initiatives for CSIDs. BID Market Tour Observations, November 7, 2008. 53(10):3-4.
- 37 TV Acres. Advertising Mascots – Objects. Available at: www.tvacres.com/admascots_sprite.htm, accessed on November 17, 2008.
- 38 Rehm CD, Mutte TD, Van Wye G, Young C, Frieden TR. Demographic and behavioral factors associated with daily sugar-sweetened soda consumption in New York City adults. *Journal of Urban Health*. May, 2008, 85(3):375-385.
- 39 Interview with Marion Nestle, conducted by Hilary Abramson between April and June, 2008.
- 40 Young MR, Nestle M. The contribution of expanding portion sizes to the US obesity epidemic. *American Journal of Public Health*. February, 2002, 92(2):246-249.
- 41 Hein, Kenneth. Has The Bottled Water Well Finally Run Dry? *Brandweek*, September 8, 2008. Available at: www.brandweek.com/bw/content_display/news-and-features/packaged-goods/e3id9a975e26c8545c5072c754a26383666, accessed on November 14, 2008.
- 42 O'Leary, Noreen. Junk Science. *AdWeek*, September 22, 2008. Available at: www.adweek.com/aw/content_display/special-reports/other-reports/e3ifb7c0deaad627f7c543424ea8c0daca6, accessed on November 14, 2008.
- 43 See http://cspinet.org/new/pdf/vitaminwater_filed_complaint.pdf, accessed on January 21, 2009.
- 44 Bush, Michael. Sustainability and a smile: Coca-Cola joins green-marketing crush, tries to redefine the term in \$10 mil push. *Advertising Age*, February 25, 2008, page 1.
- 45 LATINA Style Magazine Announces the 10 Year Anniversary of the LATINA Style 50. *CapitalWirePR*, May 14, 2008. Available at: http://capitalwirepr.com/pr_description.php?id=2eabac75-dbbd-4081-0182-482b53bb486b, accessed on November 17, 2008.
- 46 Interview with John Rutkauskas, conducted by Hilary Abramson between April and June, 2008.
- 47 Rutkauskas interview.
- 48 The Beverage Institute For Health & Wellness. The Coca-Cola Company Steps Up Innovation With New Research Center For Chinese Medicine, October 14, 2007. Available at: www.beverageinstitute.org/beverages_and_health/chinese_medicine.shtml, accessed on November 18, 2008.
- 49 Interview with Connie Diekman, conducted by Hilary Abramson between April and June, 2008.
- 50 Nestle, Marion. OK. Time to talk about the American Dietetic Association's Alliances with Food Companies. *WhatToEatBook.com*, March 17, 2008. Available at: <http://whattoeatbook.com/tag/american-dietetic-association/>, accessed on November 17, 2008.
- 51 PepsiCo. Wellness/News, September 26, 2007. Available at: www.pepsico.com/PEP_Citizenship/HealthWellness/News, accessed between April and June, 2008.
- 52 Interview with Bruce Silverglade, conducted by Hilary Abramson between April and June, 2008.
- 53 Tuckman, Jo. Pepsi tackles childhood obesity with videogames in Mexico. *The Guardian*, January 2, 2008. Available at: www.guardian.co.uk/world/2008/jan/02/mexico.jotuckman, accessed on November 17, 2008.
- 54 Tuckman, 2008.
- 55 Silverglade interview.
- 56 Kluger, Jeffrey. How Bill Sealed the Soda Deal. *TIME*, May 7, 2006. Available at: www.time.com/time/printout/0,8816,1191803,00.html, accessed on November 17, 2008.
- 57 Kluger, 2008.
- 58 Kluger, 2008.
- 59 Interview with Tricia Garrison, conducted by Hilary Abramson between April and June, 2008.
- 60 Interview with Elaine Kolish, conducted by Hilary Abramson between April and June, 2008.
- 61 Interview with Dale Kunkel, conducted by Hilary Abramson between April and June, 2008.
- 62 Nestle interview.
- 63 Wilcox B, Cantor J, Dowrick P, et al. Report of the APA Task Force on Advertising and Children. February 20, 2004.
- 64 Interview with Diana Garza-Ciarlante, conducted by Hilary Abramson between April and June, 2008.
- 65 Interview with Anne Elliot, conducted by Hilary Abramson between April and June, 2008.
- 66 Kaplan interview.
- 67 Stanley TL. Seeking more pop from sponsorship: Why soft drink companies are taking a hard look at the money they spend in sports. *Sports-Business Journal*, May 14, 2007, page 17.
- 68 PepsiCo. Timeline of Diversity. Available at: www.pepsico.com/PEP_Diversity/timeline/index.cfm, accessed on November 17, 2008.
- 69 McKay, Betsy. Coke, Pepsi Bottlers Try New Sizes to Boost Sales. *Wall Street Journal*, May 2, 2008, page B3.

This Framing Brief was written by Hilary Abramson and edited by Lori Dorfman for the Strategic Alliance's Rapid Response Media Network and The California Endowment's Healthy Eating, Active Communities program. Both Strategic Alliance and the Rapid Response Media Network are funded by the California Endowment. Thanks to the Prevention Institute, the California Center for Public Health Advocacy, Public Health Law and Policy, and the Center for Weight and Health for their thoughtful review and assistance. Special thanks to Jeff Chester from the

Center for Digital Democracy for generously sharing his knowledge of digital marketing campaigns.

For more information about this piece or the Rapid Response Media Network, contact Sana Chehimi, sana@preventioninstitute.org or 510-444-7738.



OVERVIEW OF THE IOM REPORT ON *FOOD MARKETING TO CHILDREN AND YOUTH: THREAT OR OPPORTUNITY?*

Through a congressional directive, the Centers for Disease Control and Prevention (CDC) requested that the Institute of Medicine (IOM) of the National Academies conduct a study to review the influence of food marketing on the diets and health of children and youth in the United States. *Food Marketing to Children and Youth: Threat or Opportunity?* explores what is known about current food and beverage marketing practices, the influence of these practices on the diets and health of children and youth, and public and private strategies that can be used to promote healthful food and beverage choices in children and youth. The report was prepared by an Institute of Medicine (IOM) committee, chaired by Dr. J. Michael McGinnis, that convened 16 members with expertise in nutrition, child and adolescent development, psychology, media and advertising, consumer marketing and behavior, social marketing, evaluation, education, public health and policy, industry (e.g., food, beverage, and entertainment), constitutional law, and business ethics.

Dietary Patterns of Children and Youth

The diets of America's children and adolescents depart substantially from recommendations and reflect a pattern that puts their health at risk. Overall, children and youth are not achieving basic nutritional goals. They are consuming excessive calories and exceed recommended intakes of total fat, saturated fats, added sugars, and sodium. The report reveals that the dietary and health-related patterns of children and youth are influenced by the interplay of many factors, including genetics and biology, culture and values, economic status, physical and social environments, and commercial and media environments. Among these environments, the media, in its multiple forms and broad reach, plays a central socializing role for young people and is an important channel for promoting branded food and beverage products in the marketplace.

MAJOR CONCLUSIONS

- Along with many other intersecting factors, food and beverage marketing influences the diets and health prospects of children and youth.
- Food and beverage marketing practices geared to children and youth are out of balance with recommended healthful diets and contribute to an environment that puts their health at risk.
- Food and beverage companies, restaurants, and marketers have underutilized the potential to devote creativity and resources in promoting food, beverages, and meals that support healthful diets for children and youth.
- Achieving healthful diets for children and youth will require continued, multisectoral, and integrated efforts that include industry leadership and initiative.
- Public policy programs and incentives do not currently have the support or authority to address many of the current and emerging marketing practices that influence the diets of children and youth.

RECOMMENDATIONS

A Multi-Faceted Approach to Improve the Diet-Related Health of Children and Youth

This report presents recommendations for different segments of society to guide the development of effective marketing strategies that promote healthier food, beverage, and meal options to children and youth. Recommendations are also offered for research necessary to chart the path of future improvements, and the capacity to monitor and track improvements in marketing practices that have an influence on children's and youth's diets and diet-related health. These recommendations reflect the current context and information in a rapidly changing environment, and should be implemented together as a package to support and complement one another.

Food, Beverage, and Restaurant Industries

The food, beverage, and restaurant industries should use their creativity, resources, and full range of marketing practices to promote and support more healthful diets for children and youth. To achieve this, the industries should:

- Shift their product portfolios in a direction that promotes new and reformulated child- and youth-oriented foods and beverages that are substantially lower in total calories, lower in fats, salt, and added sugars, and higher in nutrient content.
- Shift their advertising and marketing emphasis to child- and youth-oriented foods and beverages that are substantially lower in total calories, lower in fats, salt, and added sugars, and higher in nutrient content.

- Restaurants should expand and actively promote healthier food, beverage, and meal options for children and youth and provide calorie content and key nutrition information on menus and packaging that is prominently visible at the point of choice and use.

- Engage the full range of their marketing vehicles and venues to develop and promote healthier, appealing, and affordable foods and beverages for children and youth.

Advertising, Marketing, Entertainment Industry, and Media

The food, beverage, restaurant, entertainment, and marketing industries should work with government, scientific, public health, and consumer groups to establish and enforce the highest standards for the marketing of foods, beverages, and meals to children and youth. To achieve this, it should:

- Work through the Children's Advertising Review Unit (CARU) to revise, expand, enforce, and evaluate explicit industry self-regulatory guidelines beyond traditional advertising to include evolving vehicles and venues for marketing communications.
- Assure that licensed characters are used only to promote foods and beverages that support healthful diets for children and youth.

The media and entertainment industry should direct its extensive power to promote healthful foods and beverages for children and youth. To achieve this, it should:

- Incorporate into the multiple media platforms (e.g., print, broadcast, cable, the Internet, and wireless-based programming) foods, beverages, and storylines that promote healthful diets.
- Strengthen their capacity to serve as accurate interpreters and reporters to the public on findings, claims, and practices related to the diets of children and youth.

Parents, Caregivers, and Families

To support parents, caregivers, and families in promoting healthful diets for children and youth, the government, in partnership with the private sector, should create a long-term, multi-faceted, and financially sustained social marketing program that should:

- Include a full range of evolving and integrated marketing tools with widespread educational and community-based efforts.
- Target parents of children from birth to the age of four years to build skills for selecting healthful and affordable food and beverage choices for their children.
- Offer a reliable support stream that should be in place for social marketing programs through public-appropriated funds and counterpart cooperative support from the businesses that market foods, beverages, and meals to children and youth.

Government

- Government, in partnership with the private sector, should create a long-term, multi-faceted social marketing program targeting parents, caregivers, and families to promote healthful diets for children and youth (see above recommendation).
- Government at all levels should marshal the full range of public policy approaches (e.g., subsidies, taxes, legislation, regulation, federal nutrition programs) to foster the development and promotion of healthful diets for children and youth.
- If voluntary efforts related to advertising during children's television programming are unsuccessful in shifting the emphasis away from high-calorie and low-nutrient foods and beverages to the advertising of healthful foods and beverages, Congress should enact legislation mandating the shift on both broadcast and cable television.

- The nation's formidable research capacity should be better directed to sustained, multidisciplinary work on how marketing influences the food and beverage choices of children and youth.
- The Secretary of the U.S. Department of Health and Human Services should designate a responsible agency, with adequate and appropriate resources, to formally monitor and report regularly on the progress of the various entities and activities related to the recommendations included in this report.

Schools

State and local educational authorities, with support from parents, health authorities, and other stakeholders, should educate about and promote healthful diets for children and youth in all aspects of the school environment (e.g., commercial sponsorships, meals and snacks, curriculum). To achieve this, it should:

- Develop and implement nutrition standards for all competitive foods and beverages sold or served in the school environment.
- Adopt policies and best practices that promote the availability and marketing of foods and beverages that support healthful diets.
- Provide visible leadership in this effort by public and civic leaders at all levels such as the National Governors Association, the State and Local Boards of Education and the Parents Teachers Organization, as well as trade associations representing private-sector businesses such as distributors, bottlers, and vending machine companies that directly interface with the school administration.

State of Food and Beverage Marketing to Children and Youth: Influence on Diets and Health

The commercial advertising and marketing of food and beverages are intersecting factors that influence the diets and diet-related health of children and youth. The review indicates that, among many factors, food and beverage marketing influences the preferences and purchase requests of children, influences short-term consumption, may contribute to less healthful diets, and contributes to an environment that puts their health at risk.

- Advertising and marketing messages reach young consumers through a variety of vehicles such as television, radio, magazines, music, and the Internet, and through many different venues including homes, schools, child-care settings, grocery stores, shopping malls, theaters, sporting events, and airports.
- Food advertising to children affects their preferences, purchase behaviors, and consumption habits for different food and beverage categories, as well as for different product brands.
- Food and beverage advertising on television influences children ages 2–11 years to prefer and purchase high-calorie and low-nutrient foods and beverages.
- Of the more than \$200 billion children and youth collectively spend annually, the top four leading items children ages 8–12 years select, without parental permission, are high-calorie and low-nutrient foods and beverages.
- Food and beverages, particularly candy, carbonated soft drinks, and salty snacks or chips, were ranked among the top leading items that teens ages 13–17 years old purchase with their own money.
- The purchase influence of children and youth increases with age and is currently estimated at \$500 billion for 2–14 year-olds.

Committee on Food Marketing and the Diets of Children and Youth

J. MICHAEL MCGINNIS (*Chair*), Institute of Medicine, Washington, DC; **DANIEL R. ANDERSON**, Department of Psychology, University of Massachusetts, Amherst; **J. HOWARD BEALES III**, School of Business, George Washington University, Washington, DC; **DAVID V. B. BRITT**, Sesame Workshop (*Sesame Street*), Amelia Island, FL; **SANDRA L. CALVERT**, Children's Digital Media Center, Georgetown University, Washington, DC; **KEITH T. DARCY**, Ethics Officer Association, Waltham, MA; **AIMÉE DORR**, Graduate School of Education and Information Studies, University of California, Los Angeles; **LLOYD J. KOLBE**, Department of Applied Health Science, Indiana University Bloomington; **DALE L. KUNKEL**, Department of Communication, University of Arizona, Tucson; **PAUL KURNIT**, KidShop, Kunit Communications, and Lubin School of Business at Pace University, Chappaqua, New York; **ROBERT O. POST**, Yale Law School, New Haven, CT; **RICHARD SCHEINES**, Department of Philosophy, Carnegie Mellon University, Pittsburgh, PA; **FRANCES H. SELIGSON**, Nutrition Consultant, Hershey, PA; **MARY STORY**, Division of Epidemiology, School of Public Health, University of Minnesota Minneapolis; **ELLEN A. WARTELLA**, Office of the Executive Vice Chancellor and Provost, University of California, Riverside; **JEROME D. WILLIAMS**, Department of Advertising, University of Texas, Austin

Liaison from the Food and Nutrition Board

NANCY F. KREBS, Department of Pediatrics, University of Colorado Health Sciences Center, Denver

Staff

Study Directors: **JENNIFER A. GOOTMAN** and **VIVICA I. KRAAK**

Research Associates: **LESLIE J. SIM** and **SHANNON L. WISHAM**

Interns: **AMIEE M. ADASCZIK** and **KELLY D. HORTON**

Download fact sheets and the executive summary at: www.iom.edu/kidsfoodmarketing.

Copies of *Food Marketing to Children and Youth: Threat or Opportunity?* are available at www.nap.edu.

The Impact of Industry Self-Regulation on the Nutritional Quality of Foods Advertised on Television to Children

Commissioned by:
Children Now

Conducted by:
Dale Kunkel, Ph.D.
Christopher McKinley, M.A.
Paul Wright, M.A.
University of Arizona

December 2009

Executive Summary **3**

Background	5
Report Objectives	6
Key Findings	6
Conclusion	7

Complete Report **9**

Background	11
Method	13
Results	15
Conclusion	34

Appendix A **39**

Executive Summary

Background

For the first time in modern history, the current generation of children may face a life expectancy that is shorter than that of their parents. This is due to the childhood obesity epidemic. Among the many health complications associated with childhood obesity are the earlier onset and growing rates of type 2 diabetes, high blood pressure and heart disease. There is a strong consensus that aggressive actions are urgently needed to better defend the nation's children from this growing crisis.

Numerous factors have been shown to contribute to childhood obesity, including reduced physical activity, the wider availability of nutritionally poor convenience foods, fewer family meals and advertising that promotes unhealthy foods. This study addresses food advertising to children, a factor of particular interest because it impacts virtually every child in the nation. Children are exposed to tens of thousands of commercials each year on television alone, including ads for fast food, sugared cereals and sugared beverages. Most of these foods are high in added sugar, salt and fat, and they are unhealthy when consumed on a regular basis.

In 2004, Congress commissioned the Institute of Medicine (IOM) of the National Academies to evaluate the role of food marketing as a contributing factor to childhood obesity. The IOM report, released in 2006, reviewed all existing scientific studies and determined that food and beverage advertising targeted at children influences their product preferences, requests and diet. It concluded that "food and beverage marketing practices geared to children and youth are out of balance with healthful diets, and contribute to an environment that puts their health at risk" (Institute of Medicine, 2006, p. 10).

Given the severity of the childhood obesity epidemic, the IOM recommended that the food and beverage industry shift its marketing practices to children away from products high in added sugar, salt and fat, and toward healthy products that children can safely consume as part of their everyday diet. To underscore the importance of this goal, the IOM specified that if the industry proved unable to achieve such reform voluntarily, Congress should intervene with legislation.

The IOM's conclusions confirmed the role of food and beverage marketing practices in the childhood obesity crisis, subsequently increasing attention to the issue among public health officials and children's advocates. In response to this growing pressure for change, the food and beverage industry responded with a self-regulatory program aimed at reducing unhealthy food advertising to children. This program is known as the Children's Food and Beverage Advertising Initiative.

The Children's Food and Beverage Advertising Initiative

In 2006, in partnership with the Council of Better Business Bureaus, a coalition of major food companies announced that it would significantly improve the nutritional quality of foods advertised to children. The publicly stated goal of this voluntary industry effort, called the Children's Food and Beverage Advertising Initiative, is to "change the landscape of child-directed advertising" by encouraging healthier dietary choices and healthy lifestyles in all advertising to children (Peeler, Kolish, & Enright, 2009, p.1).

The initiative introduced the term "better-for-you" to identify the products that participating companies had self-selected as the healthier food and beverage products they would continue to advertise to children. The initiative, however, lacked uniform criteria specifying the minimum nutritional standards for the "better-for-you" designation. Rather, each of the participating companies issued its own detailed pledge that defined "better-for-you" in its own way, resulting in substantial variability in the nutritional criteria used from one company to the next.

At the time this study was conducted, 15 companies were participating in the initiative (please see page 11 for company list), in which they publicly pledged to dramatically improve the nutritional profile of their food marketing to children. One additional company (Post Foods) has joined the initiative since then, bringing the current number of participants to 16.

Report Objectives

The Impact of Industry Self-Regulation on the Nutritional Quality of Foods Advertised on Television to Children provides the first independent, comprehensive evaluation of the Children's Food and Beverage Advertising Initiative and its impact on the children's food marketing environment on television. Commissioned by Children Now and conducted by Dale Kunkel, Ph.D., and colleagues at the University of Arizona, this research report examines the food advertising environment during children's television programming. The report compares advertising patterns in 2005, several years before the Children's Food and Beverage Advertising Initiative went into effect, to those in 2009, after the initiative was in place. The data from this report indicate the extent to which this initiative has succeeded at achieving the goals specified by the Institute of Medicine in 2006.

One of the key measures Dr. Kunkel used to assess the impact of the Children's Food and Beverage Advertising Initiative is the U.S. Department of Health and Human Services' Go-Slow-Whoa food rating system. This framework is part of the We Can! (Ways to Enhance Children's Activity & Nutrition) program, designed to help parents make healthier choices for their children and families. Please refer to the Appendix of this report for information on the Go-Slow-Whoa food rating system.

Key Findings

The majority of advertisements from companies participating in the Children's Food and Beverage Advertising Initiative are for nutritionally poor Whoa products, which should only be consumed on special occasions (see Figure 1, p. 7).

Despite industry claims that food marketing to children would be limited to healthier products through the initiative, this study finds that more than two-thirds (68.5%) of all advertising by participating companies is for foods and beverages in the Whoa category, the lowest category of nutritional quality. These Whoa products should be consumed only on "special occasions, such as your birthday."

Roughly one-third (31%) of the food ads from companies participating in the initiative are for Slow products, which have moderate nutritional value but should be consumed only "sometimes, at most several times a week."

Healthy food advertising is invisible.

Ads for truly healthy Go products, such as vegetables, fruits, whole grain breads and other products that can be consumed "anytime," account for less than 1% of all advertising from participating companies. There is no increase in the proportion of ads for healthy products in 2009 from 2005, before the initiative went into effect.

It would require 10 hours of viewing children's television programs to find one healthy food ad. During that same period, a child viewer would see 55 ads for Whoa foods and 20 ads for Slow foods. In summary, fewer than one in 100 food ads promote a healthy product that can be eaten safely on a daily basis.

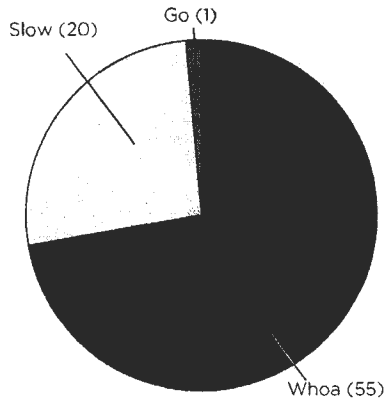
Licensed characters are increasingly used to promote nutritionally poor food and beverage products to children.

Research shows that licensed characters are particularly effective at influencing children because children trust the characters they see in program content. Consequently, the Institute of Medicine's report recommended that licensed characters should be used "only for the promotion of foods and beverages that support healthful diets." Yet companies participating in the initiative have nearly doubled their use of licensed characters over the past four years, from 8.8% in 2005 to 15.2% in 2009, and roughly half of all ads with such characters (49.4%) are for nutritionally poor Whoa products.

Despite the industry's self-regulatory pledges, which specify that participating companies will only use licensed characters to promote their "better-for-you" products, none of the healthier foods and beverages they marketed with licensed characters qualify as a Go product that children can consume every day.

More than one-quarter of all food and beverage advertising to children originates from companies that do not participate in the initiative.

FIGURE 1
Nutritional Quality of Food Ads in 10 Hours of Children's Programs



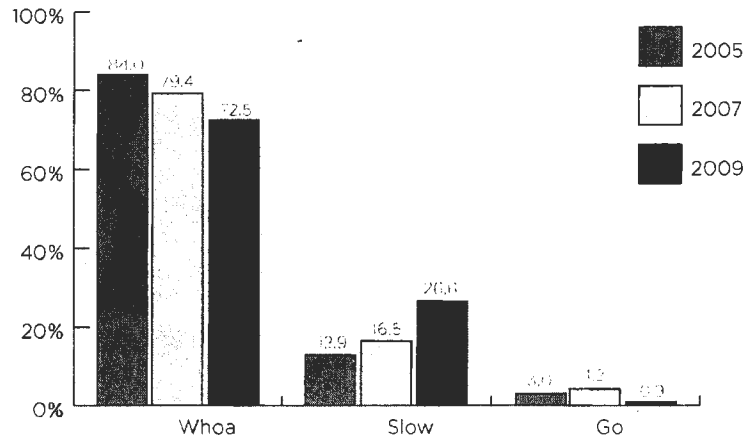
Across all children's food ads on television, 28.7% are by companies that do not participate in the Children's Food and Beverage Advertising Initiative; therefore, their marketing practices to children are not guided by its measures.

In addition, the major broadcast networks and cable channels that deliver children's programming and advertising play no role in the initiative. This creates another loophole, allowing a substantial proportion of food advertising to children to occur, without holding it to standards regarding the nutritional quality of the advertised products.

Under self-regulation, overall improvement in the nutritional quality of foods marketed on television to children is negligible (see Figure 2, this page).

Despite calls for dramatic reform from public health officials and advocates, food and beverage advertising to children continues to be predominated by products of poor nutritional value. In 2005, prior to the inception of the Children's Food and Beverage Advertising Initiative, 84% of foods marketed to children were for Whoa products. In 2009, Whoa products have decreased only to 72.5%. Thus, at this pace, it would take until 2017 for nutritionally poor Whoa products to decline to only half of all foods marketed to children and until 2033 for them to disappear entirely.

FIGURE 2
Over Time Comparisons of Nutritional Quality in Food Ads for Industry, Overall



Conclusion

The findings in this report demonstrate that the Children's Food and Beverage Advertising Initiative has not improved the overall nutritional quality of ads targeting children. Moreover, the food and beverage industry has failed to meet the Institute of Medicine's principal recommendation to voluntarily shift the balance of children's food marketing away from low-nutrient, high-density foods to "advertising strategies that promote healthier foods, beverages, and meal options."

The advertising environment targeting children continues to expose them to nutritionally poor food products, contributing to the current childhood obesity epidemic. Children Now's study illustrates that the Children's Food and Beverage Advertising Initiative has failed to significantly improve this situation. As such, it is time for our nation's leaders to step forward and help ensure a healthy food advertising environment for our children.

Complete Report

Background

Childhood obesity has become one of the most serious threats to public health. Numerous factors contribute to this increasing epidemic, including reduced levels of physical activity for many children, shifting sociological elements that impact family eating patterns and the increased availability of convenience foods with little nutritional value (Institute of Medicine, 2005; Krishnamoorthy, Hart, & Jellalian, 2006). This study addresses yet another distinct factor that contributes to childhood obesity: television advertising that promotes low-nutrient, high-calorie food products to children.

Existing research shows that children's exposure to television advertising for non-nutritious food products is a significant risk factor contributing to childhood obesity (Institute of Medicine, 2006; Kaiser Family Foundation, 2004; Vandewater & Cummings, 2008). In the most comprehensive review of research to date, the Institute of Medicine of the National Academies concluded that television commercials significantly influence children's food preferences, purchase requests and dietary intake. The fact that younger children do not comprehend the persuasive intent of advertising messages (Calvert, 2008; Gunter, Oates, & Blades, 2005; Kunkel et al., 2004) and televised food advertising has long been dominated by low-nutrient, high-calorie products (Larson & Story, 2008; Palmer & Carpenter, 2006) exacerbates concern in this area. The IOM (2006) report summed up the situation, observing that "food and beverage marketing practices geared to children and youth are out of balance with healthful diets, and contribute to an environment that puts their health at risk" (p. 374). A comparable conclusion was reached in an earlier review of research conducted for a similar United Kingdom government inquiry (Hastings et al., 2003).

Prodded by this scientific evidence, policymakers have devoted increasing attention to the issue of food marketing to children as they seek to address the growing epidemic of childhood obesity. The Federal Trade Commission (FTC) has conducted hearings and issued reports on the topic (FTC, 2008; Holt, Ippolito, Desrochers, & Kelley, 2007); the Federal Communications Commission (FCC) hosted an

inter-governmental Task Force on Media and Childhood Obesity (FCC, 2009); and individual members of Congress have issued statements reflecting their concern (Harkin, 2007; Markey, 2007). Indeed, concern about the topic is not limited to the U.S. For example, the United Kingdom recently adopted strict governmental regulation that prohibits the advertising of non-nutritious, or so-called "junk food," products during programs that attract significant audiences of children (Hawkes, 2007).

In an effort to respond to public concern about the nutritional quality of the foods marketed to children, a coalition of major food companies has collaborated with the Council of Better Business Bureaus to establish an industry self-regulatory framework designed to improve the nutritional quality of foods advertised to children (Council of Better Business Bureaus, 2007). This effort has been termed the Children's Food and Beverage Advertising Initiative. Among the companies participating in the initiative are many of the nation's largest food conglomerates. At the time this study began in early 2009, a total of 15 companies participated in the initiative. These include:

- Burger King Corporation
- Cadbury Adams USA
- Campbell Soup Company
- Coca-Cola Company
- ConAgra Foods, Inc.
- The Dannon Company
- General Mills, Inc.
- Hershey Company
- Kellogg Company
- Kraft Foods, Inc.
- Mars, Inc.
- McDonald's USA
- Nestle USA
- PepsiCo, Inc.
- Unilever United States

As part of the industry initiative, each company has issued a detailed pledge of its commitment to limit its marketing efforts targeted at children to healthier food products, or in some cases, messages that promote healthy lifestyles. It is important to note, however, that each company defines what constitutes a "healthier" food product based on differing nutritional criteria. Participating companies have also pledged to restrict the use of licensed characters solely to advertising for foods that meet their specific nutritional standards for

healthier products, or in some cases, products that are generically considered to be “healthy dietary choices.” Proponents of industry self-regulation assert that this initiative should resolve the concerns that have been raised and neutralize any need for direct governmental regulation of food marketing to children. Indeed, the initiative asserts it will “change the mix of food and beverage products advertised to children to encourage healthier dietary choices and healthy lifestyles” in an effort to “change the landscape of child-directed advertising” (Peeler, Kolish, & Enright, 2009, p. 1).

This study provides an independent evaluation of how well industry self-regulation has accomplished these goals. While some of the participating companies’ pledges were announced as early as the summer of 2007, others have been added more recently, and some aspects of the pledges did not become fully operational until January 1, 2009. Clearly, then, 2009 provides the first opportunity to evaluate the efficacy and impact of the Children’s Food and Beverage Advertising Initiative on the overall marketplace of advertising to children.

To pursue this research agenda, there are two key focuses that we scrutinize. The first is the issue of whether or not each company has succeeded in fulfilling all of the elements of its pledge. Evaluating this issue requires careful measurement, given the lack of a uniform nutrition standard for defining healthy foods across the various companies. To accomplish this, products advertised in commercials targeted at children are linked to their parent corporation and then assessed for conformity with the applicable nutritional standards specified by that company.

A second and arguably more critical issue to be examined is the impact of the industry initiative on the overall environment of food advertising to children. Not all food companies participate in the program, which means the efforts of the initiative could be diluted by advertising for less healthy foods that originates from other sources (i.e., companies not participating in the initiative). Indeed, it is important to assess the proportion of the total volume of food marketing to children that is provided by companies participating in the initiative, in order to help evaluate its reach and impact.

Given the varying nature of the definitions of “healthier” food products that have been established by the participating companies, it is also critical to independently evaluate the nutritional quality of the overall marketplace of food advertising directed at children and to compare the patterns that are observed once the initiative is in effect with the levels that existed in the past. This is particularly important because of the inclusion of foods defined by industry as “better-for-you” as part of the self-regulatory marketing reforms. It remains to be seen whether foods defined as “better-for-you” (e.g., reduced fat Oreos) are indeed “good-for-you” (i.e., healthful), which is the requisite goal of advertising reform that seeks to reduce childhood obesity.

This study engages both of these critical tasks. It examines a broad base of advertising contained in a sample of more than 100 children’s television programs monitored between February and April of 2009. In the first part of the report, we present detailed information about the nature and extent of food marketing messages targeted at child audiences. In the second portion of the report, we provide an evaluation of the effectiveness of the industry’s effort at self-regulation, known as the Children’s Food and Beverage Advertising Initiative. In examining both of these areas, we benefit from previous studies of food marketing to children that we conducted in 2005 and 2007 (Kunkel, McKinley, & Stitt, 2008; Stitt & Kunkel, 2008). These previous studies employ sampling strategies and measures identical to the research reported here and, thus, provide optimal comparison points for tracking change over time in the food industry’s advertising practices targeted at children.

Finally, we note that this study employs a unique measurement strategy for evaluating the nutritional quality of advertised foods. Many previous studies that have assessed food advertising to children either measure product type without any evaluation of nutritional quality or, alternatively, report such detailed nutritional information that it is difficult to draw practical conclusions from the evidence. The former approach is clearly inadequate, particularly as marketers’ practices evolve toward healthier foods. While one might reasonably have surmised that a “fast food” ad represented a non-nutritious product in the distant past,

today such an ad might just as well present a fruit plate as opposed to a burger and fries. This underscores that the nutritional quality of the foods advertised in each commercial must be carefully measured, rather than inferred. We (Kunkel, McKinley, & Stitt, 2008; Stitt & Kunkel, 2008) successfully employed a measurement framework based on a consumer-oriented nutritional scheme devised by the Department of Health and Human Services as part of the agency's We Can! (Ways to Enhance Children's Activity and Nutrition) public information initiative. These measures simplify the evaluation of nutritional quality, while maintaining strong rigor and credibility, thus maximizing the value of the evidence produced by the study.

To summarize, this study provides a detailed examination of the overall landscape of televised food advertising to children. It identifies changes that have occurred between 2005 and 2009. The study also assesses compliance with the industry self-regulatory program known as the Children's Food and Beverage Advertising Initiative. Finally, the study analyzes the impact of industry self-regulation on the nutritional quality of foods advertised in the overall children's television marketplace.

In the next section, we detail the methodology used to conduct the research.

Method

Sample

This study examines food advertising in a broad base of children's programming on broadcast and cable television channels. The sampling design involves the creation of two composite days (one weekday, one Saturday) for each network included in the study. Composite days are created by videotaping programming at randomly selected times over a period of several months, until an entire day's schedule has been recreated through the composite collection process for each network. The sampling strategy captures one episode of all children's programming that regularly airs between 7:00 a.m. and 10:00 p.m. on each of the targeted channels. (See Stitt & Kunkel, 2008 for more detailed information regarding the procedure for creating composite days, as well as an explanation of how the technique

maximizes the representativeness of content-based findings.)

Children's programs were defined as any show with a V-chip rating of TV-Y (appropriate for all children) or TV-Y7 (appropriate for children, ages 7 and above), or any show with an FCC rating of E/I (educational/informational for children) that claims to target children under 12 years of age. The audiences for all of the programs sampled are consistently predominated by children younger than 12.

The channels examined in the study include all five national broadcast networks that deliver children's programming: ABC, CBS, Fox, NBC and CW. In addition, two national cable networks that are among the largest providers of children's programming are also included: Cartoon Network and Nickelodeon. The Disney Channel was omitted because the network does not present "outside" (i.e., non-Disney-based) advertising and, thus, food marketing would likely be minimal, if not absent entirely.

The 2009 sample included a total of 139 shows, representing 70.5 hours of children's programming across the seven networks included in the study. The programs were recorded between February 1, 2009, and April 15, 2009. Where applicable, we compared our 2009 data with findings from our previous studies, using identical methods and measures to analyze food advertising (Kunkel, McKinley, & Stitt, 2008; Stitt & Kunkel, 2008).

Measures

All non-program content (see Condry, Bence, & Scheibe, 1988) that appeared during each children's program was categorized by SEGMENT TYPE (i.e., commercial, program promotion, public service announcement) and measured for length of time. In order to provide context, descriptive information is provided at the outset of the Results section regarding the overall amount of time devoted to advertising. All other data reported in the study, however, are derived solely from the examination of commercials devoted to food products.

Each food commercial was first categorized by PRODUCT TYPE. Categories were constructed in an effort to discriminate more healthy from less healthy foods, while at the same time facilitating comparisons with previous research to

the greatest extent possible. When necessary, information required to properly classify advertised products was obtained by consulting ingredient labels on products and/or company websites. Categories included: sugared snacks, salted snacks, sugared beverages, sugared cereals, pastries/waffles, pasta, fast food/restaurants, dairy, fruits/vegetables/100% fruit juice, and prepackaged lunches, among others. Applicable products were considered 'sugared snacks' or 'sugared cereals' if sugar was one of the first three ingredients listed. Drinks were considered 'sugared beverages' if they included any added sugar.

In addition to classifying food commercials descriptively by product type, each advertisement was also categorized according to its fit with an evaluative food rating scheme devised by the U.S. Department of Health and Human Services (2005). The department employs the We Can! campaign to help parents select a healthy diet for their children (www.nhlbi.nih.gov/health/public/heart/obesity/wecan/index.htm), of which the centerpiece is a food rating system that differentiates products in three categories: Go, Slow and Whoa. Go foods are products rich in nutrients and relatively low in calories. They are low in fat and added sugar and, therefore, can be consumed "almost anytime" (U.S. Department of Health and Human Services, 2005, p. 14). Examples include vegetables, fruits, whole grain breads or breakfast cereals, fig bars, low-fat yogurt, nonfat milk and diet soda. Slow foods are higher in fat, added sugar and calories than Go foods, and should be consumed "sometimes, at most several times a week" (U.S. Department of Health and Human Services, 2005, p. 14). Examples include broiled hamburgers, nuts or peanut butter, waffles, most pastas, 100% juice, sports drinks and 2% low-fat milk. Whoa foods are high in calories and low in nutrients. They are highest in fat and added sugar, and should be consumed "only once in awhile or on special occasions" and then only in small portions (U.S. Department of Health and Human Services, 2005, p. 14). Examples include french fries, fried chicken or hamburgers, cookies, cakes, pies, ice cream, candy, whole milk and regular soda.

Food commercials were also evaluated for the advertisement's PRIMARY THEME/APPEAL. This measure has been used consistently in content

analyses of advertising to children (Barcus, 1977; Kunkel & Gantz, 1992) to represent the predominant promotional strategy embodied in the segment overall and is judged as a mutually exclusive variable for each commercial. Categories included: fun/happiness, taste/flavor/smell, premium, unique product, popularity of product and healthy product, among others. Ads are placed in a given category if they associate the product with the applicable theme. For example, a McDonald's ad in which Ronald McDonald jumps in a swimming pool and is shown exercising vigorously while playing various water activities with lots of children would be classified as a physical activity theme. An example of a fun/happiness appeal is an ad where children are shown going on a scavenger hunt to find the cereal they love, with the ad ending with the children shown as satisfied upon finding and eating the cereal. An example of a popularity theme/appeal is an ad where a parent is shown serving the product to a crowd of neighborhood children, all of whom are clamoring loudly for the food item.

Other tactics used by food marketers to increase the attractiveness of their products to children were measured, including the use of SPOKES-CHARACTERS (characters associated solely with the product, e.g., Cap'n Crunch, Ronald McDonald); LICENSED CHARACTERS/CELEBRITY PRODUCT ENDORSERS (characters whose popularity is not originally associated with a food product, e.g., Spongebob Squarepants, Dora the Explorer); CONTESTS; and WEBSITE PROMOTIONS (ad identifies a website address for children to visit that is sponsored by the food company, e.g., www.postopia.com).

To evaluate compliance with self-regulatory pledges regarding the nutritional standards and use of licensed characters, we compared all products observed in advertising monitored by the study to the nutritional guidelines specified by the relevant parent company, as part of the Children's Food and Beverage Advertising Initiative. To confirm product ingredients, we relied on information included in the labeling for each product.

Coding and Reliability

The classification of data for the study was accomplished by a group of seven coders. All coders were trained over an eight-week period and practiced extensively in order to achieve

acceptable levels of inter-coder reliability before beginning the process of generating data for the study. Reliability was assessed at the end of training and roughly once per week during the two-month period required to complete all classification of data. All advertising contained in a total of 10 randomly selected half-hour programs was evaluated by all coders and compared using Scott's pi to determine reliability coefficients. The programs contained 48 food commercials. All variables examined in the study achieved a level of reliability of .90 or above with the exception of primary theme/appeal, which yielded agreement at .76. This judgment is inherently more subjective and interpretive, and, thus, findings involving this attribute should be viewed with caution. Notwithstanding this one variable, all measures in the study proved highly reliable and, thus, the data can be viewed with confidence.

Results

This report of findings addresses two distinct topic areas investigated by our research. The first provides descriptive information analyzing the amount and type of food advertising delivered during children's television programming. Where possible, we compare the findings from our current data gathered in 2009 with patterns observed in our previous studies in 2005 and 2007. This first section also includes an overall assessment of the nutritional quality of the foods marketed on television to children. These data provide a clear picture of the environment of food advertising on television to children and how it has changed over the past four years.

The second aspect of our findings (below) evaluates the efficacy of the Children's Food and Beverage Advertising Initiative that was implemented in July 2007. As of early 2009, a total of 15 companies participated in the initiative, each one offering a unique commitment to improve their marketing activities targeted at children. Our evaluation employs two complementary perspectives. The first assesses whether or not the companies complied with their pledges, issued under the initiative program. The second, and arguably more critical analysis, examines the extent to which the industry's self-regulatory program

has successfully transformed the landscape of food marketing to children, from an emphasis on low-nutrient, high-density food products to an emphasis on healthier foods and beverages.

Amount and Type of Televised Food Marketing to Children

The findings reported in this section are organized according to key research questions addressed by the study.

QUESTION: How much food advertising is presented during children's programming?

Across the entire sample of 139 programs monitored in 2009, a total of 1,819 commercials were observed. Of these, 534 (29.5%) were food and beverage ads. This total base of advertising for food products is the foundation of all evidence presented in this report.

Table 1 compares the amount of food advertising on broadcast and cable television. Broadcast carried slightly higher levels of food advertising (8.8 ads/hour) than cable (7.2 ads/hour) in 2009. Across both media, children's programming presented an average of 7.6 food ads per hour.

The rate at which food ads appear during children's shows has declined over the four-year span of this study (see Table 2). Our previous research found an average of 10.9 food ads per hour appeared in 2005, and 8.5 per hour in 2007, compared to our current finding of 7.6 per hour in 2009. Across the 2005-09 study period, the overall number of commercial messages included in children's programming has remained relatively stable, with means ranging from 23.3 to 25.8 total ads per hour. Thus, food ads represent a smaller proportion of the overall children's advertising environment today than in the past, and young viewers are likely to encounter fewer food ads while watching children's programs in 2009 as compared to recent years. This shift is consistent with the widespread pattern of incremental reductions in traditional measured-media advertising practices by most marketers as they implement a corresponding increase in online and other digital media promotional efforts (Chester, 2008). Despite this

TABLE 1

Comparison of Time Devoted to Food Ads and Non-Food Ads

	Food Ads (N=534)		Non-Food Ads (N=1285)		Total Ads (N=1819)	
	N per hour	Minutes per hour	N per hour	Minutes per hour	N per hour	Minutes per hour
Broadcast	8.8	3:06	15.3	6:45	24.1	9:52
Cable	7.2	2:39	19	8:06	26.3	10:45
Overall	7.6	2:44	18.2	7:49	25.8	10:34

TABLE 2

Differences in Time Devoted To Food and Non-Food Ads Across Channel Type, 2005-2009

	Broadcast			Cable			Overall		
	2005	2007	2009	2005	2007	2009	2005	2007	2009
Food ads per hour	12.7 ^a	8.2 ^b	8.8 ^b	9.9 ^a	8.6 ^a	7.2 ^b	10.9 ^a	8.5 ^b	7.6 ^b
Minutes per hour devoted to food ads	5:14 ^a	3:16 ^b	3:06 ^b	3:52 ^a	3:33 ^a	2:39 ^b	4:22 ^a	3:29 ^b	2:44 ^c
Non-food ads per hour	11.5 ^a	13.3 ^{a,b}	15.3 ^b	13.4 ^a	15.2 ^a	19.0 ^b	12.8 ^a	14.7 ^b	18.2 ^c
Minutes per hour devoted to non-food ads	4:30 ^a	5:32 ^{a,b}	6:45 ^b	5:59 ^a	6:11 ^a	8:06 ^b	5:29 ^a	6:02 ^a	7:49 ^b
All ads per hour	24.2 ^a	21.5 ^a	24.1 ^a	23.2 ^a	23.8 ^a	26.3 ^b	23.7 ^a	23.3 ^a	25.8 ^b
Minutes per hour devoted to all ads	9:45 ^a	8:49 ^a	9:52 ^a	9:52 ^a	9:45 ^a	10:45 ^b	9:51 ^a	9:32 ^a	10:34 ^b

Findings with different subscripts are significantly different at $p < .05$.

shift, however, food commercials remain a significant presence on television and are still one of the most heavily advertised product types on that medium.

QUESTION: What types of food products are advertised to children?

A small number of popular categories accounts for the large majority of food advertising to children. In 2009, commercials promoting sugared cereals, fast foods/restaurants and sugared snacks comprised over 70% of all food advertising during children's shows (see Table 3). This pattern has held relatively stable over the past several decades (Kunkel & McIlrath, 2003; Palmer & Carpenter, 2006). Consistent with this pattern, these same categories accounted for 67.6% of all food commercials in our 2005 study.

A noticeable shift from 2005 to 2009 is that fast foods/restaurants have increased their share from 20.8% to 35.5% of the total volume

of food advertising during children's programming. In contrast, the proportion of ads devoted to sugared snacks declined from 20.8% to 10.1% during that same period. Most other aspects of the product profiles advertised to children remained relatively stable, just as they have in the past. Of particular note, the category of fruits/vegetables/100% juice remained almost invisible, accounting for 0.4% of all advertising during children's programs in 2009, as compared to 0.7% in 2005.

QUESTION: What types of persuasive tactics are used to promote food products to children?

Each food commercial observed during the study was judged for its primary theme or appeal. Associating fun/happiness with the advertised product was the most common tactic used in advertising to children, accounting for 30.7% of all ads (see Table 4). Nearly as common, taste/flavor/smell was the primary theme in 28.8% of all ads. The offering of a

TABLE 3

Distribution of Food Product Types Shown During Televised Food Advertising to Children

Product Type	Broadcast	Cable	Overall
Sugared cereals (N=138)	18.2%	28.4%	25.8%
Fast foods/restaurants (N=190)	40.9%	33.8%	35.5%
Sugared snacks (N=54)	12.1%	9.5%	10.1%
Sugared beverages (N=38)	12.9%	5.2%	7.1%
Pasta (N=34)	4.5%	7.0%	6.4%
Salted snacks (N=33)	5.3%	6.5%	6.2%
Dairy (N=30)	5.3%	5.7%	5.6%
Pre-packaged lunches (N=8)	0.8%	1.7%	1.5%
Easy to prepare meals (N=4)	-	1.0%	0.7%
Fruits/Veggies/100% Juice (N=2)	-	0.5%	0.4%
Other (N=3)	-	0.7%	0.6%
<i>Columns sum to 100%</i>	<i>N=132</i>	<i>N=402</i>	<i>N=534</i>

TABLE 4

Primary Themes and Appeals Used in Televised Food Advertising to Children

	Broadcast	Cable	Overall
Fun/happiness (N=164)	31.1%	30.6%	30.7%
Taste/flavor/smell (N=154)	34.1%	27.1%	28.8%
Premium (N=90)	10.6%	18.9%	16.9%
Popularity of product (N=34)	2.3%	7.7%	6.4%
Unique (N=22)	5.3%	3.7%	4.1%
Product performance (N=10)	3.8%	1.2%	1.9%
Physical strength (N=10)	3.8%	1.2%	1.9%
Economy/price (N=10)	3.0%	1.5%	1.9%
Quantity/size/amount (N=8)	0.0%	2.0%	1.5%
Social context (N=6)	0.8%	1.2%	1.1%
Convenience (N=5)	3.0%	0.2%	0.9%
Texture (N=4)	0.8%	0.7%	0.7%
Healthier food (N=1)	-	0.2%	0.2%
Other (N=16)	1.5%	3.5%	3.0%
<i>Columns sum to 100%</i>	<i>N=132</i>	<i>N=402</i>	<i>N=534</i>

premium in addition to the product (e.g., a toy included with purchase of a children's meal) was the principal message in 16.9% of all food ads. Collectively, these three tactics account for the primary persuasive appeal in roughly three-fourths (76.4%) of all food advertising to children.

Among the least common type of themes were ads devoted primarily to information about the food product. For example, 1.9% of all ads emphasized economy or price, while 1.5% of ads focused on quantity/size/amount of the product. Commercials that emphasize the advertised food is a healthy product are extremely rare, at 0.2% of all food ads. As with

the types of products advertised, the primary theme/appeal in children's advertising has also remained remarkably stable across past decades (Kunkel & Gantz, 1992; Kunkel & McIlrath, 2003), so it is not surprising our current data show little, if any, change from the long-standing trends that have emphasized fun/happiness over product information.

Table 5 reveals that certain types of persuasive appeals are more closely associated with some products than others. For example, fun/happiness themes are used frequently in ads for salted snacks (66.7%) and fast foods/restaurants (48.4%). Fun/happiness themes are even more common in ads for children's easy-to-prepare meals (75%), though the small number of cases observed for this type of product (N=4) suggests some caution in interpreting this finding. The use of premiums is another tactic often employed to attract children to food products. Commercials for dairy products—primarily yogurt—were the most likely to use premiums as a persuasive tactic (56.7%), although fast foods/restaurants also used this technique as their primary appeal in more than a quarter of all ads (27.4%).

Table 6 assesses how frequently several other promotional tactics were employed within ads, including efforts to encourage children to visit food marketing company websites. While the overall volume of food advertising to children on television is down, as we reported above, other research has documented an increasing amount of online food marketing to children (Chester & Montgomery, 2007; Weber, Story, & Harnack, 2006). Thus, it is not surprising that more than half (57.1%) of all food ads airing during children's television programs in 2009 promote a food marketing company website (e.g., postopia.com, millsberry.com). In contrast, only 18.7% of all children's food advertising in 2005 promoted a company website, which means the rate of web site promos has more than tripled since 2005.

Food and beverage marketers also employ the use of product-based spokes-characters, as well as licensed characters, in their commercial messages. Research shows that licensed characters are particularly effective at influencing children because children trust the characters they are repeatedly exposed to in program content (Institute of Medicine, 2006). Indeed, the licensed characters chosen by advertisers

to promote food products are typically the most popular figures across the landscape of children's television.

Table 6 also indicates that product-based spokes-characters appear in roughly half (53.9%) of all food ads during children's programs. Spokes-characters are frequently used to promote sugared cereals, appearing in 68.1% of all such commercials, as well as fast foods/restaurants, appearing in 58.4% of their spots aired during children's programming. Licensed characters are used less often overall, but are still a regular presence, appearing in 15.7% of children's food ads.

Important policy questions have been raised regarding the use of licensed characters within commercials that promote non-nutritious food products to children. This issue will be addressed in the next section of this report.

QUESTION: What is the nutritional quality of the foods advertised during children's television programming?

As noted earlier, many previous studies of food marketing to children have limited their analysis to descriptive statistics regarding the frequency with which various product types (e.g., sugared cereals, salted snacks) are advertised. Unfortunately, this approach requires that inferences be drawn about the nutritional value of various food product categories. In some cases, such as ads for sugared cereals, these inferences may well be sound; but in others, such as tallying ads for fast foods/restaurants, a problem can occur, because a commercial could be devoted either to a fruit salad offering or a hamburger and fries meal. Each of these cases would clearly hold different implications for evaluating the nutritional quality of the foods advertised to children, yet both would simply be classified as a fast food/restaurant ad if measurement was limited strictly to product type.

One of the strengths of this study is its independent analysis of the nutritional quality of each food product presented in all commercials shown during children's television programming. To accomplish this analysis, we employ the U.S. Department of Health and Human Services' Go, Slow, Whoa food rating framework. Figure 1 demonstrates two clear trends in our findings regarding the nutritional

TABLE 5

Primary Themes and Appeals Used in Televised Food Advertising to Children, by Product Type

Product Type	Primary theme/appeal				
	Fun/happiness	Taste/flavor/smell	Premium	Popularity of product	Healthy product
Sugared cereals (N=138)	16.7%	44.2%	7.2%	13.8%	-
Fast foods/restaurants (N=190)	48.4%	7.4%	27.4%	1.0%	-
Sugared snacks (N=54)	7.4%	46.3%	18.5%	5.6%	-
Sugared beverages (N=38)	34.2%	39.5%	-	15.8%	-
Pasta (N=34)	5.9%	70.6%	2.9%	8.8%	-
Salted snacks (N=33)	66.7%	18.2%	-	3.0%	-
Dairy (N=30)	10.0%	-	56.7%	-	-
Pre-packaged lunches (N=8)	12.5%	87.5%	-	-	-
Easy to prepare meals (N=4)	75.0%	-	-	-	-
Fruits/Veggies/100% Juice (N=2)	50.0%	-	-	-	50.0%
Other (N=3)	-	66.7%	-	-	-
Overall (N=534)	30.7%	28.8%	16.9%	6.4%	0.2%

TABLE 6

Frequency of Selected Advertising Tactics, by Product Type

Product Type	Contests	Website Promotion	Product-based spokes-character	Licensed character
Sugared cereals (N=138)	2.9%	30.4%	68.1%	18.1%
Fast foods/restaurants (N=190)	-	55.3%	58.4%	23.2%
Sugared snacks (N=54)	18.5%	88.9%	18.5%	5.6%
Sugared drinks (N=38)	-	97.4%	2.6%	-
Pasta (N=34)	32.4%	91.2%	82.4%	-
Salted snacks (N=33)	-	36.4%	33.3%	-
Dairy (N=30)	56.7%	60.0%	66.7%	30.0%
Pre-packaged lunches (N=8)	-	100%	100%	12.5%
Easy to prepare meals (N=4)	50.0%	50.0%	100%	50.0%
Fruits/Veggies/100% Juice (N=2)	78.9%	100%	-	-
Other (N=3)	-	-	33.3%	-
Overall (N=534)	8.2%	57.1%	53.9%	15.7%

quality of the foods advertised to children in 2009.

First, the large majority of foods advertised to children in 2009 are nutritionally deficient

products that should be avoided in a child's regular diet. Nearly three-fourths (72.5%) of all food ads presented during children's programs promote Whoa products. Moderately healthy Slow products comprise roughly one-

TABLE 7
Average Number of Food Ads Per Hour by Nutritional Quality Categories

Product Type	Nutritional Quality Category		
	Whoa (N=387)	Slow (N=142)	Go (N=5)
Sugared cereals (N=138)	1.96	-	-
Fast foods/restaurants (N=190)	1.16	1.52	0.01
Sugared snacks (N=54)	0.77	-	-
Sugared beverages (N=38)	0.52	0.01	-
Pasta (N=34)	0.21	0.27	-
Salted snacks (N=33)	0.31	0.16	-
Dairy (N=30)	0.37	0.04	0.01
Pre-packaged lunches (N=8)	0.11	-	-
Easy to prepare meals (N=4)	0.06	-	-
Fruits (N=2)	-	-	0.03
Other (N=3)	0.01	0.01	0.01
Overall (N = 534)	5.52	2.01	0.09

fourth (26.6%) of the total volume of food ads, while genuinely healthy Go food products are almost never advertised on television to children. They represent less than 1% (0.9%) of the 534 total food ads identified in the study.

The second important trend illustrated by Figure 1 is strong consistency in the nutritional quality of foods marketed to children across the two platforms of broadcast and cable television. That is, the nutritional quality of foods advertised does not vary during children's programming, regardless of whether one watches broadcast or cable channels. In either case, children will see an equivalent preponderance of nutritionally poor foods during the commercial interruptions.

Another perspective on the nutritional quality of foods marketed to children can be gained by evaluating the ad content contained in an average hour of programs. This perspective is presented in Table 7, which analyzes the average number of food ads shown per hour, with breakdowns for each of the three categories in the U.S. Department of Health and Human Services' Go, Slow, Whoa framework.

As reported above, our 2009 data show that youngsters will see an average of 7.6 food ads for every hour they spend watching children's programming (see Table 7). This overall total

breaks down as follows. Viewers will see 5.5 ads per hour for Whoa products and 2.0 ads per hour for moderately healthy Slow products. The frequency with which genuinely healthy food ads appear is so low, however, that a child would need to watch more than 10 hours of children's programs before he or she would encounter just one commercial for a Go product (see Figure 2). During the 10 hours of viewing that would be required to encounter just one healthy food ad, a child meanwhile would be exposed to a total of 55 ads for Whoa products and 20 ads for Slow products.

Summary of Key Findings

To review, this section of our report identifies two critical findings. First, food advertising to children on television has declined in volume between 2005 and 2009. The average number of food ads appearing during children's programming has dropped from 10.9 per hour in 2005 to 7.6 per hour in 2009. While most other patterns in televised food marketing to children have remained stable, this finding reflects a 30% reduction in the amount of food advertising presented during children's programming. Although that reduction is meaningful, it is important to recognize that food and beverage marketing retains a significant presence in the children's television environment, and young viewers will still be see thousands of food

FIGURE 1
Nutritional Quality of Products Shown in Food Ads, by Channel Type

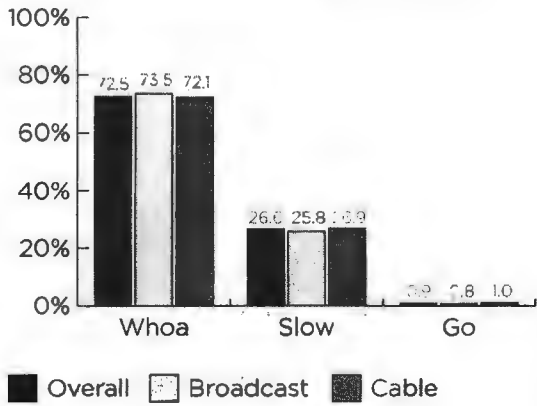


FIGURE 2
Nutritional Quality of Food Ads In 10 Hours of Children's Programs

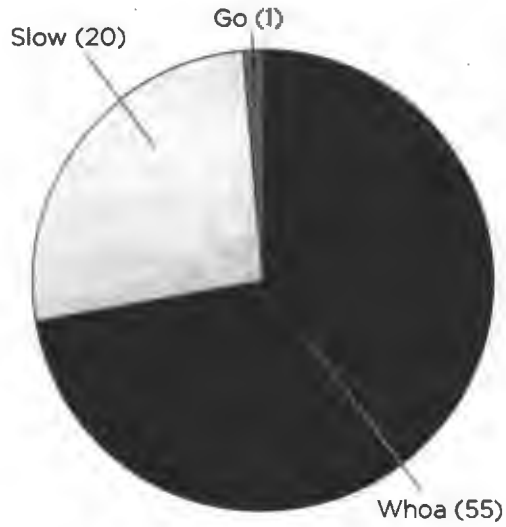
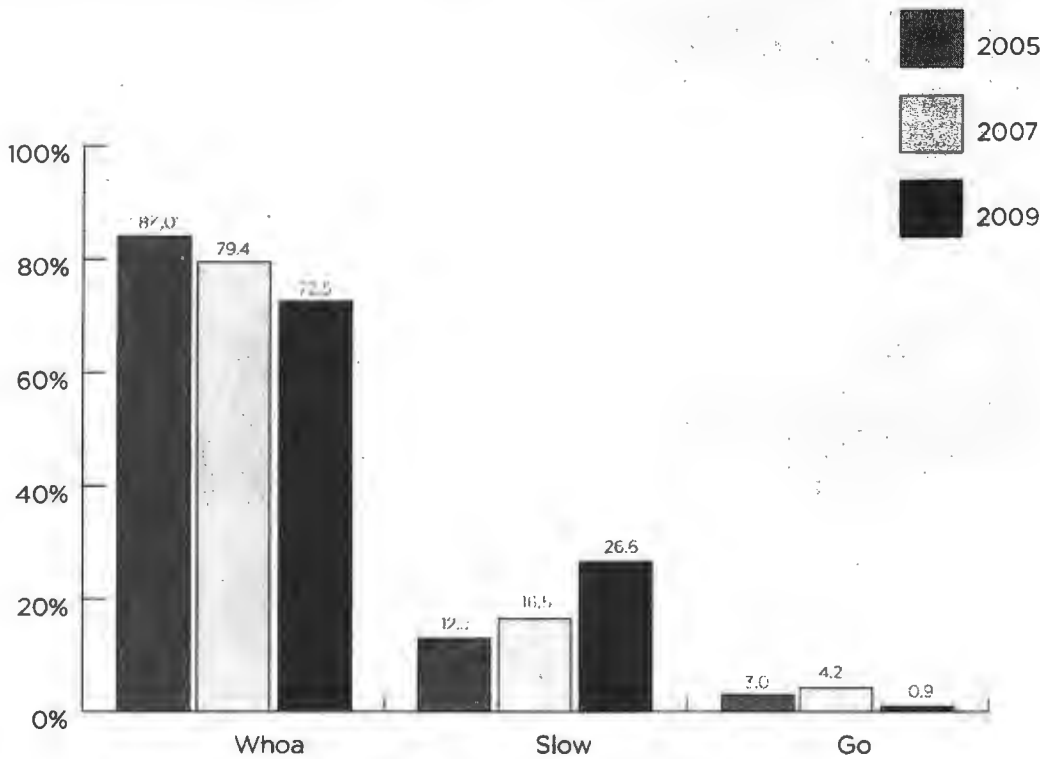


FIGURE 3
Over Time Comparisons of Nutritional Quality in Food Ads for Industry, Overall



ads each year during childhood, even at this reduced rate of exposure. Thus, the nutritional content of the foods advertised to children remains critically important, which leads to our second key conclusion.

The nutritional quality of foods marketed to children remains heavily skewed toward non-nutritious products that should not be part of a child's regular diet. That is, nearly three of every four food ads aired during children's television programs (72.5%) promote a Whoa product. Whoa products are high in calories and low in nutrients, and should be consumed "only once in awhile or on special occasions," according to the U.S. Department of Health and Human Services (2005, p. 14). The extent to which unhealthy foods predominate over healthier fare has declined somewhat since 2005, when Whoa products accounted for 84.0% of all food ads targeted to children (see Figure 3). Nonetheless, our data demonstrate that nutritionally poor food products continue their strong predominance in the children's advertising environment. Of arguably equal importance, ads for truly healthy foods, classified as Go products under the U.S. Department of Health and Human Services' scheme remain virtually invisible. Indeed, less than one of every 100 (0.9%) food ads aired on children's shows promotes a healthy product that children can eat safely on a daily basis.

Based on these findings, it is clear that, as of 2009, the food marketing industry has failed to meet the recommendation of the Institute of Medicine (2006) of the National Academies to voluntarily shift the longstanding emphasis in children's food marketing away from low-nutrient, high-density foods to a clear reliance on healthy food options. Moreover, it is worth noting that the Institute of Medicine issued a contingency recommendation if voluntary industry efforts were not successful in reversing the existing pattern. Specifically:

If voluntary efforts related to advertising during children's television programming are unsuccessful in shifting the emphasis away from high-calorie and low-nutrient foods and beverages to the advertising of healthful foods and beverages, Congress should enact legislation mandating the shift on both broadcast and cable television. (IOM, 2006, pp. 14-15)

Before drawing final conclusions from our research, it is important to consider the following section of this report, which provides a detailed evaluation of the efficacy of all key dimensions of the Children's Food and Beverage Advertising Initiative. Nonetheless, the findings from this first section of our study present the fundamental evidence for measuring achievement of the IOM's industry-wide goals.

While self-regulatory efforts have clearly accomplished slight change in the desired direction, reducing the prevalence of nutritionally poor food advertising from 84% to 72.5% of advertising during children's programs between 2005 and 2009, the reform accomplished to date falls far below the stated goal. In addition, the change observed is occurring at a pace that does not reflect the urgency of the public health crisis the nation faces involving childhood obesity.

Consider the following extrapolation. With the measuring stick for nutritionally poor food ads starting at 84.0% in 2005, and a demonstrated rate of change that has reduced this level roughly 12% over four years' time, one can project future expectations. At the current pace, it would take approximately eight more years, or until 2017, to reach the tipping point where the proportion of children's food advertising devoted to nutritionally poor products would first drop below the 50% level. Yet this calculation represents only half of the basic goal structure.

In addition to halting the predominance of nutritionally poor food products in TV advertising to children, the Institute of Medicine also implored the food and beverage industry to exercise its marketing muscle to promote genuinely healthy food options. In this regard, our data show that literally no progress has yet been achieved. Whereas 3% of all televised food marketing to children in 2005 were for genuinely healthy Go products, that level has fallen slightly to 0.9% in 2009. In other words, to the extent that industry advertising efforts have modestly reduced promotion of the worst possible food products to children, they have so far replaced those undesirable options with only slightly improved food offerings that are still of limited nutritional value in a child's daily diet.

Evaluation of Industry Self-Regulation

The second principal aspect of the study involves examination of the Children's Food and Beverage Advertising Initiative. In this section of the report, we address two key focuses: (1) how well do companies that participate in the initiative fulfill their pledges, and (2) how has the initiative impacted the overall nutritional quality of foods marketed on television to children?

To qualify as a participant in the Children's Food and Beverage Advertising Initiative, companies must agree "to devote at least half of their advertising directed to children under 12 on TV, radio, print and the Internet to 'better-for-you' products and/or to messages that encourage good nutrition or healthy lifestyles" (Kolish & Peeler, 2008, p. 4). In addition, participants commit to "reduce the use of third-party licensed characters in advertising primarily directed to children under 12, unless such advertising is for 'better-for-you' foods or includes healthy lifestyle messaging" (Kolish & Peeler, 2008, p. 4). While the initiative also includes commitments to limit advertising in terms of product placement, interactive games and in elementary school environments, only the two prongs cited above are relevant to this study's examination of television advertising and, thus, are the focus of this evaluation.

In addition to subscribing to the core principles of the initiative, each participating company offers an individual pledge that specifies its own unique criteria for defining a healthy food product. There is no uniform nutrition standard applied across all companies involved in the initiative pledge program. Rather, each participant establishes a distinct pledge, indicating its commitment in terms of:

- (a) overall restrictions on food advertising to children, such as a promise not to advertise any products to child audiences;
- (b) standards that must be met regarding the nutritional quality of food products that will be advertised to children;
- (c) limits on the use of licensed characters in food advertising to children,

such as a promise to not use licensed characters at all, or to use them only in ads for products that meet certain nutritional standards.

Across the 15 companies studied, four of them (Cadbury Adams, Coca-Cola, Hershey's, Mars) have pledged not to advertise any of their products to audiences of children under 12. The remaining 11 companies have all devised independent criteria for defining a healthy food product (labeled "better-for-you" under the initiative program specifications), and all but one have pledged to limit their advertising to children exclusively to these products. Finally, the 11 participating companies that advertise to children all include in their pledges some type of commitment to limit the use of licensed characters to advertising that promotes healthy foods. The most common pledge is that licensed characters will be featured only in ads for products that meet a company's nutritional standards for healthy foods, although some participants offer a more vague commitment to limit licensed characters to advertising for "healthy dietary choices" (McDonald's) or that will "support sound food choices" (Campbell Soup).

As with the prior section, the findings to evaluate the industry's self-regulatory initiative (below) are organized according to key research questions addressed by the study.

QUESTION: Are companies that participate in the Children's Food and Beverage Advertising Initiative fulfilling their individual pledges regarding the nutritional quality of advertised foods?

As noted above, four of the participating companies have pledged not to advertise any food products to children. Across the entire sample for this study, spanning a total of 139 children's programs on broadcast and cable channels, no commercials from any of these companies were ever observed, and thus their portion of the pledge program was fulfilled. It is possible that this aspect of the initiative program contributes to the reduction observed in the overall amount of food advertising presented during children's programming in 2009, as compared to 2005.

Of the remaining 11 companies, our study observed advertising messages aired by eight of

them (Burger King, Campbell Soup, ConAgra Foods, Dannon, General Mills, Kellogg, Kraft Foods, and McDonald's USA) (see Table 8). No advertising was encountered for products marketed by Nestle, PepsiCo or Unilever across any of the 139 children's programs sampled for our research between February and April 2009.

Of 381 total ads from the eight companies participating in the initiative program, all complied with the unique criteria specified by the parent company's nutritional guidelines. That is, each ad featured a product that met all elements of the applicable company's nutrition standards, as specified in its individual pledge. Some ads were encountered that placed little, if any, emphasis on a specific food product. For example, a McDonald's ad that showed Ronald McDonald preparing for bedtime never mentioned a particular advertised product, though it included a one-second-long visual depiction of a pledge-compliant Happy Meal on his night stand in the background. While this commercial might be argued to promote McDonald's general brand and overall product portfolio, which includes many non-nutritious options, the study ultimately judged this ad and a handful of others like it to be devoted to a pledge-compliant food product, based on the brief visual presentation of a pledge product. Thus, such ads were not considered a violation.

In sum, our data make clear that all participants in the Children's Food and Beverage Advertising Initiative have complied with all aspects of their commitments regarding nutritional guidelines for the foods advertised to the child audience, as specified by each company. We demonstrate with additional data below, however, that this finding does not warrant the conclusion that the foods marketed to children by participating companies should necessarily be considered healthy.

QUESTION: How much of the televised food advertising targeted at children originates with companies that participate in the Children's Food and Beverage Advertising Initiative?

Of 534 total food ads identified in the study during 2009, 71.3% (N=381) came from companies participating in the industry self-regulatory program. The remaining 28.7% (N=153) were

for products from companies that are not participating in the pledge program. Thus, the current reach of industry self-regulation stands at a bit more than two-thirds of all commercials presented during children's programs.

Table 8 indicates that four companies predominate in the marketplace of children's food advertising. Kraft, McDonald's, General Mills, and Kellogg collectively account for 58.3% of food advertising observed overall and for 81.9% of all advertising from pledge companies.

The level of participation in the industry's self-regulatory initiative has grown since July 2007, when the program was initially unveiled with 11 participating companies. According to the Council of Better Business Bureaus, the parent organization that supervises the Children's Food and Beverage Advertising Initiative, the original 11 companies accounted for "at least two-thirds of the television advertising expenditures for food and beverage advertising to children in 2004" (Kolish & Peeler, 2008, p. 3). It is important to note, however, that the inclusion of four additional companies has not appreciably expanded the reach of food advertising to children that is subject to industry self-regulation, which stands at 71.3% in 2009.

This may be due, in part, to the fact that some companies participating in the pledge program have reduced and/or discontinued entirely their food marketing efforts targeted at children. For example, five companies (Cadbury Adams, Hershey, Nestle, PepsiCo and Unilever) that currently participate in the self-regulatory program collectively accounted for 15% of the total of 557 ads observed in our previous study in 2005. In contrast, no advertising by any of these companies was identified across 139 children's programs in 2009.

This creates an ironic situation. While it may be desirable from a public health perspective to see companies that offer low-nutrient, high-density food products voluntarily discontinue their advertising to children, this outcome may provide opportunity for other companies that do not adhere to industry self-regulation to enter the market and/or to increase their advertising efforts in order to gain competitive advantage over initiative participants. Should this be the case, the net impact of the industry self-regulatory initiative, in terms of significantly affecting the overall landscape of

TABLE 8

Distribution of Food Ads, by Pledge Company

Pledge Company	N of Ads	% of All Food Ads	% of Pledge Co. Food Ads
Kraft	89	16.7%	23.4%
McDonalds	85	15.9%	22.3%
General Mills	81	15.2%	21.2%
Kellogg's	57	10.5%	15.0%
Campbell's	19	3.6%	5.0%
Dannon	19	3.6%	5.0%
Con Agra	17	3.2%	4.5%
Burger King	14	2.6%	3.6%
Overall	381	71.3%	100%

1. Four participating companies (Cadbury Adams, Coca-Cola, Hershey's, Mars) pledged not to engage in child-directed food and beverage advertising.

2. No ads were observed for Nestle, Pepsi, or Unilever during the study period.

televised food advertising, could be severely compromised.

Regardless of any conjecture about future developments, our data indicate that more than one-quarter (28.7%) of all televised food marketing to children is not subject to any of the precautions or protections provided by the Children's Food and Beverage Advertising Initiative.

QUESTION: What is the nutritional quality of the foods marketed to children by companies that participate in the Children's Food and Beverage Advertising Initiative?

Despite the fact that all food advertising by industry self-regulatory participants complies with each company's nutritional pledge, our data indicate that two-thirds of all pledge company advertising to children is devoted to products of the poorest nutritional quality, according to the Go-Slow-Whoa food rating system. Specifically, 68.5% of all food ads aired by participating companies promote non-nutritious Whoa products, while 31.0% feature moderately healthy Slow products and only 0.5% are for truly healthy Go products. These data illustrate a fundamental disconnect between the way in which food products are defined as "healthy," according to the pledge criteria employed for the Children's Food and Beverage Advertising Initiative, and the way in which healthy nutritional quality is judged from an independent perspective.

As noted in a previous section, the fundamental policy goal advocated by the Institute of Medicine is to reverse the children's food advertising environment by "shifting the emphasis away from high-calorie and low-nutrient foods and beverages to the advertising of healthful foods and beverages" (IOM, 2006, pp. 14-15). To be clear, this recommendation does not seek to have the industry merely reduce the unhealthy ingredients in high-calorie, low-nutrient foods and beverages in a manner that renders them less unhealthy. Rather, the Institute of Medicine clearly articulates a goal that food marketers should shift their advertising to healthy foods and beverages—with "healthy" judged from an absolute, not a relative, perspective. Herein lies the disconnect between the aspirations of the industry's self-regulatory program and the public health goals currently sought to help defeat the epidemic of childhood obesity.

The near-term public health goal is to achieve a predominance of healthy foods in advertising to children, rather than the opposite pattern that has prevailed long into the past. Yet the near-term industry response, in the form of the Children's Food and Beverage Advertising Initiative, addresses the issue from a different angle. Most companies allow products to qualify as "healthier" under their nutritional standards simply as a function of altering the ingredients to modestly reduce health risk from heavy consumption. For example, an existing

FIGURE 4
Over Time Comparisons of Nutritional Quality in Food Ads for Pledge Companies

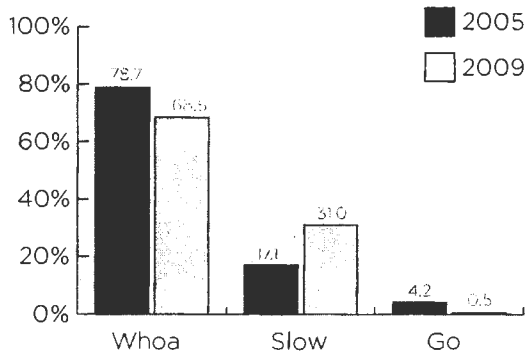


FIGURE 5
Over Time Comparisons of Nutritional Quality in Food Ads for Non-Pledge Companies

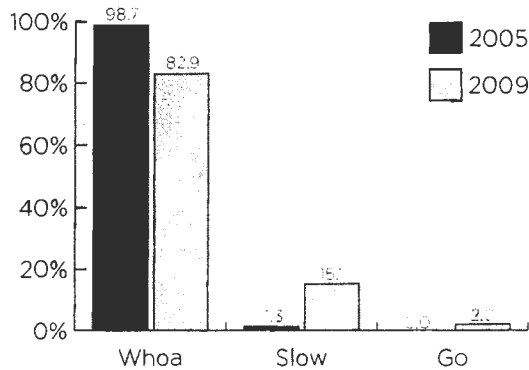
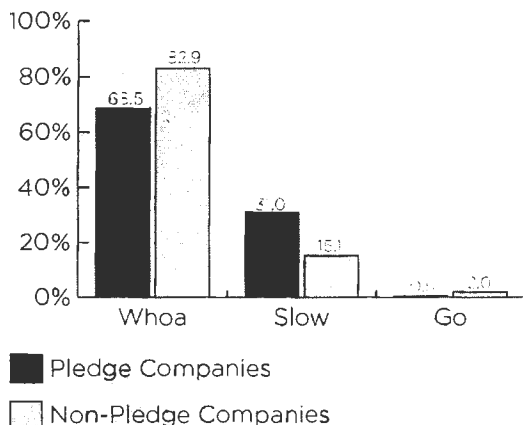


FIGURE 6
Comparisons of Nutritional Quality in Food Ads for Pledge and Non-Pledge Companies, 2009



product that has substantial added sugar, fat or salt can qualify for the “healthier” designation if part of the added ingredient is removed from the product recipe, despite the fact that the product still includes levels of added ingredients (i.e., sugar, fat, salt) considered to be excessive. Indeed, the food marketing industry has coined the term “better-for-you” specifically to describe such products in an effort to imply they represent a healthful food.

This study demonstrates that the majority of food products advertised to children that are classified as “better-for-you” are not really good-for-you, at least according to the U.S. Department of Health and Human Services’ consumer food rating scheme. When viewed from an absolute, rather than a relative perspective, the majority of foods that comply with the nutritional standards of the industry’s self-regulatory initiative are not considered healthful. Indeed, almost none (0.5%) are truly healthy Go products, while only about one-third (31.0%) are considered moderately healthy Slow products.

Figure 4 illustrates precisely what the industry’s self-regulatory initiative has achieved in terms of improving the overall nutritional quality of foods marketed to children. In 2005, the concern about food marketing to children had not fully surfaced as a critical public health issue, and no self-regulation could be seen on the horizon. In 2007, the initial pledges for the Children’s Food and Beverage Advertising Initiative were announced and became operational. Thus, by comparing the industry’s advertising practices in 2005 to those of 2009, it is possible to quantify the improvements accomplished by the Children’s Food and Beverage Advertising Initiative. Over a four-year span, the predominance of Whoa products diminished from an initial level of 78.7% of all ads from participating pledge companies to 68.5% in 2009. In complementary fashion, the share of pledge company advertising devoted to moderately healthy Slow products increased from 17.1% in 2005 to 31.0% in 2009, while advertising for Go products remained extremely low across both times of measurement.

This change is a positive one, and the industry deserves some credit for achieving it. That said, the degree of improvements accomplished in the overall nutritional quality of foods marketed to children clearly fall far short

TABLE 9

Distribution of Food Ads, by Non-Pledge Company

Non-Pledge Company	N of Ads	% of All Food Ads	% of Non-Pledge Co. Food Ads
Chuck E. Cheese's	66	12.4%	43.1%
Topps	29	5.3%	19.0%
Sunny Delight	14	2.6%	9.2%
Subway	14	2.6%	9.2%
Perfetti Van Melle	11	2.1%	7.2%
IHOP	9	1.7%	5.9%
Wrigley's	2	0.4%	1.3%
Yum!	2	0.4%	1.3%
Jack in the Box	2	0.4%	1.3%
Johnson & Johnson	2	0.4%	1.3%
Novartis	1	0.2%	0.6%
Mrs. Butterworth	1	0.2%	0.6%
Overall	153	28.7%	100%

of the objectives specified by the Institute of Medicine.

QUESTION: What is the nutritional quality of the foods marketed to children by companies that do not participate in the Children's Food and Beverage Advertising Initiative?

Another means of evaluating the benefit of industry self-regulation is to examine the advertising practices of those companies that do not participate in the initiative. As noted above, companies that do not participate in the pledge program accounted for 28.7% of all food advertising during children's programming. Chuck E. Cheese's is the most prominent non-participant, accounting for 12.4% of children's food advertising observed in the study (see Table 9). Another visible non-participant is Topps, makers of Ring Pop and Baby Bottle Pop candies, among others. This company accounts for 5.3% of all food ads observed in 2009. Our study also identified ads from 10 other national companies that are not included in the voluntary program.

Figure 5 presents the nutritional profile of the food products advertised by non-participating companies. In 2009, 82.9% of ads from non-pledge companies were for Whoa products. Of the remainder, 15.1% were for Slow products and 2.0% were Go products.

In terms of change over time, the nutritional quality of advertising by non-pledge companies improved from 2005 to 2009. The proportion of ads devoted to Whoa products declined from 98.7% in 2005 to 82.9% in 2009, while the frequency of moderately healthy Slow product advertising increased from 1.3% to 15.1% over the same period. Advertising for Go products remained extremely low across both times of measurement.

It is important to compare the advertising practices of companies that do and do not participate in the industry's program of self-regulation. That issue is addressed in the next section.

QUESTION: How does the nutritional quality of food marketed by non-pledge companies compare with the nutritional quality of foods marketed by pledge companies that participate in the Children's Food and Beverage Advertising Initiative?

Figure 6 demonstrates that non-pledge companies advertise nutritionally poor Whoa products at a much higher rate than companies that participate in the voluntary pledge program. Specifically, 82.9% of non-pledge company food advertising was for Whoa products in 2009, as compared to only 68.5%

for pledge companies. Conversely, pledge companies are twice as likely to advertise a moderately healthy Slow product to children (31% of all their food advertising) compared to non-pledge companies (15.1% of all their food advertising). The amount of advertising devoted to healthy Go products is so low overall as to render any comparison meaningless.

These data indicate that, from a comparative perspective, companies participating in the Children's Food and Beverage Advertising Initiative tend to devote more of their marketing efforts to foods of better nutritional quality than non-participating companies. Concomitantly, pledge participants devote less of their advertising to foods of the poorest nutritional quality, as compared to non-participants.

QUESTION: What proportion of foods marketed to children by pledge companies meet the best nutritional standards specified by all companies that participate in the Children's Food and Beverage Advertising Initiative?

A significant limitation of the self-regulatory program is the lack of any uniform nutritional standard for identifying food products that qualify as healthy and are, therefore, considered appropriate for advertising to audiences of young children. Indeed, it is puzzling that a food product classified as healthy by one company's standards can fall short of the nutritional guidelines of another because of the varying nutritional criteria employed across the full range of participating companies. In such a case, an identical product could be judged as either healthy and pledge-compliant or non-nutritious and a pledge violation, depending on its affiliation with one particular corporate parent, as compared to another.

Consider the following example: Cocoa Puffs cereal meets all the applicable criteria specified by its parent corporation, General Mills, to qualify as a healthy product. It contains no more than 175 calories per serving, no trans fats and no more than 12 grams of added sugar, among other criteria. If the same product was marketed by Kraft Foods, however, it would fall short of Kraft's guideline, which specifies that a healthy product contains no more than 25% of total calories from added sugar. This means that Cocoa Puffs is considered a healthy product according to nutritional guidelines for cereals specified by General Mills, but it would

not qualify as healthy according to the nutritional guidelines for cereals marketed by Kraft Foods.

This is not an isolated example. There are seven other General Mills' cereal products featured in ads and observed in this study that fit the same profile; they are judged as healthy by their parent company's set of standards but would not be classified as such by the nutritional guidelines of another participating company. Moreover, this example is not an indictment of lax nutritional standards on the part of General Mills. This pattern of inconsistency is pervasive, and examples of similar conflicts can be identified when comparing many products across differing pairs of company standards.

At its root, this situation suggests that each company tailors its unique nutritional guidelines to define healthy foods by carefully weighing the implications of each factor for its particular product portfolio. It implies that shades of grey in close call decision-making may be shaped at least in part by a company's self-interest in qualifying more of its products in the "healthy" category. As a result, it means that even though each participating company may fully comply with its pledge commitments, that does not necessarily mean all foods marketed to children that meet those company-based standards would actually qualify as healthy when judged from an independent, neutral perspective.

Many observers suggest the optimal approach to evaluate the nutritional quality of foods marketed to children would be to employ a uniform nutritional standard, whether or not that standard is applied by industry self-regulation or governmental regulatory policy (Miller, 2008). In an effort to apply a level-playing field test that fairly evaluates the nutritional quality of foods marketed to children by self-regulatory participants, we have devised a set of uniform nutritional standards based entirely on guidelines already implemented by one or more companies as part of the Children's Food and Beverage Advertising Initiative. We have devised a metric that we term an Optimal Composite Nutritional Standard (OCNS). The OCNS is specific to certain types of products, such as (a) children's meals and (b) breakfast cereals, which are the two examples we employ here.

To construct the OCNS for children's meals, we

consider the basic nutritional guidelines specified by the initiative, one criterion at a time: number of calories per serving, amount of calories from fat, amount of calories from saturated fat, amount of calories from added sugar and amount of sodium. For each criterion, we search through the entire range of standards indicated in the pledges across all participating companies and then identify the guideline that is the best, or “optimal,” from a child health perspective. For example, when devising the OCNS for children’s meal products, we note that Burger King defines a healthy meal as no more than 560 calories per serving; ConAgra (maker of Kid Cuisine) specifies no more than 500 calories; and both Kraft (maker of Lunchables) and McDonald’s stipulate no more than 600 calories per serving in order for a meal to qualify as healthy. Across this entire range, the optimal standard offered by a participating company is 500 calories per serving, so we select that as the applicable standard for the OCNS. The process is then repeated across all criteria to identify the optimal guidelines offered by participants in the self-regulatory program, ultimately yielding an Optimal Composite Nutritional Standard for children’s meal products.

Before proceeding further, we acknowledge that the product of this process might be criticized as insufficient in terms of promoting child health. While it is based on the best standards already endorsed and implemented by the industry, one could argue that even those standards might privilege corporate self-interest over children’s needs. Some nutritionists assert that all guidelines employed by the industry to identify “healthy” foods are inherently suspect and inadequate (Neuman, 2009), with more rigorous and independent criteria called for. Without necessarily defending the OCNS, we offer it as a vehicle to assess how well food marketing to children currently meets the best nutritional standards specified by companies participating in the Children’s Food and Beverage Advertising Initiative. We offer it to gain a complementary perspective that assesses the adequacy of the initiative at improving the nutritional quality of foods marketed to children in addition to our use of the U.S. Department of Health and Human Services’ Go-Slow-Whoa framework.

Table 10 reports the results from our application

of the OCNS to all ads aired by pledge companies for children’s meal products. As noted in a previous section, all ads from Children’s Food and Beverage Advertising Initiative participants complied with the applicable company-specific nutritional guidelines. Table 10, however, indicates that, across all commercials for meal products from participating companies, only 12% of products meet the OCNS criteria. Only meals marketed by Burger King comply with all applicable standards. In contrast, 88% of the meal products advertised by participating companies fall short on one or more of the uniform nutritional standards that comprise the OCNS, including all offerings from ConAgra, Kraft, and McDonald’s.

A second area to which we apply the Optimal Composite Nutritional Standard is breakfast cereals. A total of 20 different cereal products marketed by three participating companies appeared in 138 ads identified by the study. Table 11 reveals that only 8% of all the products featured in ads meet the OCNS criteria and, thus, would be classified as healthy foods. These include two well-known products, General Mills’ Cheerios and Kellogg’s Rice Krispies. In contrast, 92% of all cereals advertised by companies participating in self-regulation fall short on one or more of the uniform nutritional standards that comprise the OCNS.

To summarize, because the Children’s Food and Beverage Advertising Initiative lacks a uniform nutrition guideline, and, thus, the standards for defining a healthy food vary substantially from one company to another, this study compiled a list of the best nutritional standards employed by self-regulatory participants in two food product areas: children’s meals and breakfast cereals. This set of standards is termed the Optimal Composite Nutritional Standard. Our analysis revealed the overwhelming majority of advertising from companies participating in the initiative do not meet these best standards shared by their peers in the two product areas we examined. Specifically, 88% of all advertised products fell short in the area of children’s meals, while 92% failed the test among breakfast cereals.

Like our previous finding—that roughly three-fourths of all food advertising that fully complies with the pledges of self-regulatory participants is actually of poor nutritional category—the outcome here seems to further question the

TABLE 10

Pledge Company Compliance with Optimal Composite Nutritional Standard for Children's Meal Products

Parent Company	Product	% of all meals shown	< 500 Calories	< 30% Calories from fat	< 10% Calories from saturated fat	< 10% Calories from added sugar	< 600 mg Sodium
Burger King	Kids Meal: Macaroni & Cheese	11	+	+	+	+	+
	Meal 3: Burger Shots	1	+	+	+	+	+
Conagra	Kid Cuisine: All Star Chicken Nuggets	2	+	-	+	-	-
	Kid Cuisine: Magic Cheese Stuffed Crust Pizza	2	+	+	-	-	-
Kraft	Lunchables: Extra Cheesy Pizza	8	+	+	+	-	-
McDonalds	Happy Meal: Chicken McNuggets	57	+	+	+	-	-
	Happy Meal: Snack Wrap	19	+	+	+	-	-

legitimacy of the nutritional guidelines used for the Children's Food and Beverage Advertising Initiative. Simply put, most foods considered "healthier" by the nutritional guidelines established by one company would not qualify under the standards employed by one or more of their competitors. This lack of a level playing field means consumers may be confused or misled, while companies are allowed to define products as healthy when clearly they are not, as judged from an independent perspective.

QUESTION: Are companies that participate in the Children's Food and Beverage Advertising Initiative fulfilling their individual pledges regarding the use of licensed characters?

The predominant type of pledge offered in this area is that a company will use licensed characters only in advertising for products that meet its nutritional guidelines for defining a healthy food. Compliance for this commitment is essentially a given, based on the previous finding that all participants met their pledge to advertise only products that meet the standards specified by each company's nutritional

guidelines. To make sense of this, it is important to comprehend the nesting of these two types of pledges.

First, each company pledges to advertise only products that meet its nutritional guidelines. Then each company pledges to use licensed characters solely in advertising for foods that meet its nutritional guidelines. Since we have already confirmed that the first aspect of the pledges addressing the nutritional guidelines was properly fulfilled by all companies, it follows logically that the licensed character commitments must also be fulfilled. Our data corroborate this. When licensed characters are used by self-regulatory participants, the characters appear solely in ads that comply with the company's nutritional standards.

As was the case with the nutritional guidelines, however, evaluating compliance with the pledges alone does not tell the whole story. Consider the policy goals applicable in this area. In the Institute of Medicine (2006) report, *Food Marketing to Children*, it was recommended that licensed characters be used

TABLE 11

Pledge Company Compliance with Optimal Composite Nutritional Standard for Breakfast Cereals

Parent Company	Product	% of all cereals shown	< 170 Calories	< 30% Calories from fat	< 10% Calories from saturated fat	< 25% Calories from added sugar	< 230 mg Sodium
General Mills	Cookie Crisp	9	+	+	+	-	+
	Trix	8	+	+	+	-	+
	Lucky Charms	7	+	+	+	-	+
	Cinnamon Toast Crunch	7	+	+	+	-	+
	Cocoa Puffs	5	+	+	+	-	+
	Cheerios	4	+	+	+	+	+
	Reese's Puffs	3	+	+	+	-	+
	Honey Nut Cheerios	2	+	+	+	-	+
	Frosted Cheerios	0.5	+	+	+	-	+
Kellogg's	Kellogg's Apple Jacks	10	+	+	+	-	+
	Kellogg's Froot Loops	9	+	+	+	-	+
	Kellogg's Corn Pops Cereal	5	+	+	+	-	+
	Kellogg's Frosted Flakes	4	+	+	+	-	+
	Kellogg's Rice Krispies	4	+	+	+	+	+
	Frosted Mini Wheats	4	-	+	+	+	+
	Kellogg's Cocoa Krispies Cereal Straws	4	+	+	-	-	+
	Kellogg's Froot Loops Cereal Straws	3	+	+	-	-	+
Kraft	Fruity Pebbles	4	+	+	+	-	+
	Cocoa Pebbles	4	+	+	+	-	+
	Honey-comb Cereal	3	+	+	+	-	+

"only for the promotion of foods and beverages that support healthful diets for children and youth" (IOM, 2006, p. 12). Based on this recommendation, it is important for us to assess the use of licensed characters according to the Go-Slow-Whoa nutritional metric. That analysis is presented below, along with a comparison,

over a period of time, which clarifies whether or not the use of licensed characters to promote food products to children has increased or decreased over recent years.

In 2009, six companies participating in the pledge program included licensed characters

in their ads. Listed in order of frequency, these are McDonald's, Kraft, Kellogg, Dannon, Burger King and ConAgra (see Table 12). Across all of their commercials with licensed characters, there was nearly a 50-50 split between products classified as Whoa and products classified as Slow. None of the ads that featured licensed characters promoted a truly healthy Go product.

The finding that roughly half (49.4%) of all ads from self-regulatory participants with licensed characters are devoted to nutritionally poor Whoa products represents a clear violation of the mandate to restrict the use of such figures to market genuinely healthy foods. At the same time, that finding reflects a significant improvement for the industry, as compared to past practice. In 2005, 87.8% of pledge company advertising with licensed characters was devoted to nutritionally poor Whoa products. Thus, the change accomplished since the advent of the self-regulatory program is that the industry has gone from a ratio of 7:8 to a ratio of 4:8 ads that use licensed characters to promote foods of the poorest nutritional quality. Again, while this may represent a step in the desired direction, it comes in the face of a policy recommendation that calls for a ratio of 0:8. Licensed characters should never be used to promote foods of the poorest nutritional quality to children.

Finally, our data indicate the frequency with which licensed characters are used to promote food products to children is on the rise among food companies that participate in the self-regulatory program. Table 13 shows that 15.2% of all food ads from participating companies included a licensed character in 2009. For some companies, a much higher proportion of their overall advertising uses this tactic. For example, Burger King (50.0%), Dannon (47.4%) and McDonald's (43.5%) all are well above the mean. But the key finding in this table is that the use of licensed characters is up from 8.8% of ads by self-regulatory participants in 2005 to 15.2% in 2009. This finding is consistent with the pattern observed in the industry overall, where the levels were up to 15.7% in 2009, as compared to 9.7% in 2005.

In summary, the use of licensed characters to market food products to children is on the rise, and this raises substantial concern given that roughly half of all ads featuring licensed figures

promote non-nutritious food options. Licensed characters are never used to promote truly healthy Go food products, which are good for children and can be consumed in abundance.

Summary of Key Findings

This section addresses the impact of self-regulation. To review, our analysis produced two disparate key findings. The first is that the Children's Food and Beverage Advertising Initiative has fulfilled the "letter of the law" in terms of complying with the promises offered by each participating pledge company. Our data make clear that all 15 companies involved in the initiative at the time this study was conducted met their individual pledges by either (a) not advertising on television to child audiences or (b) advertising only food products that meet nutritional guidelines specified by the parent corporation. Moreover, companies also met their pledge to use licensed characters solely in advertising food products that comply with the parent corporation's guidelines for healthier products, which the self-regulatory program calls "better-for-you" foods.

The second key finding, at odds with the first, is that roughly two-thirds (68.5%) of all foods that comply with company nutritional guidelines established by the self-regulatory initiative are actually classified in the poorest nutritional category, according to the U.S. Department of Health and Human Services. To ensure the point is clear, we underscore the disconnect between these two key findings. The Children's Food and Beverage Advertising Initiative labels all foods that comply with its standards as "better-for-you" and, implicitly, healthy. In contrast, however, roughly two of every three of these pledge-compliant items are classified as Whoa products, which should be consumed "only once in awhile or on special occasions, such as your birthday," according to the U.S. Department of Health and Human Services (2005, p. 14).

Our study also applied a novel analysis identified as the Optimal Composite Nutritional Standard. These guidelines were devised by selecting criteria that best protect child health from among all standards employed by participating pledge companies in devising their own unique nutritional standards for self-regulation. Our findings demonstrate that only a very small minority of food products from

TABLE 12

Pledge Company Use of Licensed Characters, by Nutritional Quality Category

Pledge Company	2009				2005			
	N of Ads	Whoa	Slow	Go	N of Ads	Whoa	Slow	Go
McDonalds	37	8.1%	91.9%	-	3	100%	-	-
Kraft	17	100%	-	-	13	92.3%	7.7%	-
Kellogg's	9	100%	-	-	15	93.3%	6.7%	-
Dannon	9	100%	-	-	-	-	-	-
Burger King	7	-	100%	-	4	75.0%	25.0%	-
ConAgra	2	100%	-	-	-	-	-	-
General Mills	-	-	-	-	3	-	100%	-
Pepsi	-	-	-	-	6	100%	-	-
Nestle	-	-	-	-	5	100%	-	-
Overall	81	49.4%	50.6%	-	49	87.8%	12.2%	-

TABLE 13

Pledge Company Use of Licensed Characters, by Year

Pledge Company	2009		2005	
	N of Ads	% of Ads with Licensed Character	N of Ads	% of Ads with Licensed Character
Kraft	89	19.1%	66	19.7%
McDonalds	85	43.5%	48	6.3%
General Mills	81	-	50	6.0%
Kellogg's	57	15.8%	120	12.5%
Dannon	19	47.4%	8	-
ConAgra	17	11.8%	10	-
Burger King	14	50.0%	12	33.3%
Pepsi	-	-	45	13.3%
Nestle	-	-	24	20.8%
Overall	534	15.2%	557	8.8%

companies participating in self-regulation meet these standards. Only 12% of children's meal products and 8% of cereal products complied with the applicable Optimal Composite Nutritional Standard. The most striking implication of these findings is the lack of consistency in the standards employed to define healthier products across the range of participating companies. Our data show that roughly nine out of every 10 products that meet the nutritional standards adopted by one company violate the standards of one or more of their competitors.

Finally, our study determined that the use of licensed characters in food marketing to children is on the rise. Indeed, the proportion of ads featuring licensed characters from companies that participate in self-regulation has nearly doubled over the past four years, from 8.8% in 2005 to 15.2% in 2009. The finding that raises most concern, however, is that roughly half of all ads from pledge companies that use licensed characters promote foods of the poorest nutritional quality to children. This practice stands squarely in contrast to the recommendation of the Institute of Medicine

(2006) of the National Academies, which called for the food industry to limit all use of licensed characters to products that support healthful diets for children.

The best accomplishment of the industry's program of self-regulation is illuminated by comparisons between companies that participate in the initiative and those that do not. Participating pledge companies devote less of their overall advertising to foods of the poorest nutritional quality (68.5%) than companies that eschew self-regulation (82.9%); similarly, pledge companies devote more of their advertising (31.0%) to moderately healthy foods than non-participating companies (15.1%). Neither group delivers any meaningful amount of advertising for truly healthy food products.

It's important to note that more than one-quarter of all televised food advertising to children (28.7%) originates with companies outside the umbrella of industry self-regulation. This factor, coupled with the finding that most of the products that meet the nutritional guidelines for self-regulation are not in fact considered healthy foods, yields the most compelling finding of the entire study. The marketplace of televised food advertising to children remains dominated by products of the poorest nutritional quality, a pattern that has persisted for decades. While modest gains have been achieved in terms of improving the nutritional quality of foods marketed to children on television, those accomplishments do not begin to approach the goals sought by public health agencies such as the Institute of Medicine.

Conclusion

It has become increasingly clear in recent years that our nation faces a childhood obesity crisis. Both the profound personal costs to victims as well as the economic implications of treatment expenses underscore the need for strong and timely action to reverse this epidemic. Many factors contribute to childhood obesity, and among them are the billions of dollars invested annually by food companies to promote low-nutrient, high-density food products to children. These marketing efforts mean that virtually every child in the nation experiences thousands of exposures annually to

advertising messages intended to persuade young people to consume foods that are not part of a healthy diet (Holt, Ippolito, Desrochers, & Kelley, 2007).

In response to growing public concern, a large segment of the food marketing industry implemented a program of self-regulation intended to improve the nutritional quality of the foods advertised to children. The effort is known as the Children's Food and Beverage Advertising Initiative. This study evaluates the impact of the self-regulatory program by comparing overall levels of nutritional quality in the foods advertised during children's television programs in 2005, before the advent of this initiative, to levels in 2009, when the initiative was in full force.

The data from our study demonstrate that industry self-regulation has achieved only the slightest degree of improvement in televised food marketing to children. Across all television advertising to children, the proportion of foods of the poorest nutritional quality has been reduced from 84% in 2005 to 72.5% in 2009. Meanwhile, advertising to children for truly healthy foods remains virtually invisible, while only modest improvements have been accomplished by increasing the percentage of advertising devoted to moderately nutritious foods, from 12.9% in 2005 to 26.6% in 2009.

This overall outcome is a significant disappointment, given industry self-regulation has been argued by its proponents as an effective alternative to governmental regulation to achieve the dramatic reforms needed in food advertising targeted at our nation's children. As the Institute of Medicine (2006) has noted, the advertising environment contributes significantly to the obesity crisis, as it breeds unhealthy eating habits early on that may last a lifetime, while it also exerts short-term influence on the consumption of products that are unhealthy when consumed in abundance. One of the most simple, yet telling, findings of this study is that, for every one ad for a truly healthy food product that appears on television, a total of 75 other food and beverage commercials are shown, with 55 of these for products that are classified in the poorest nutritional quality category. Reform at this level is clearly insufficient to address the current crisis of childhood obesity. The effort to improve eating habits of our nation's children cannot be successful in a

business-as-usual environment that continues to allow unhealthy food products to predominate in advertising directed to children.

The data in this study illuminate the fundamental limitations of industry self-regulation. In the face of recommendations that the industry discontinue its marketing of non-nutritious foods to children, and emphasize only healthy food offerings in its child-oriented advertising, the Children's Food and Beverage Advertising Initiative embraced "better-for-you" products as the solution. Indeed, most participating companies pledged to advertise only "better-for-you" foods to child audiences. The problem is that the majority of these so-called "better-for-you" foods are not genuinely healthy for children. Despite the fact that a portion of the undesirable ingredients (e.g., fat, sugar, salt) has been removed, the overall nutritional value of most of these offerings remains so low that they are still classified as *Whoa* products, which should not be consumed on a regular basis.

In conclusion, our evidence distills to two key points: (1) the industry has done everything it promised in terms of fulfilling the details of its self-regulatory pledges and (2) that effort has been completely ineffective in shifting the landscape of food marketing to children away from its overwhelming emphasis on non-nutritious products that place children at risk of becoming obese. With self-regulation fully implemented, nearly three-quarters (72.5%) of all food advertising to children continues to promote low-nutrient, high-density products that are classified in the poorest nutritional category by governmental standards.

This outcome can hardly be said to meet the industry's stated goal of changing the overall landscape of food marketing to children. Moreover, it falls far short of the Institute of Medicine's recommendation that the industry reverse its reliance on marketing low-nutrient, high-density food products to children. At the present pace of industry reform—a reduction of roughly 12% in the proportion of child-oriented food ads for *Whoa* products over four years' time—it would take until the year 2017 for moderately healthy *Slow* food products and truly healthy *Go* products to outnumber the advertisements for nutritionally poor *Whoa* products that children see. Worse still, it would take until the year 2033 to end child-

targeted advertising for nutritionally poor *Whoa* products entirely, and that prediction presumes that the current pace of reform observed by this study would be maintained over the next quarter of a century, which is hardly a safe bet.

Given this evidence, public health officials and policymakers need to seriously consider regulatory intervention to achieve more stringent reductions in the advertising of nutritionally deficient food products to children. As noted previously, the Institute of Medicine (2006) has recommended that Congress should intervene and adopt legislation to ensure that food marketers emphasize healthful food and beverage products in their child-oriented advertising, if the industry failed to achieve this outcome through its voluntary efforts. The data from our study could not provide a clearer verdict documenting the failure of self-regulation, an outcome that some have already predicted (Brownell & Warner, 2009). In the face of pleas for significant reform, the industry has accomplished what might generously be labeled as baby steps. With the current childhood obesity crisis approaching the number one threat to our nation's public health, it is clear that the failure to act strongly and swiftly holds serious adverse implications for generations of America's children. Bold strides, rather than tiny steps, will be required to reverse the longstanding predominance of unhealthy food products in the children's advertising environment.

References

- Barcus, F.E. (1977). *Children's television: An analysis of programming and advertising*. New York: Praeger.
- Brownell, K.D., & Warner, K.E. (2009). The perils of ignoring history: Big tobacco played dirty and millions died. How similar is big food? *The Milbank Quarterly*, 89(1), 259-294.
- Calvert, S. (2008). Children as consumers: Advertising and marketing. *The Future of Children*, 18 (1), 205-234.
- Chester, J. (2008). *Digital destiny: New media and the future of democracy*. New York: The New Press.
- Chester, J., & Montgomery, K. (2007). *Interactive food and beverage marketing: Targeting children and youth in the digital age*. Berkeley, CA: Berkeley Media Studies Group. Available at <http://www.digitalads.org>
- Condry, J., Bence, P., & Scheibe, C. (1988). Nonprogram content of children's television. *Journal of Broadcasting & Electronic Media*, 32, 255-270.
- Council of Better Business Bureaus. (2007, July 18). Better Business Bureau announces food and beverage advertising commitments from 11 industry leaders. Press release. Arlington, VA: Council of Better Business Bureaus, Inc. Available at: <http://www.bbb.org/us/article/better-business-bureau-announces-food-and-beverage-advertising-commitments-from-11-industry-leaders-723>
- Federal Communications Commission. (2009). Media and childhood obesity. Available at: <http://www.fcc.gov/obesity/>
- Federal Trade Commission. (2008, July). *Marketing food to children and adolescents: A review of industry expenditures, activities, and self-regulation*. Washington, DC: author.
- Gunter, B., Oates, C., & Blades, M. (2005). *Advertising to children on TV: Content, impact, and regulation*. Mahwah, NJ: Lawrence Erlbaum Associates.
- Harkin, T. (2007). Preventing childhood obesity: The power of policy and political will. *American Journal of Preventive Medicine*, 33 (48), S165-S166.
- Hastings, G., Stead, M., McDermott, L., Forsyth, A., MacKintosh, A., Rayner, M., et al. (2003). *Review of research on the effect of food promotion to children*. Glasgow, Scotland: Center for Social Marketing.
- Hawkes, C. (2007). Regulating food marketing to young people worldwide: Trends and policy drivers. *American Journal of Public Health*, 97 (11), 1962-1973.
- Holt, D., Ippolito, P., Desrochers, D., & Kelley, C. (2007). *Children's exposure to TV advertising in 1977 and 2004: Information for the obesity debate*. Washington, DC: Federal Trade Commission, Bureau of Economics Staff Report.
- Institute of Medicine, National Academies. (2005). *Preventing childhood obesity: Health in the balance*. Washington, DC: The National Academies Press.
- Institute of Medicine, National Academies. (2006). *Food marketing to children and youth: Threat or opportunity?* Washington, DC: The National Academies Press.
- Kaiser Family Foundation. (2004). *Issue brief: The role of media in childhood obesity*. Menlo Park, CA: Henry J. Kaiser Family Foundation.
- Kolish, E., & Peeler, L. (2008, July). *Changing the landscape of food and beverage advertising: The Children's Food and Beverage Advertising Initiative in action*. Arlington, VA: Council of Better Business Bureaus.
- Krishnamoorthy, J.S., Hart, C., & Jelalian, E. (2006). The epidemic of childhood obesity: Review of research and implications for public policy. *SRCD Social Policy Report*, 19, 2-17.
- Kunkel, D., & Gantz, W. (1992). Children's television advertising in the multi-channel environment. *Journal of Communication*, 42(3), 134-152.
- Kunkel, D., & McIlrath, M. (2003). Message content in advertising to children. In E. Palmer & B. Young (Eds.), *Faces of televisual media: Teaching, violence, selling to children*, pp. 287-300. Mahwah, NJ: Lawrence Erlbaum Associates.
- Kunkel, D., McKinley, C., & Stitt, C. (2008, May). *Quantity and nutritional quality of food advertising during children's television programming in the U.S.* Paper presented at the annual conference of the International Communication Association, Montreal, Canada.
- Kunkel, D., Wilcox, B., Cantor, J., Palmer, E., Linn, S., & Dowrick, P. (2004). *Psychological issues in the increasing commercialization of childhood*. Report of the APA Task Force on Advertising and Children. Washington, DC: American Psychological Association.
- Larson, N., & Story, M. (2008, October). *Food and beverage marketing to children and adolescents: What changes are needed to promote healthy eating habits?* Princeton, NJ: Robert Wood Johnson Foundation.
- Markey, E. (2007, June 22). Let's give parents the tools to fight unhealthy media images. Opening statement at Hearings of the Subcommittee on Telecommunications and the Internet, U.S. House of Representatives. Available at: http://markey.house.gov/index.php?option=com_content&task=view&id=2933&Itemid=88
- Miller, Patti (2008, September 23). Testimony of Patti Miller, Vice President of Children Now. Hearings before the U.S. Senate. Committee on Appropriations, Subcommittee on Labor, Health and Human Services, and Related Agencies. Washington, DC. Available at: <http://appropriations.senate.gov/ht-labor.cfm?method=hearings.view&id=1735e2de-afb5-4179-81bc-d0bld2ce504a>
- Neuman, William. (2009, September 5). For your health, Fruit Loops. *New York Times*, pp. B1, B5.
- Palmer, E., & Carpenter, C. (2006). Food and beverage marketing to children and youth: Trends and issues. *Media Psychology*, 8, 165-190.
- Peeler, L., Kolish, E., & Enright, M. (2009, October). *The Children's Food and Beverage Initiative in action: A report on compliance and implementation during 2008*. Arlington, VA: Council of Better Business Bureaus.
- Stitt, C., & Kunkel, D. (2008). Food advertising during children's television programming on broadcast and cable channels. *Health Communication*, 23, 573-584.
- U.S. Department of Health and Human Services. (2005). Ways to enhance children's activity and nutrition. Retrieved Feb. 2, 2006 from <http://www.nhlbi.nih.gov/health/public/heart/obesity/wecan/>
- Vandewater, E.A., & Cummings, H.M. (2008). Media use and childhood obesity. In S. Calvert & B.J. Wilson (Eds.), *The handbook of children, media, and development* (pp. 355-380). Malden, MA: Blackwell Publishing.
- Weber, K., Story, M., & Harnack, L. (2006). Internet food marketing strategies aimed at children and adolescents: A content analysis of food and beverage brand web sites. *Journal of the American Dietetic Association*, 106 (9), 1463-1466.

Acknowledgements

The authors would like to convey their appreciation and gratitude to the following individuals who made significant contributions to this research. Their efforts and commitment were integral to the success of the project.

Business Manager: Carey Willits

Technology Supervisor: Vanessa Garrison

Coders: Caroline Duff, Austin Harrell, Christina Johnson, Lareese Lopez, Whitney Mediano, Jennifer Mota, Katherine Schober.

Children Now would like to thank The California Endowment for its support of this research. The organization would also like to thank the following staff: Wilma Chan, Eileen Espejo, Brian Kennedy, Ted Lempert, Jeff McIntyre, Ronald Pineda, Kristi Schutjer-Mance, Kathryn Washburn Main and Annie Xu.

Children Now Board of Directors

Jane K. Gardner, *Chair*
Harbour Strategic Consulting

Peter D. Bewley, *Vice Chair*
The Clorox Company (Retired)

Grace Won, *Secretary*
Farella Braun + Martel LLP

Kathleen Abernathy
Wilkinson Barker Knauer, LLP

Neal Baer, M.D.
Wolf Films/Universal Television

Laura Casas Frier
Foothill-De Anza Community College District

Jim Cunneen
California Strategies, LLC

John Garcia
Kaiser Permanente

David G. Johnson
Johnson-Roessler Company

Allan K. Jonas
Jonas & Associates

Donald Kennedy
Science Magazine

Gay Krause
Foothill College, Krause Center for Innovation

Lenny Mendonca
McKinsey & Company

Theodore R. Mitchell
NewSchools Venture Fund

Molly Munger
The Advancement Project

Nancy Murray
Pillsbury Madison & Sutro LLP (Retired)

Craig Parsons
Communications Consultant

Hon. Cruz Reynoso
UC Davis, School of Law

Karen R. Schievelbein
UnitedHealth Group (Retired)

Katharine Schlosberg
Educational Consultant

Gloria Tristani
Spiegel & McDiarmid

Jennie Ward Robinson, Ph.D.
Institute for Public Health and Water Research

Holly L. Sutton, *Of Counsel*
Farella Braun + Martel LLP

Appendix A

The U.S. Department of Health and Human Services'
Go-Slow-Whoa Food Rating System Example Chart

Food Group	GO (Almost Anytime Foods)	SLOW (Sometimes Foods)	WHOA (Once in a While Foods)
	Nutrient-Dense ←		→ Calorie-Dense
Vegetables	Almost all fresh, frozen, and canned vegetables without added fat and sauces	All vegetables with added fat and sauces; oven-baked French fries; avocado	Fried potatoes, like French fries or hash browns; other deep-fried vegetables
Fruits	All fresh, frozen, canned in juice	100 percent fruit juice; fruits canned in light syrup; dried fruits	Fruits canned in heavy syrup
Breads and Cereals	Whole-grain breads, including pita bread; tortillas and whole-grain pasta; brown rice; hot and cold unsweetened whole-grain breakfast cereals	White refined flour bread, rice, and pasta. French toast; taco shells; cornbread; biscuits; granola; waffles and pancakes	Croissants; muffins; doughnuts; sweet rolls; crackers made with trans fats; sweetened breakfast cereals
Milk and Milk Products	Fat-free or 1 percent low-fat milk; fat-free or low-fat yogurt; part-skim, reduced fat, and fat-free cheese; low-fat or fat-free cottage cheese	2 percent low-fat milk; processed cheese spread	Whole milk; full-fat American, cheddar, Colby, Swiss, cream cheese; whole-milk yogurt
Meats, Poultry, Fish, Eggs, Beans, and Nuts	Trimmed beef and pork; extra lean ground beef; chicken and turkey without skin; tuna canned in water; baked, broiled, steamed, grilled fish and shellfish; beans, split peas, lentils, tofu; egg whites and egg substitutes	Lean ground beef, broiled hamburgers; ham, Canadian bacon; chicken and turkey with skin; low-fat hot dogs; tuna canned in oil; peanut butter; nuts; whole eggs cooked without added fat	Untrimmed beef and pork; regular ground beef; fried hamburgers; ribs; bacon, fried chicken, chicken nuggets; hot dogs, lunch meats, pepperoni, sausage; fried fish and shellfish; whole eggs cooked with fat
Sweets and Snacks*		Ice milk bars; frozen fruit juice bars; low-fat or fat-free frozen yogurt and ice cream; fig bars, ginger snaps, baked chips; low-fat microwave popcorn; pretzels	Cookies and cakes; pies; cheese cake; ice cream; chocolate; candy; chips; buttered microwave popcorn
Fats/Condiments	Vinegar; ketchup; mustard; fat-free creamy salad dressing; fat-free mayonnaise; fat free sour cream	Vegetable oil, olive oil, and oil-based salad dressing; soft margarine; low-fat creamy salad dressing; low-fat mayonnaise; low-fat sour cream**	Butter, stick margarine; lard; salt pork; gravy; regular creamy salad dressing; mayonnaise; tartar sauce; sour cream; cheese sauce; cream sauce; cream cheese dips
Beverages	Water, fat-free milk, or 1 percent low-fat milk; diet soda; unsweetened ice tea or diet iced tea and lemonade	2 percent low-fat milk; 100 percent fruit juice; sports drinks	Whole milk; regular soda; calorically sweetened iced teas and lemonade; fruit drinks with less than 100 percent fruit juice

**Though some of the foods in this row are lower in fat and calories, all sweets and snacks need to be limited so as not to exceed one's daily calorie requirements.*

***Vegetable and olive oils contain no saturated or trans fats and can be consumed daily, but in limited portions, to meet daily calorie needs.*

Children Now is a nonpartisan research and advocacy organization working to raise children's well-being to the top of the national policy agenda. The organization focuses on ensuring quality health care, a solid education and a positive media environment for all children. Children Now's strategic approach creates awareness of children's needs, develops effective policy solutions and engages those who can make change happen.

1212 Broadway, 5th Floor

Oakland, CA 94612

T: 510.763.244

F: 510.763.1974

childrennow.org

Local Perspective



Central California REGIONAL OBESITY PREVENTION PROGRAM

Healthy Environments • Healthy Choices • Healthy People

Working together, we can create healthy communities in the San Joaquin Valley.

The Central California Regional Obesity Prevention Program (CCROPP) is dedicated to creating environments that support healthy eating and active living. In many of our communities, people can't easily buy healthy food such as fresh fruits and vegetables. Likewise, many neighborhoods are designed in ways that make it difficult and unsafe to be physically active outdoors. We all need to work together to assure that families have access to healthy choices.

Obesity and Health

The San Joaquin Valley faces alarming rates of obesity among all age groups, leading to equally alarming rates of childhood and adult diabetes, heart disease, high blood pressure and other obesity-related illnesses. A recent study by the Central Valley Health Policy Institute at California State University, Fresno, revealed that the percentage of overweight and obesity in the Valley is significantly higher than the statewide average: 65% of adults 18-64 versus 56.2% statewide, 63% versus 55.7% among seniors and 15.5% versus 14.2% among adolescents ages 12-17. According to the U.S. Surgeon General's office, obesity is now the fastest-growing cause of illness and death in America today.

Changing Policies, Changing Lives

Historically, obesity has been addressed through interventions that focus on individual and behavioral change. Although behavior is important, the influence that food and physical activity environments have on health is often overlooked. CCROPP is committed to addressing childhood and adult obesity through place-based policy change that supports healthy eating and active living throughout the Valley. This unique, comprehensive approach is being carried out by partnerships between public health departments, community-based organizations and community councils in Fresno, Kern, Kings, Madera, Merced, San Joaquin, Stanislaus and Tulare counties. The program was developed by the Central California Public Health Partnership and is administered through Fresno State. Funding for this initiative was made possible by The California Endowment.



It Takes a Community

CCROPP partners have formed community councils in each county, bringing together community members, schools, local government, health care providers and others to fight the obesity epidemic. School administrators, teachers and PTA Boards are partnering to ensure that only healthy foods, including fruits and vegetables, are offered at school. City officials are helping maintain parks, offer recreational programs and support community gardens. Neighborhoods and public officials are working together to make The Valley Region healthier.

Healthy Changes for the San Joaquin Valley

CCROPP advocates for new and improved policies throughout the Valley to make opportunities for healthy food and physical activity choices more available. Physical activity is not a real option if there are no safe places to play. Healthy eating won't happen if there are no places for people to buy healthy foods they can afford. CCROPP is working towards change in a variety of ways to help Valley community members lead healthier lives:

- Establishing farmers markets, community gardens and markets with fresh foods in low-income neighborhoods.
- Increasing physical activity and physical education in schools and after-school programs.
- Improving parks, sidewalks and other opportunities for physical exercise, including walking trails and bicycle lanes.
- Supporting healthy food and physical activity policies in the workplace, including government, private enterprises, community organizations and gatherings.
- Promoting "smart growth" strategies to help create healthier communities.
- Encouraging physical activity and healthy eating opportunities in communities through various media outlets.
- AND MUCH MORE!



Join us!

Creating communities that support healthy eating and active living will help everyone in the Valley – children and adults – avoid chronic diseases, feel better, and live longer. With your help, we can find solutions we need and bring about healthy changes for our communities.

Get involved!

Contact the Central California Regional Obesity Prevention Program at:

1625 East Shaw Avenue, Suite 106

Fresno, CA 93710-8106

559-228-2140

Or visit our website at www.ccropp.org



The
California
Endowment

This project is funded by

The Health Challenge: Creating a Policy Agenda Focused on Place

By: Mary Lee, PolicyLink

There is no question that health in the United States is stratified by race. Blacks and Latinos suffer disproportionately from alarmingly high rates of disease and poor health outcomes. Health disparities for both groups are not only persistent; they have severe consequences that result in a reduced quality of life, and even early death.

Health policymakers and the public have generally made the state of individual health, access to health care, and health insurance the focus of policy debates. While an individual's genetic predisposition or personal behavior certainly play a role in health, individual characteristics account for just a fraction of the problem of health disparities. According to the Centers for Disease Control, lack of access to care accounts for only 10 percent of total mortality in the U.S. Environmental conditions, along with social and economic factors, actually play a much larger role in health. It is becoming increasingly clear that where you live affects your health. Accordingly, the health of Latinos and African Americans is determined by a range of environmental factors that occur in the neighborhoods where they live.

The charts and data below illustrate the gravity of health disparities by displaying selected examples of the rates of illness among Blacks and Latinos compared to those of Whites.¹

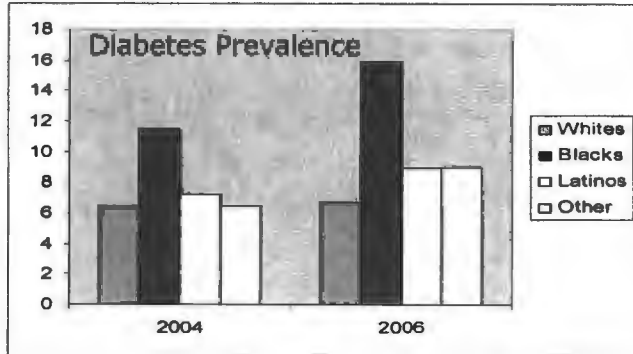
Facts

Asthma²

- Black children have a 60 percent higher prevalence of asthma than White children.
- Black people had asthma-related emergency room visits 4.5 times more often than White people.
- While deaths from asthma are rare, the death rate for Puerto Ricans specifically was 400 percent higher than the non-Hispanic White population.

Diabetes

- Black people are 2.2 times as likely to have diabetes as their White counterparts.
- Latinos are 1.5 times as likely to have diabetes as Whites.
- The death rate from diabetes in Latinos is 60 percent higher than the death rate of non-Hispanic Whites.



Source: Behavioral Risk Factor Surveillance System

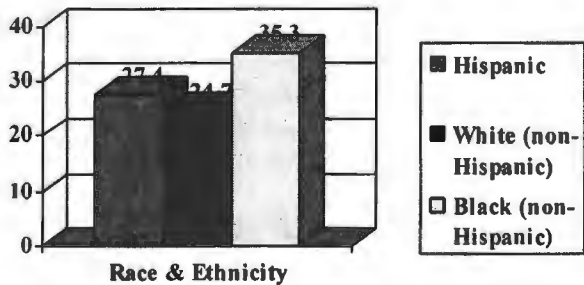
Cardiovascular Disease

- Black men are 30 percent more likely to die from heart disease than non-Hispanic White males, despite the fact that 10 percent of Blacks have been diagnosed with heart disease versus 12 percent of Whites.
- 31.6 percent of Black people have hypertension compared to 22.4 percent of White people.

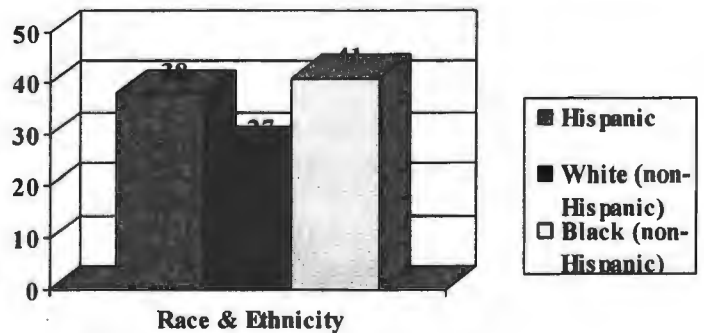
Obesity

- Black women are 70 percent more likely to be obese than non-Hispanic White women; in general, Black women have the highest rates of obesity compared to all other groups. About four out of five Black women are overweight or obese.
- Black people are 1.4 times as likely to be obese as non-Hispanic White people.
- 73 percent of Mexican American women (the largest sub-population of Latinos in the U.S.) are overweight or obese, as compared to 61.6 percent of the general female population.
- Latinos are 1.1 times as likely to be obese as non-Hispanic Whites.

Percent Obese by Race/Ethnicity
(18 years and older)



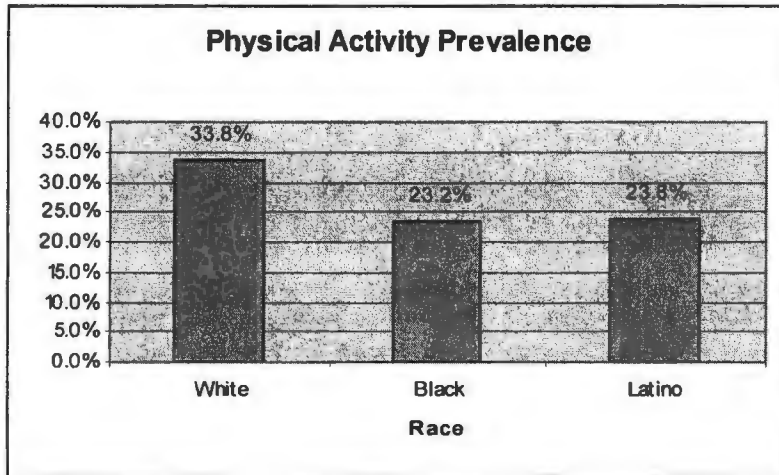
Percent of Adolescents Overweight & Obese
by Race/Ethnicity (Ages 10 to 17)



Source: Behavioral Risk Factor Surveillance System

Physical Activity During the Past Month

- Non-Hispanic White adults are more likely to engage in regular leisure-time physical activity than Latinos or non-Hispanic Black adults.
- The age-sex adjusted percentage of adults* who engage in regular leisure-time physical activity is 23.8 percent for Latinos, 33.8 percent for non-Hispanic Whites, and 23.2 percent for non-Hispanic Blacks.



Source: 2007 National Health Interview Survey on Physical Activity

Cancer

- Black people are 33 percent more likely to die from all types of cancer than White people.
- Black men are over twice as likely to die from prostate cancer than Whites.
- Black women are 34 percent more likely to die from breast cancer than White women, although Black women are diagnosed 10 percent less frequently.

Health Care Access

- 13 percent of White adults have no health care coverage.
- 22.9 percent of Black adults have no health care coverage.
- 43.3 percent of Latino adults have no health care coverage.

HIV/AIDS

- Black people made up 47 percent of all HIV/AIDS cases diagnosed in 2006.
- Black men have 7.4 times the AIDS rate and are nine times more likely to die with AIDS than non-Hispanic White men.
- Latinos made up 18 percent of AIDS cases in 2006, despite making up only 14 percent of the U.S. population.
- Latinos are 3.3 times more likely to be diagnosed with AIDS than non-Hispanic Whites.

* Age and sex are known correlates of physical activity participation so researchers statistically controlled for those variables to get an accurate depiction of how race influences physical activity.

Infant Mortality

- The infant mortality rate for Black infants is more than twice the rate for non-Hispanic White infants (13.6 deaths per 1,000 live births vs. 5.8 deaths per 1,000 live births.)
- The infant mortality rate for Puerto Ricans specifically is 40 percent higher than non-Hispanic Whites.
- Puerto Rican infants are twice as likely to die from causes related to low birthweight, compared to non-Hispanic White infants.
- Black infants are 2.4 times as likely to die from causes related to low birthweight, compared to non-Hispanic White infants.

Implications of Inaction

To understand more clearly how place affects health, consider socioeconomic status. Poverty is a predictor of health, and poor health increases as socioeconomic status (SES) decreases. In the U.S., race and ethnicity remain primary determinants of SES. Many low-income Latino and African American people live in neighborhoods defined by race and class, an example of residential segregation that is deeply entrenched.³ Poverty compounds the isolation for poor Latino and African American children who are significantly more likely to live in “double jeopardy,” meaning that they live in both poor families and poor neighborhoods.⁴ Only 1.4 percent of White children live in double jeopardy. By living in poor and racially isolated neighborhoods, Latino and Black people are more likely to suffer from dire health consequences.⁵

The health risks in these neighborhoods are cumulative and worsen over time, placing a disproportionate burden on residents that can have long-term consequences. Leading researchers have demonstrated the direct and indirect link to conditions in the community that contribute to poor health outcomes, including infant mortality, asthma and other respiratory infections, cancer, hypertension, and cardiovascular diseases. Even more alarming is research showing that the cumulative effect of high levels of stress can cause physical and mental illness, and even contribute to a shortened lifespan.

The following neighborhood conditions are among those known to be health risks and are common in low-income communities and communities of color:

Pollution

- **Poor air and water quality:** homes and schools in communities of color are frequently sited near highways, factories, and other sources of pollution and residents in these areas are subject to higher levels of exposure.
- **Undesirable land uses:** low-income neighborhoods in both rural and urban settings frequently bear the burden of toxic waste treatment or disposal facilities, diesel truck depots and rail yards, etc. Lax regulation of such operations, and of businesses such as automotive repair and dry cleaners, puts residents at risk of health hazards from contaminants.
- **Exposure to chemical and biological agents in the workplace:** residents of low-income communities and communities of color in both rural and urban locations are likely to work in industries where the risk of exposure to toxics is high.

- **Agricultural threats:** exposure to chemicals in fertilizer impact both farm workers who must handle the products, and residents of adjacent areas who are exposed to run-off present in soil and water.

Housing

Dilapidated housing is prevalent in neighborhoods where people of color and low-income people live. Threats include exposure to lead paint; mold; vermin; unsafe building materials; overcrowded units; unsafe wiring; missing or inoperable smoke detectors or other fire safety features.

Access to Healthy Food

Communities of color and low-income communities are frequently categorized as “food deserts”—areas with limited access to healthy food. Residents of both urban and rural communities are affected when they lack access to grocery stores and produce stands that offer fresh and healthy food yet are saturated with fast food outlets and convenience stores that predominantly offer highly processed packaged foods and junk food. This combination has proven to be strongly correlated with diabetes and obesity.⁶

The Built Environment

- **Lack of parks, recreation, and open space:** the number of such venues is typically inadequate in low-income neighborhoods and, where they do exist, parks and recreation centers are poorly maintained and provide little or no staff or programming.
- **Public school facilities:** frequently, schools in low-income communities and communities of color are dilapidated, overcrowded, lack recreation space, and do not offer healthy food options.
- **Unsafe streets:** typically low-income areas contain streets that are poorly designed or maintained, are too close to traffic, too narrow and lack lighting, and therefore discourage walking and biking. More urgently, lack of scrutiny and security put residents in these communities at risk of criminal activity.
- **Inadequate transit:** residents in communities of color are often isolated from employment, educational and cultural opportunity, as well as recreation and healthy food options.
- **Unhealthy, dangerous land uses:** alcohol outlets; bars; motels; recycling centers that are magnets for criminal behavior such as drug dealing and drug use; prostitution; gang activity; and violence are prevalent in these communities.
- **Public and private disinvestment:** as the aftermath of Hurricane Katrina demonstrated, inadequate public services, neglected infrastructure, and neighborhood isolation impair residents’ ability to survive or recover from a natural disaster.

Economic Opportunity and Education

Residents of low-income communities and communities of color are often isolated from opportunities that lead to economic well-being. These neighborhoods are disconnected from living-wage jobs that provide career pathways, and from quality of education.

The absence of economic opportunity and the other elements listed above expose community residents to risks that are harmful and lead to poor health.

Conversely, the presence of clean air, water, parks and recreation, safe streets, good housing, and jobs all support health. The challenge now is to secure environments for Latino and Black people that facilitate healthy choices, not impede them.

Policy Solutions

The health status of Black and Latino people is remarkably similar; so are the neighborhoods where they live. Both groups are contending with factors in their communities which impede their health. Accordingly, a focus on the relationship between place and health may yield the greatest opportunity for meaningful collaboration in the development of policy changes that would promote good health and prove mutually beneficial.

To improve health, a wide array of “non-health” strategies will need to be adopted, including: decent housing, quality schools, and living-wage jobs with career paths.⁷

In order to succeed, a wide array of non-traditional partners will need to be involved—public health advocates, researchers, academics, policymakers, community residents, community organizers, media, private industry, and philanthropy.

Moreover, the actions undertaken must be urgent and sustained and must take place at a local, regional, and national level.⁸ Fortunately, there are viable policy solutions and strategies to achieve results being implemented in communities across the nation. Some examples:

- Collaboration between public health and land use advocates and agencies, utilizing planning tools such as Health Impact Assessments, and including Health Elements in General Plans
- Joint use agreements between public agencies and private organizations, or between two public agencies, increasing the accessibility of open space and public facilities for physical activity
- School-based health clinics that include mental health services and provide treatment for students and their families
- Transit oriented development that cuts down on air pollution and increases access to good jobs, healthy food, and recreation
- Creation of living-wage jobs that provide health insurance, and support emotional and economic well-being for workers
- Incentives, such as low-interest loans, to encourage grocery stores to locate in underserved areas and existing smaller stores to carry more fresh fruits and vegetables
- Expanding local farmers’ markets, mobile vendors, food cooperatives, and community-supported agriculture to increase the availability of healthy, fresh food

Health disparities can be eliminated. To make progress, alignment is needed on a joint policy agenda that could be a powerful force for improving the health of Black and Latino people in the neighborhoods where they live.

Notes

¹ Please note that the focus of this brief is the health status of Black and Latino people in the U.S.; other communities of color also experience significant health disparities, including Asian, Pacific Islander, and Native Americans. Further, note that the data presented focus on adults. The results for youth are equally alarming. For example, 40 percent of Latino children and 30 percent of Black children are obese.

Also, please note that we recognize that the racial categories used here—Black and Latino—do not break out the vast number of ethnicities and nationalities encompassed in each group, and the data shown here are only disaggregated in a few instances. Effective policy advocacy will take such variations into account. For instance, the health status of immigrants can change depending on the length of residency in the U.S. Immigrants often have better health outcomes initially, despite the presence of risk factors, due to strong social support, kinships networks, and cultural resiliency. Yet as their length of time in U.S., and acculturation increase, health status for many immigrant groups often decreases.

² Health disparities data on asthma, diabetes, cardiovascular disease, obesity, cancer, HIV/AIDS, and infant mortality are drawn from the Office of Minority Health.

³ Data from the 2000 Census indicate that White people in the U.S. typically live in neighborhoods that are more than 80 percent white, Blacks in neighborhoods more than 50 percent Black, and Latinos in neighborhoods more than 40 percent Latino.

⁴ According to the March/April 2008 issue of the journal *Health Affairs*, <http://content.healthaffairs.org/current.shtm>.

⁵ <http://content.healthaffairs.org/cgi/content/abstract/27/2/321>.

⁶ *Designed for Disease: The Link Between Local Food Environments and Obesity and Diabetes*, PolicyLink 2008, <http://www.policylink.org/documents/DesignedforDisease.pdf>.

⁷ *Unnatural Causes: Is Inequality Making Us Sick?*, <http://www.unnaturalcauses.org/>.

⁸ Note as well the urgent need for global efforts on health disparities, as articulated by the World Health Organization, http://whqlibdoc.who.int/hq/2008/WHO_IER_CSDH_08.1_eng.pdf.

Joint Informational Hearing

Senate Food and Agriculture Committee

Senator Dean Florez, Chair

and

Senate Health Committee

Senator Elaine Kontominas Alquist, Chair

**Food and Beverages: Strategies to Recoup the Health Costs of
Excessive Sugar Consumption**

April 20, 2010

Written Testimony and/or Material Submitted

Scheduled Witnesses

Gail Woodward Lopez, MPH, RD, Associate Director, the Dr. Robert C. and Veronica Atkins Center for Weight and Health, University of California, Berkeley

Harold Goldstein, DrPH, Executive Director, California Center for Public Health Advocacy

Danielle Greenberg, PhD, FACN, Director of Nutrition and Scientific Affairs at PepsiCo. Former Associate Research Professor of Psychology in Psychiatry at Cornell University Medical College

Robert Achermann, Director of Government Relations, California Nevada Soft Drink Association

Julian Canete, Executive Director, California Hispanic Chamber of Commerce

Samantha Dabish, Vice President, Neighborhood Market Association

Michael I. Goran, PhD, Professor of Preventive Medicine, Physiology & Biophysics and Pediatrics, The Dr. Robert C. and Veronica Atkins Endowed Chair in Childhood Obesity and Diabetes, Keck School of Medicine, University of Southern California, Director of USC Childhood Obesity Research Center

Lisa Katic, Registered Dietician, Principle, K Consulting

Katie Woodruff, MPH, Deputy Director, Berkeley Media Studies Group

Genoveva Islas-Hooker, MPH, Regional Coordinator, Central California Regional Obesity Prevention Program

Dana Richardson, Representative of the Healthy Eating Active Communities Initiative

Statements Submitted

The Coca-Cola Company

American Beverage Association

**Written testimony and/or additional material
submitted by:**

**Gail Woodward Lopez, MPH, RD
Associate Director
the Dr. Robert C. and Veronica Atkins
Center for Weight and Health
University of California, Berkeley**

Joint Informational Hearing of the Senate Food/Agriculture and Health Committees
April 20, 2010
Testimony by Gail Woodward-Lopez

Madame Chair, Mr. Chair and members, thank you for inviting me here today to share findings on the relationship between sweetened beverages and obesity. I am the Associate Director of the Dr. Robert C. and Veronica Atkins Center for Weight and Health at the University of California, Berkeley and the author of a recent book on the dietary determinants of obesity

The Center for Weight and Health is well known for its work in the area of child obesity prevention and particularly for evaluating programs and policies and synthesizing research to inform policy and action.

We all know that we are in the midst of an obesity epidemic that will have catastrophic consequences if not addressed. We also know that calorie intake has increased dramatically over the same time that obesity rates have skyrocketed. This increase in calories alone is more than enough to account for the rise in obesity we have observed in recent decades. But obesity is not merely the result of eating too much of everything, it is also influenced by *what* we eat. Therefore it is critical to identify which dietary factors are contributing most to excess weight gain.

At the Center for Weight and Health we conducted an extensive, systematic literature review and found that sweetened beverage intake was the single dietary factor with the strongest evidence linking it to obesity.

Let me share with you the 4 lines of evidence demonstrating the link between obesity and sweetened beverage consumption:

The first line of evidence compares secular trends in dietary intake and obesity. The parallel rise in sweetened beverage intake and obesity is quite striking. Between 1977 and 2002 when obesity rates were climbing most steeply, Americans more than doubled their intake of sweetened beverages. Sweetened beverage intake accounted for 43% of the increase in overall calorie intake during this time period. By 2004, Americans were consuming between 9-13% of their total calories from sweetened beverages alone. Among food and beverage items consumed by Americans in recent decades soft drinks are the number one contributor to our calorie intake.

The second line of evidence addresses biological feasibility and answers the question "*how* do sweetened beverages contribute to excess weight gain?". Researchers have found that when we consume calories in liquid form – compared to solid foods - we don't compensate with an equivalent reduction in the intake of other foods and beverages. One analysis of over 40 studies concluded that 91% of liquid calories are not compensated for. In other words the higher the sweetened beverage consumption the higher the total calorie intake. Several well-conducted studies have reported significant associations between sweetened beverage intake and calorie intake. These extra calories will lead to weight gain if there is no equivalent increase in energy expenditure.

The third line of evidence includes over 50 studies that looked at the direct relationship between sweetened beverage intake and some measure of body fat or body weight. The majority of these studies and especially those of more rigorous design found that higher levels of sweetened beverage intake were associated with higher weight. These results were consistent across all age ranges and ethnic groups examined and were especially strong for children.

The fourth and final line of evidence is the most powerful. These findings are from 9 experimental studies where sweetened beverage intakes are either increased or decreased and then the resulting change in weight is measured. Studies of strongest design, the majority of which were randomized, controlled trials, showed that reducing intake of sweetened beverages resulted in measurable and significant decreases in body fat. Conversely when sweetened beverage intake was increased subjects gained weight, up to 3.5 pounds in just 10 weeks.

So, do sweetened beverages cause excess weight gain? The evidence is quite compelling. There are several well-established criteria that must be met to establish causation. The evidence we reviewed meets all these criteria and therefore supports a causal link between sweetened beverage intake and obesity. Compared to other dietary components, sweetened beverages stand out as a major contributor to the obesity epidemic. The evidence therefore suggests that a reduction in the intake of sweetened beverages alone could have a measurable impact on obesity rates.

An analysis we conducted revealed that the increase in sweetened beverage intake between 1977 and 2002 was equivalent to 43% of the total increase in calories over the same time period. Assuming that no more than 50% of the increase in calories from sweetened beverages was compensated for by a reduction intake of other foods and beverages, we estimate that at least 22% of the weight gained over that time period was due to the increased intake in sweetened beverages.

In conclusion, it is clear that not all calories are equal when it comes to obesity. The same number of calories provided in different foods has a different impact on how full or satisfied we feel and therefore how likely we are to continue eating.

Further, most Americans have little room in their diets for "empty" calories such as those from sweetened beverages, that do not provide any additional naturally occurring nutrients. These beverages therefore are either consumed in addition to healthier options thereby leading to obesity or are replacing healthier foods thereby reducing the intake of other needed nutrients.

Some may claim that sweetened beverages sales are essential for funding services and programs in institutions such as our schools. However, our studies conducted in nearly 100 schools found that the profits were less than \$400 per year per vending machine, indicating that the schools are getting a very small portion of the dollars that students are spending on these unneeded and/or potentially harmful beverages.

So what can we do? Committees convened by the Institute of Medicine and the Centers for Disease Control and Prevention, as well as reviews conducted by our Center and others, suggest that alterations in pricing and access are among the most promising strategies for changing dietary intake. Therefore policies that increase the price and/or reduce access to sugar sweetened beverages are merited.

**Written testimony and/or additional material
submitted by:**

**Harold Goldstein, DrPH
Executive Director
California Center for Public Health Advocacy**

JOINT SODA TAX HEARING

April 20, 2010

Harold Goldstein, DrPH
Executive Director
California Center for Public Health Advocacy

Mr. Chair, Madame Chair, and members. My name is Dr. Harold Goldstein, I am the Executive Director of the California Center for Public Health Advocacy, a nonpartisan, nonprofit organization dedicated to protecting the health of all Californians. Thank you for inviting me to speak here today about sugar sweetened beverages and the economics of soda taxes.

In 1985 when obesity was first measured in California, 9% of us were obese. Today, do you know how many of us are obese today? 24% -- almost tripled. As you heard from the last speaker, national data shows that sugar sweetened beverages have been the single largest contributor to the obesity epidemic in the US, responsible for almost half other additional daily calories consumed by the average American since the 1970s and directly responsible for 20-40% of the epidemic. And this obesity epidemic is expensive, costing California more than \$20 billion dollars per year.

How can that be? How can sodas be such a large part of the problem? Think about all the ways the beverages world has changed in the last 30 years? Those little 6 and 8 ounce bottles have been on steroids, growing exponentially (SLIDE), as you can see here. 6 packs have become 12 and 24 packs; small ice filled glasses at restaurants have been replaced by free massive refills; and until recently, these products were widely available in California's public schools, taken out against massive opposition from the beverage industry (something they now seem to take credit for).

And remember, one 20 ounce soda has 17 teaspoons of sugar, its like drinking a piece of chocolate cake every time you are thirsty. Imagine going to Starbucks and putting 17 little bags of sugar in your coffee!

I was a kid in the 1970s, and back then soda was a treat for children. Today in California, one-third of children age 2-5 drink a soda or more a day in California. One-third of 2-5 year olds. Nationwide, the average person drinks 50 gallons of sugar-sweetened beverages per year, not just carbonated soft drinks, but sports drinks and energy drinks and ice teas, and Vitamin waters. 50 gallons translates to 39 pounds of sugar per year, as much as you see displayed here.

Leading health organizations have reviewed the research and are all calling to reduce consumption of sugar and sugar-sweetened beverages, organizations like the CDC, the USDA, the Institute of Medicine, and several major medical organizations and the World Health

Organization. About the only organizations that don't agree with this premise are ones that make money selling these products or affiliated with them.

As new research has come to light about the harmful effects of these beverages, there have been growing efforts across the country to establish needed public health interventions: everything from public information campaigns to taxing the whole range of sugar sweetened beverages to pay for their harmful consequences. Industry has responded with a script that is pretty well known, much is which is remarkably similar to what the tobacco industry said when they were fighting public health interventions.

Today, I expect you are going to hear 10 things from industry today: 2 will be true, 8 will be false.

1. ONE: First, they will say that many things contribute to the obesity problem. That is **TRUE**. But that's not an argument for doing nothing. We have to start where the science is strongest, and the science is strongest on sugar-sweetened beverages.
2. TWO: Second, they'll probably say that just reducing sugar-sweetened beverage consumption won't solve the obesity problem, that even studies in California show that half of people who are overweight don't drink soda. That is **TRUE** as well. But the half that DO drink soda are 27% more likely to be overweight. Again, there is no one magic bullet. But that doesn't mean you ignore the biggest culprit.
3. THIRD, industry will argue that sugar sweetened beverages are a tiny portion of calories that people consume. That is **FALSE**. Sodas are the single largest source of discretionary calories in the American diet, they are far and away the biggest source of added sugar, and they have been the single largest source of new calories in the American diet. The average child drinks 175 calories a day in sugar-sweetened beverages. Industry will probably claim that sodas make up only 5% of the foods supply – quoting food supply data, not what people report consuming, which is more like 10-13%.
4. FOURTH, industry will say that all calories are the same, that a food is a food is a food. That it is unfair to single out one industry. This is **FALSE** for two reasons.
 - a. First, all other products provide at least SOME nutritional benefits along with their calories. Sodas are completely empty calories. We drink soda like its water, but its not – you could consider nothing more than a sugar delivery device.
 - b. And more importantly, there is poor calorie compensation for calories consumed in liquid form. When you go out to lunch, you don't feel more full if you drink a regular Coke instead of a diet Coke, like you would if you had an extra sandwich. As mammals, we evolved consuming two beverages: mother's milk as infants and water as children and adults. Our bodies not biologically equipped to recognize or respond to liquid calories. So you can imagine the cumulative effect

of drinking all of these calories, especially when the sugar in those beverages may actually stimulate hunger, rather than satiate it.

5. FIFTH, industry will claim – FALSELY, as the tobacco industry did when they said that smoking doesn't cause lung cancer —that the science showing the link between soda consumption and obesity is unclear. They say this even though the scientific evidence is absolutely overwhelming – having convinced the CDC and so many others. They will quote the few studies that show contrary results (many of which they paid for themselves), and they will bring those few experts – most on their own payroll – to testify on their behalf. Of course there are some scientists who say soda isn't bad for you, just like there are scientists who say that global warming hasn't been proven.
6. SIXTH: They will say that raising the price of sugar sweetened beverages will not lower consumption. This too is **FALSE**.
 - a. A comprehensive review of the literature has shown that for every 10% increase in price in sugar sweetened beverages, there is a 7.8% decrease in consumption. The beverage industry's own data suggest the relationship is even stronger. I have provided you with a copy of Beverage Digest dated November 21, 2008, that says, and I quote, "Industry sources have long said that carbonated sweetened drinks are highly price elastic, meaning that price increases depress volume." The article describes how Coke, Pepsi and others raised their prices after Labor Day in 2008, and how an 8% increase in prices leads, in general, to decreased consumer demand by even more than 8%.
 - b. The beverage industry is fond of citing a recent study by RAND researcher Rolland Sturm that looks at the impact of current state sales taxes for soda on children's consumption, which found little effect of sales taxes averaging 4%. Of course sales taxes don't reduce consumption because consumers don't see a difference in price when they are deciding what to buy. The sales tax on soda is hidden in the total sales tax on the purchase receipt, which they don't even get until after they make their purchase. This points to the clear imperative that soda taxes, like tobacco taxes, must be excise taxes so that the consumer sees the price difference when making a purchasing decision. AS for this paper's conclusion that soda taxes must be sufficiently high to see an effect of consumption, the tax proposed by Senator Florez of one penny per teaspoon of added sugar would be an effective tax of 15%, four times the average rate considered in this study – and it would be an excise tax, not a sales tax.
7. SEVENTH: The beverage industry will say that soda taxes won't have the desired public health benefit. This too is **FALSE**. California has proven beyond ANY doubt that taxes on harmful products can be used as a central part of a successful public health campaign. I am, of course, talking about California's world renowned tobacco tax. IN 1988, California put an excise tax of 25 cents on each pack of cigarettes. Ten years later, smoking rates had gone down 27% and lung cancer rates had gone down by almost

20%, making our lung cancer rates significantly lower than the rest of the country. The key: funds raised through the price increase were used to support directly relevant prevention programs – just as the tax proposed in SB 1210 would do.

8. EIGHTH. The beverage industry will say that consumers don't want a soda tax. This too is **FALSE**. A public opinion poll conducted by the reputable Field Research Corporation – was released just today showing that a strong majority of registered voters – 56% compared to 43% -- support a soda tax when the monies go to support childhood obesity and other children's health programs. And while the beverage industry professes to be protecting poor people by fighting the soda tax, it is EXACTLY these communities who support the tax the most: by almost a 2:1 margin.
9. NINTH: Industry will claim that government should not interfere with the market price of their product. This too is **FALSE**. The government already interferes with the price of soda by subsidizing corn and keeping the price of high fructose corn syrup artificially low. Sugar sweetened beverages represent 2/3 of all high fructose corn syrup consumed in the U.S., and you sure don't hear beverage industry arguing to end government's intrusion into market when it benefits their bottom line.
10. TENTH: Finally, industry will likely argue that efforts to reduce soda consumption will hurt business. This too is **FALSE**. I know of not a single study suggesting that either tobacco or alcohol taxes have ever resulted in job losses. Look at those states that currently have soda taxes, did they result in job losses? Once again, industry is crying wolf.

In conclusion, a soda tax would do two things. It would create a market mechanism to encourage people to buy healthy beverages instead of unhealthy beverages – the more sugar, the greater higher the price. And it funds school and community programs that we all know we need to protect our children's health – like healthier food in schools and more and better physical education in our schools. So rather than padding beverage industry profits, the funds raised by this surcharge will pay for more PE teachers, healthier school food, and a guarantee of clean water to drink in every school and community. We are talking about a \$41 billion obesity epidemic. Studies show that the beverage industry is responsible for AT LEAST 20% of the problem. A soda tax would hold the beverage companies accountable for their portion of the obesity epidemic for which they are responsible.

Not surprisingly, industry is fighting soda taxes with everything they've got. Last year, the beverage industry spent an estimated \$37 million lobbying Capitol Hill to squash consideration of a soda tax to fund health care reform. They have bought full page ads in the NY Times highlighting their recent decision to take sodas out of schools now that states across the country have banned their sale. They are putting a little more calorie information on their bottles. Pepsi has come up with its brilliant Refresh Campaign giving out \$20 million to community organizations all over the country. Talk about a brilliant PR campaign!

For decades they have been marketing their products relentlessly and it has worked. They have used Michael Jackson, Tina Turner, David Bowie, Madonna, Paula Abdul, George Michael, and

Elton John to convince us to drink their products. Coke and Pepsi have used all their creativity to compete against each other for customers, young and old. And they BOTH succeeded and as a result became the single greatest contributor to the obesity epidemic. And what's more, unlike every other source of calories – unlike solid foods – we are not satiated by the calories we drink. We drink them like they are water, but they are not. So sugar-sweetened beverages ARE unique and therefore they deserve to be the focus of the attention they have been receiving and are receiving today.

A soda tax would have twin benefits. It would decrease consumption of the single largest contributor to the obesity epidemic, and it would pay for some of the programs that we all know are needed to protect our children's health – programs that will counteract the negative impact of these products.

Sugar-Sweetened Soft Drink Tax
\$0.01 per teaspoon of sugar
12 ounce container

Beverage	Type	Manufacturer	Sugar Content	Tax
7 Up	Carbonated Soft Drink	Dr. Pepper Snapple Group	37.5	\$0.09
A & W Crème Soda	Carbonated Soft Drink	Dr. Pepper Snapple Group	45.0	\$0.11
A & W Root Beer	Carbonated Soft Drink	Dr. Pepper Snapple Group	46.5	\$0.11
AMP Energy	Energy & Vitamin Supplement	Pepsi	43.5	\$0.10
AMP Energy - Elevate	Energy & Vitamin Supplement	Pepsi	43.5	\$0.10
AMP Energy - Lightning	Energy & Vitamin Supplement	Pepsi	43.5	\$0.10
AMP Energy - Overdrive	Energy & Vitamin Supplement	Pepsi	43.5	\$0.10
AMP Energy - ReLaunch	Energy & Vitamin Supplement	Pepsi	43.5	\$0.10
AMP Energy - Traction	Energy & Vitamin Supplement	Pepsi	43.5	\$0.10
AMP Energy with Black Tea	Energy & Vitamin Supplement	Pepsi	37.5	\$0.09
AMP Energy with Green Tea	Energy & Vitamin Supplement	Pepsi	37.5	\$0.09
Arctic Shatter	Sports Drinks	Coca-Cola	22.0	\$0.05
Canada Dry Ginger Ale	Carbonated Soft Drink	Dr. Pepper Snapple Group	36.0	\$0.09
Canada Dry Green Tea Ginger Ale	Carbonated Soft Drink	Dr. Pepper Snapple Group	36.0	\$0.09
Canada Dry Tonic Water	Carbonated Soft Drink	Dr. Pepper Snapple Group	34.5	\$0.08
Cherry 7 Up	Carbonated Soft Drink	Dr. Pepper Snapple Group	39.0	\$0.09
Country Time Lemonade	Fruit Drink	Dr. Pepper Snapple Group	36.0	\$0.09
Dr. Pepper	Carbonated Soft Drink	Dr. Pepper Snapple Group	40.5	\$0.10
Dr. Pepper Cherry Vanilla	Carbonated Soft Drink	Dr. Pepper Snapple Group	25.0	\$0.06
Fruit Punch	Sports Drinks	Coca-Cola	22.0	\$0.05
G2 - Grape	Sports Drinks	Pepsi	10.0	\$0.02
G2 - Orange	Sports Drinks	Pepsi	10.0	\$0.02
G2 - Strawberry Kiwi	Sports Drinks	Pepsi	10.0	\$0.02
Gatorade G - Berry	Sports Drinks	Pepsi	22.0	\$0.05
Gatorade G - Fruit Punch	Sports Drinks	Pepsi	22.0	\$0.05
Gatorade G - Grape	Sports Drinks	Pepsi	22.0	\$0.05
Gatorade G - Ice Punch	Sports Drinks	Pepsi	22.0	\$0.05
Gatorade G - Lemon Lime	Sports Drinks	Pepsi	22.0	\$0.05
Gatorade G - Orange	Sports Drinks	Pepsi	22.0	\$0.05
Gatorade G - Strawberry	Sports Drinks	Pepsi	22.0	\$0.05
Grape	Sports Drinks	Coca-Cola	22.0	\$0.05
Green Squall	Sports Drinks	Coca-Cola	22.0	\$0.05

**Sugar-Sweetened Soft Drink Tax
\$0.01 per teaspoon of sugar
12 ounce container**

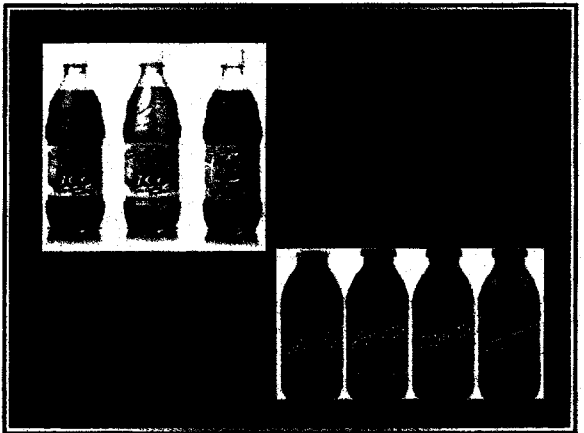
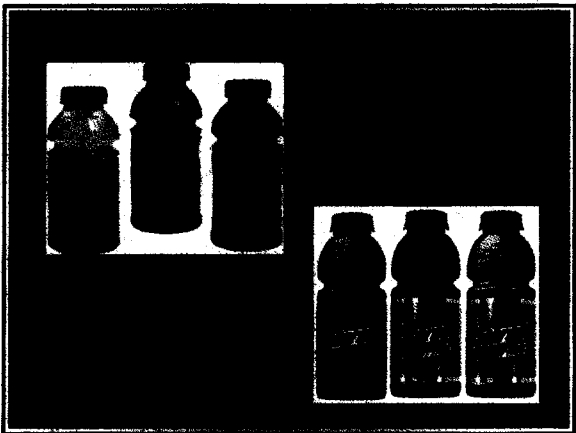
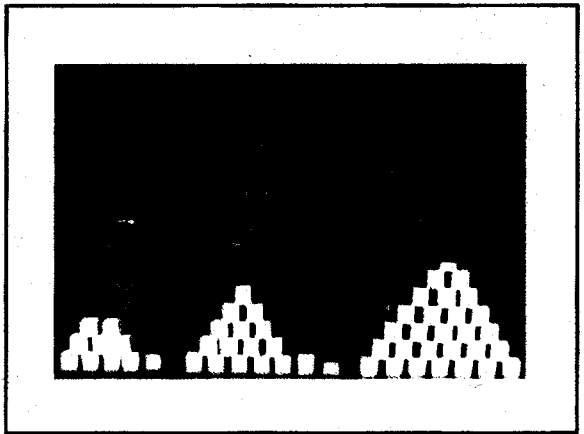
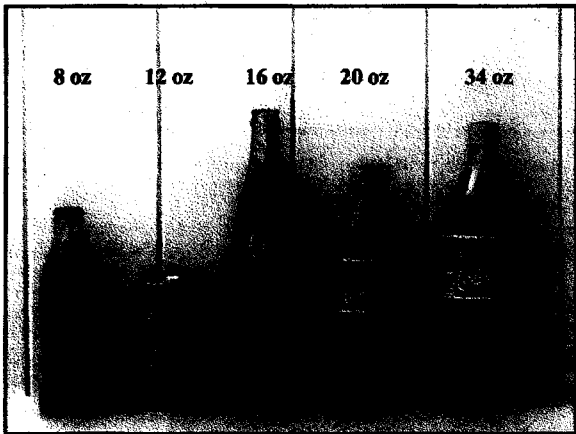
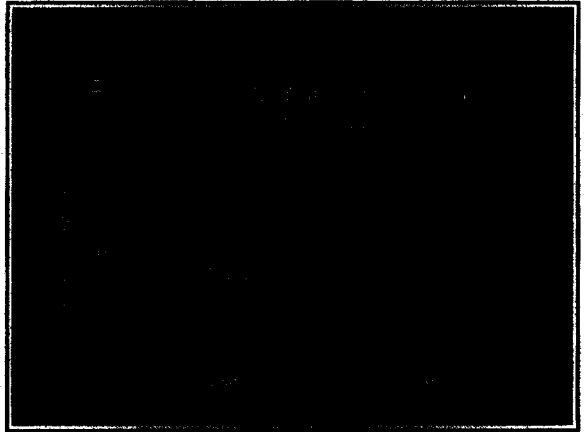
Beverage	Type	Manufacturer	Sugar Content	Tax
Hires Root Beer	Carbonated Soft Drink	Dr. Pepper Snapple Group	45.0	\$0.11
IBC Black Cherry	Carbonated Soft Drink	Dr. Pepper Snapple Group	48.0	\$0.11
IBC Cream Soda	Carbonated Soft Drink	Dr. Pepper Snapple Group	48.0	\$0.11
IBC Root Beer	Carbonated Soft Drink	Dr. Pepper Snapple Group	43.0	\$0.10
Lemon-Lime	Sports Drinks	Coca-Cola	22.0	\$0.05
Lipton Brisk - Green	Teas	Pepsi	34.0	\$0.08
Lipton Brisk - Green	Teas	Pepsi	51.0	\$0.12
Lipton Brisk - Lemon	Teas	Pepsi	33.0	\$0.08
Lipton Brisk - Lemon	Teas	Pepsi	49.5	\$0.12
Lipton Brisk - Raspberry	Teas	Pepsi	35.0	\$0.08
Lipton Brisk - Raspberry	Teas	Pepsi	52.5	\$0.13
Manzanita Sol	Carbonated Soft Drink	Pepsi	42.0	\$0.10
Mountain Blast	Sports Drinks	Coca-Cola	22.0	\$0.05
Mountain Dew	Carbonated Soft Drink	Pepsi	46.0	\$0.11
Mountain Dew (Caffeine Free)	Carbonated Soft Drink	Pepsi	46.0	\$0.11
Mountain Dew Code Red	Carbonated Soft Drink	Pepsi	46.0	\$0.11
Mountain Dew Live Wire	Carbonated Soft Drink	Pepsi	46.0	\$0.11
Mountain Dew Voltage	Carbonated Soft Drink	Pepsi	46.0	\$0.11
Mug Cream Soda	Carbonated Soft Drink	Pepsi	47.0	\$0.11
Mug Root Beer	Carbonated Soft Drink	Pepsi	43.0	\$0.10
No Fear	Energy & Vitamin Supplement	Pepsi	49.5	\$0.12
No Fear Bloodshot	Energy & Vitamin Supplement	Pepsi	34.5	\$0.08
No Fear Motherload	Energy & Vitamin Supplement	Pepsi	49.5	\$0.12
Orange	Sports Drinks	Coca-Cola	22.0	\$0.05
Pepsi	Carbonated Soft Drink	Pepsi	41.0	\$0.10
Pepsi (Caffeine Free)	Carbonated Soft Drink	Pepsi	41.0	\$0.10
Pepsi Natural	Carbonated Soft Drink	Pepsi	38.0	\$0.09
Pepsi Wild Cherry	Carbonated Soft Drink	Pepsi	42.0	\$0.10
Propel Fit Water - Berry	Sports Drinks	Pepsi	3.0	\$0.01
Propel Fit Water - Grape	Sports Drinks	Pepsi	2.0	\$0.00
Schweppes Dry Grape Ginger Ale	Carbonated Soft Drink	Dr. Pepper Snapple Group	37.5	\$0.09
Schweppes Ginger Ale	Carbonated Soft Drink	Dr. Pepper Snapple Group	33.0	\$0.08

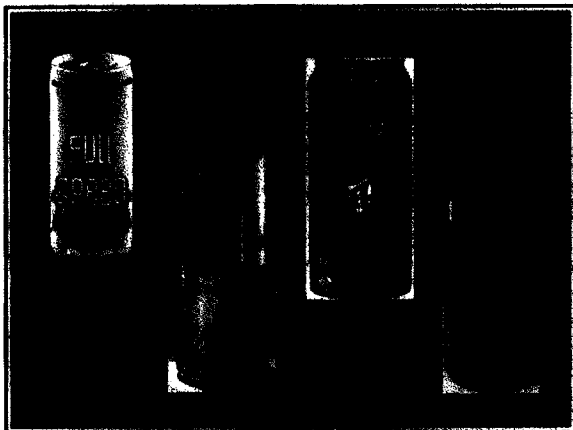
**Sugar-Sweetened Soft Drink Tax
\$0.01 per teaspoon of sugar
12 ounce container**

Beverage	Type	Manufacturer	Sugar Content	Tax
Schweppes Raspberry Ginger Ale	Carbonated Soft Drink	Dr. Pepper Snapple Group	37.5	\$0.09
Schweppes Tonic Water	Carbonated Soft Drink	Dr. Pepper Snapple Group	33.0	\$0.08
Sierra Mist	Carbonated Soft Drink	Pepsi	39.0	\$0.09
Sierra Mist Cranberry Splash	Carbonated Soft Drink	Pepsi	42.0	\$0.10
Slice - Grape	Carbonated Soft Drink	Pepsi	50.0	\$0.12
Slice - Orange	Carbonated Soft Drink	Pepsi	48.0	\$0.11
Slice - Peach	Carbonated Soft Drink	Pepsi	50.0	\$0.12
Slice - Strawberry	Carbonated Soft Drink	Pepsi	43.0	\$0.10
Snapple Antioxidant Water-Grape Pomegr	Teas	Dr. Pepper Snapple Group	19.5	\$0.05
Snapple Black Tea Earl Grey	Teas	Dr. Pepper Snapple Group	12.0	\$0.03
Snapple Green Tea Citrus Fusion	Teas	Dr. Pepper Snapple Group	33.0	\$0.08
Snapple Green Tea Original	Teas	Dr. Pepper Snapple Group	22.5	\$0.05
Snapple Lemon Iced Tea	Teas	Dr. Pepper Snapple Group	34.5	\$0.08
Snapple White Tea Nectarine	Teas	Dr. Pepper Snapple Group	22.5	\$0.05
SoBe Adrenaline Rush	Energy & Vitamin Supplement	Pepsi	49.5	\$0.12
SoBe Energy	Teas	Pepsi	40.5	\$0.10
SoBe Lean Diet Energy	Teas	Pepsi	1.5	\$0.00
SoBe Nirvana	Teas	Pepsi	43.5	\$0.10
SoBe Power	Teas	Pepsi	40.5	\$0.10
SoBe Yumberry Pomegranate	Teas	Pepsi	37.5	\$0.09
Strawberry Lemonade	Sports Drinks	Coca-Cola	22.0	\$0.05
Sunkist Grape Soda	Carbonated Soft Drink	Dr. Pepper Snapple Group	52.5	\$0.13
Sunkist Orange Soda	Carbonated Soft Drink	Dr. Pepper Snapple Group	49.5	\$0.12
Sunkist Pineapple Soda	Carbonated Soft Drink	Dr. Pepper Snapple Group	49.5	\$0.12
Tropicana Twister Soda - Grape	Carbonated Soft Drink	Pepsi	50.0	\$0.12
Tropicana Twister Soda - Orange	Carbonated Soft Drink	Pepsi	52.0	\$0.12

* based on 4.2 grams of sugar per teaspoon

Sources: <http://www.drpeppersnapplegroup.com/brands/>
http://www.thecoca-colacompany.com/us_nutrition.html
<http://www.pepsiproductfacts.com/infobycategory.php>





- > Kids: 175 calories / day
- > Adults: 50 gallons / yr
39 pounds of sugar

- > Centers for Disease Control
- > US Department of Agriculture
- > Institute of Medicine
- > American Academy of Pediatrics
- > American Medical Association
- > American Heart Association
- > World Health Organization

Many contributors to obesity	TRUE
Less SSBs won't solve obesity	TRUE
SSBs are insignificant	FALSE
All calories are equal	FALSE
The science is unclear	FALSE
Tax won't lower consumption	FALSE
Tax won't improve health	FALSE
Consumers oppose SSB tax	FALSE
Don't interfere with free market	FALSE
People will lose jobs	FALSE

- 1) Funding for Obesity Prevention
- 2) Hold beverage companies responsible for their portion of obesity epidemic

BEVERAGE DIGEST

Volume 53, No. 11

Bedford Hills, NY

November 21, 2008

Coke, PepsiCo, Dr Pepper Snapple, Red Bull, Nestlé, PBG, Coke Consolidated and Other Key Companies on Program For December 15th Conference in NYC. Jeff Dunn, CEO of Bolthouse and Former Head of Coke North America, Joins Program.

BD will hold its Future Smarts conference Monday, December 15, 2008 at the Crowne Plaza Hotel in NYC. The most recent addition to the program is: Jeff Dunn, CEO of Bolthouse Farms juice company and former head of Coke North America. Current speakers listed below. Reservation form enclosed.

PepsiCo	Indra Nooyi	Chairman and CEO
Coca-Cola Co	Joe Tripodi	Chief Marketing and Commercial Officer; Senior VP
Dr Pepper Snapple Group	Larry Young	CEO
Coca-Cola Co; Allen & Co	Don Keough	Coke Director and Former President; Chairman Allen & Co
Nestlé Waters North America	Kim Jeffery	CEO
Coke Consolidated	Bill Elmore	President and COO
Pepsi Bottling Group	Yiannis Petrides	President European Business
Red Bull	Selim Chidiac	CEO Red Bull North America
Morgan Stanley	Bill Pecoriello	Beverage Analyst
HyDrive	Mike Weinstein	CEO
BAWLS Guarana	Hoby Buppert	CEO
Bolthouse Farms	Jeff Dunn	CEO

Coke System Is Working on New Supply Chain Company. Big Savings Seen. Pepsi Faces Same Issues.

Multiple sources say Coke, CCE and other Coke bottlers are in talks about formation of a new supply chain company for the North American system (BD 11/7/08). With volume declining, it has become incumbent for both the Coke and Pepsi systems to find ways to wring out costs and increase efficiencies. Sources say evolution of new supply chain structure for Coke system may come in several steps, with ultimate goal being one company to handle production and logistics for North American Coke system. First step could be formation of new company managing transportation and logistics. Second step, say sources, could be transfer of assets to new company by Coke, CCE and possibly Coke Consolidated. Assets would include Coke's hot-fill and fountain syrup production plants and bottlers' cold-fill plants. Third step, if this can be accomplished, would be adding assets of other bottlers. Second attempt. This would be the system's second attempt at creating this kind of structure. In early 2001 (BD 3/30/01), Coke and its bottlers explored formation of a bottler cooperative for production and logistics. That became Coca-Cola Bottler Sales/Service, but it ended up just focusing on logistics; system assets were not transferred to it, and it never became a production entity.

In this issue ...

- Coke System Working On New Supply Chain Company.
- Hyatt Is Installing Machines to Bottle Water in Returnable Glass Bottles.
- Complexity in Australia: Lion Nathan Makes Bid for Coke Amatil.
- DPS Says Economy Hurts Premium Beverages. New Pepper Extension.
- 11 U.S. Markets: Big Price Increases in Some Hurt Coke Volume.
- Enhanced Waters, Sports Drinks Turn Negative in 3rd Qtr. Nestlé Attacks.
- Energy Drinks: Category Slows. Monster, Red Bull, AMP and NOS Gain.

- PepsiCo Unveils Array of New Products.
- Territory Changes, People, Briefs.
 - Coke Bottlers Team Up With Beer Wholesalers.
 - PBG to Cut 3000+ Jobs in Restructuring.

Enclosures:

- **Green Sheet:** Royal Crown U.S. Territory Map.
- **Registration Form:** Future Smarts 2008.
- **Order Form:** The Coke and Pepsi System Books.

WARNING: Unauthorized reproduction in whole or in part is a violation of Federal Law. Violators are liable for actual damages or statutory damages up to \$100,000.

CEO Rob Murray said his company would not bid for the Cadbury unit. Re Pepsi, if Cadbury (which bottles Pepsi in Australia) were to sell its business to Coke Amatil -- or to Lion Nathan owning Coke Amatil -- where would the Pepsi business end up? Informed source says that there are other avenues for Pepsi in Australia, beyond Lion Nathan and Cadbury, to maintain its presence in that market; but that source did not elaborate. Coke and Kirin. A further layer of corporate incestuousness lies in the fact that Japanese brewer Kirin owns about 46% of Lion Nathan. Kirin, according to Reuters, has agreed to invest another \$2.4 bil in Lion Nathan to help it fund the acquisition of Amatil. Kirin also owns U.S. Coke bottler Coke Northern New England. Plus, it competes vs Coke in Japanese soft drink market. Market shares. BD estimates Coca-Cola CSD brands have about a 57 all-channel share in Australia; Pepsi at about 11.4. Cadbury's CSD brands have a 16 share.

Views: 'hollow threat.' Credit Suisse Australian beverage analyst Larry Gandler confirms that Coke must approve this deal for it to go through. But he suggests that Coke not approving "is a hollow threat: How else could (Coke) have its beverages distributed in Australia if not (Amatil)?" And legal source says, "they can't realistically get to that. It would be mutually destructive." Adds, "Coke has to be reasonable, even if its (franchise contract with Amatil) doesn't say that." Gandler adds that he believes Coke wants "a soft drink CEO and chairman" to run any combined Lion Nathan/Coke Amatil business, while Lion Nathan wants its CEO and chairman to run the combined company. Morgan Stanley Australian beverage analyst Martin Yule estimates that there is "less than a fifty percent chance of Lion Nathan being successful." He adds, "this is principally because we believe that (Coca-Cola Co) would receive minimal benefits from such a transaction."

Dr Pepper Snapple Says Economy Hurting 'Premium' Beverages. Snapple Down -10%. New Dr Pepper Extension:

Dr Pepper Snapple CEO Larry Young told investors on Nov. 13 that the weakness in consumer spending "is impacting the premium end of our business, more than the core business." He said Snapple volume was down -10% in the 3rd quarter. He added, "we're quite concerned about the health" of the U.S. economy. Noting that some consumers are "trading down" to less expensive beverage, Young said that Hawaiian Punch volume in the quarter was up +20%. Overall, DPS volume was down -1% including the loss of Glaceau to Coke bottlers and up +1% excluding the Glaceau impact. Company's CSDs were up +0.5%; non-carbs up +3%. Young says in 2009, total industry LRB volume will be down -2%; CSDs down 3%-4%. DPS concentrate will be up "mid-single-digits."

Ahead. DPS's reformulated/revamped Snapple is about to launch (BD 8/28/08). Product will get new graphics and will be sweetened with sugar. Young says production set to start week of November 17. DPS is also working to expand distribution of its cold-fill Snapple, which he says should help alleviate the pricing pressure on hot-fill Snapple. However, to date, its total U.S. ACV is just 11%. Dr Pepper. In early 2009, company will launch another Dr Pepper line extension: "Dr Pepper Cherry." Will be sold in regular/diet. DPS executive says it will have a "smoother, less sharp taste" than flagship Dr Pepper. Aim is to increase U.S. penetration of Dr Pepper trademark. Note: this is not the mid-cal version of Dr Pepper that DPS has been working on (BD 5/9/08); no further word on that product at this time. Other brands. Among DPS's CSD brands, Dr Pepper +0.3% in quarter; 7UP -3%; Sunkist +3%; A&W +1%.

Elasticity: Big Price Increases Cause Coke Volume to Plummet.

CCE and several other Coke bottlers raised prices significantly after Labor Day (BD 11/7/08), and data covering 11 markets plus full U.S. shows big volume losses when prices rise above-category average (table page 4). Sources said CCE's post Labor Day price increase was about +8% overall, but higher in future consumption supermarket channel. Table shows -- for full U.S. and 11 markets -- supermarket data for the four weeks following Labor Day; comparisons are vs comparable period in 2007; in parentheses next to each market name are Coke and Pepsi bottlers.

Perspective. Industry sources have long said that CSDs are highly price elastic, meaning that price increases depress volume. In addition, consumers are facing economic pressures which likely exacerbates the elasticity pressure. Deutsche Bank recently issued a report suggesting: "Higher prices. Now really is the wrong time." Notes, "bottlers are asking consumers to 'adapt' to higher pricing as purchasing power weakens." In the U.S. where most concentrate pricing is sold on a straight or cost per unit basis -- not on an incidence model tied to wholesale/retail prices -- these kinds

Post Labor Day 2008 CSD Data for Full U.S. Plus 11 Markets

	Share	Share +/-	Vol. +/-	Pricing +/-		Share	Share +/-	Vol. +/-	Pricing +/-
Total U.S. Supermarkets					Los Angeles, CA (CCE, PBG)				
CSD Category	100.0	n/a	-7.8%	+6.8%	CSD Category	100.0	n/a	-3.8%	+7.6%
Coca-Cola	31.3	-2.5	-14.6%	+12.0%	Coca-Cola	34.1	-4.8	-15.6%	+14.8%
PepsiCo	31.4	flat	-7.9%	+5.4%	PepsiCo	24.9	+2.2	+5.3%	+3.9%
Private Label	13.0	+1.4	+3.6%	+3.2%	Private Label	3.5	+0.8	+24.8%	-7.1%
Salt Lake City, UT (Coke Swire, PBG)					Dallas/Ft. Worth, TX (CCE, PBG)				
CSD Category	100.0	n/a	+2.9%	+5.7%	CSD Category	100.0	n/a	-8.5%	+7.4%
Coca-Cola	39.6	+2.3	+9.4%	+4.2%	Coca-Cola	34.9	-4.3	-18.6%	+14.0%
PepsiCo	26.4	-1.9	-3.9%	+7.1%	PepsiCo	16.5	+1.7	+1.8%	+4.2%
Private Label	11.0	+0.1	+4.2%	+4.4%	Private Label	10.6	+1.2	+3.2%	+3.8%
Birmingham/Montgomery, AL (Coke United, Pepsi Buffalo Rock)					Atlanta, GA (CCE, PBG)				
CSD Category	100.0	n/a	-16.3%	+10.5%	CSD Category	100.0	n/a	-6.8%	+11.1%
Coca-Cola	38.7	+1.2	-13.6%	+10.5%	Coca-Cola	48.6	-3.7	-13.4%	+14.7%
PepsiCo	27.0	+1.0	-13.1%	+10.3%	PepsiCo	13.7	-0.7	-11.2%	+16.0%
Private Label	10.8	+0.7	-10.1%	+8.7%	Private Label	22.2	+4.1	+14.3%	+11.1%
Philadelphia, PA (Coke Philadelphia, PBG)					Denver, CO (CCE, PBG)				
CSD Category	100.0	n/a	-14.5%	+6.3%	CSD Category	100.0	n/a	-5.7%	+8.5%
Coca-Cola	36.7	-2.4	-19.7%	+9.5%	Coca-Cola	27.0	-2.9	-14.8%	+16.5%
PepsiCo	29.1	-0.2	-15.0%	+5.4%	PepsiCo	39.0	-0.8	-7.7%	+8.8%
Private Label	11.1	+1.5	-1.6%	+4.6%	Private Label	11.8	+1.8	+11.1%	-2.3%
Charlotte, NC (Coke Consolidated, PBG)					Detroit, MI (CCE, PBG)				
CSD Category	100.0	n/a	-9.3%	+1.2%	CSD Category	100.0	n/a	-5.2%	+1.9%
Coca-Cola	32.5	+4.4	+4.9%	+0.8%	Coca-Cola	23.0	-11.1	-36.0%	+14.4%
PepsiCo	33.5	-6.6	-24.2%	+2.4%	PepsiCo	44.3	+10.1	+22.7%	-5.1%
Private Label	8.8	+2.2	+19.9%	+0.1%	Private Label	6.7	+0.2	-2.5%	-0.3%
Oklahoma City, OK (Coke Great Plains/Browne, PBG)					Phoenix/Tucson, AZ (CCE, PBG)				
CSD Category	100.0	n/a	-12.0%	+12.6%	CSD Category	100.0	n/a	-8.9%	+9.5%
Coca-Cola	38.9	-6.4	-24.4%	+19.1%	Coca-Cola	28.7	-7.3	-27.3%	+23.4%
PepsiCo	19.3	+6.0	+27.7%	-1.9%	PepsiCo	30.1	-2.0	-14.6%	+10.6%
Private Label	4.0	+0.8	+8.4%	+8.1%	Private Label	16.0	+1.2	-1.6%	-1.3%

of results hit a concentrate company harder than a bottler. For example, for the Coca-Cola system nationally, pricing was up +12%, and volume was down -14.6%. That means bottler dollars were down about -2.5%; but Coke sold -14.6% less concentrate. Coke system executive says, "that kind of dynamic makes incidence based pricing very relevant." Adds: "Both (franchise company and bottlers) can win with a properly run incidence model."

Details. Markets on left side of table are non-CCE Coke bottlers. Coke Swire, in Salt Lake City, and Coke Consolidated, in Charlotte NC, put in moderate price increases, less than the competing Pepsi bottler. In both cases, Coke system share and volume out-perform Pepsi. In Philadelphia (where Coke operates the bottler) and Oklahoma City, Coke system pricing was up considerably more than industry average; Pepsi pricing was down or up less than industry average; and in those markets, Coke loses share, and Pepsi gains share or loses less share. Right side of table shows six markets where CCE competes with PBG. In all of these markets except Atlanta, CCE's price increase substantially higher than PBG; Coke share and volume suffer. Following month. The results for the following four weeks -- ending Nov. 2 -- are directionally the same as the results in the table. CSD volume was down -6.7%, with pricing up +7.7%. Coke's volume was down -14% with pricing up +14.2%. Pepsi's volume was down -7.9%, with pricing +7.4%. Coke lost -2.7 share points. Pepsi's share was down -0.4.

Nine-Month Non-Carbs: Most Categories Down. Enhanced Waters and Sports Drinks Turn Negative in 3rd Qtr. Nestlé Begins Attack Against Soft Drinks to Promote Its Bottled Water.

Quarterly category volume data (top table page 5) illustrates the weakening performance in the U.S. beverage business. Sports drinks turned negative in the 3rd quarter after being up modestly in the 1st and 2nd quarters; that category is now down YTD (bottom table page 5). Enhanced waters, which have been a recent growth star of the industry along with energy drinks, have also turned negative in the most recent

**Written testimony and/or additional material
submitted by:**

**Danielle Greenberg, PhD, FACN
Director of Nutrition and Scientific Affairs
PepsiCo.**

**Former Associate Research Professor of
Psychology in Psychiatry
Cornell University Medical College**

Testimony of Dr. Danielle Greenberg, PepsiCo Inc.
Before Joint Hearing: Senate Food & Agriculture and Health Committees
April 20, 2010

Good afternoon Senator Florez, Senator Alquist, and committee members. My name is Dr. Danielle Greenberg. I am a nutritionist with PepsiCo where I have worked for the past nine years. Prior to joining PepsiCo, I served on the faculty of Cornell University Medical College for 15 years, specializing in research on obesity and the control of food intake.

I want to thank you for the opportunity to be here today. We can all agree that obesity is a serious health issue. As a leading food and beverage manufacturer, PepsiCo knows we have an essential role to play in helping to find solutions to this complex problem. We have worked for decades now to reduce calories in our products and to help our consumers make healthier nutrition choices. Let me provide a few examples.

- In the past five years, the average number of calories in the beverages we sell in the United States declined by 11 percent - a significant achievement and a notable statistic against the backdrop of increasing obesity rates. To ensure this trend toward lower calories continues, PepsiCo announced a significant commitment last month - a global pledge to reduce the average amount of added sugar per serving in our beverages by 25 percent by the year 2020. We also committed to cut sodium per serving by 25 percent and saturated fat per serving by 15 percent in our food products over the same time period. And, we will report progress towards each of these goals on PepsiCo's website, ensuring transparency and accountability.
- We have also taken significant steps to help raise consumer awareness about beverage calories. For many years now, we have shown calorie counts for both full-container and 8 oz servings in the nutrition facts panels on containers smaller than 1 liter. And, we've provided calorie and nutrient information for every beverage, in every package size we sell, on our website, PepsiProductsFacts.com. Now, in support of First Lady, Michelle Obama's initiative to fight childhood obesity, we're going even further, committing to display calories on the front of all our beverage packages and on vending machines and self-serve fountain equipment. These sweeping changes are being coordinated with the White House and the FDA and will be executed over the next two years.
- The groundbreaking, calorie labeling initiative is a good example of the progress that can be made through collaborations between industry and government. At PepsiCo, we believe strongly in the power of such collaborations and work hard to find opportunities to join with our industry members, government and non-governmental agencies to pursue solutions together. For example, we collaborated with the American Heart Association, the Clinton Foundation, and other beverage industry leaders to change the mix of beverages available in U.S. schools. Through this effort, we voluntarily removed full-calorie soft drinks from schools throughout the U.S. and now offer a range of lower-calorie, smaller-portion sized drinks. In another example, PepsiCo's Chairman and CEO Indra Nooyi is playing a pivotal role in the Healthy Weight Commitment Foundation, a collaboration of more than 60 retailers, food and beverage manufacturers, NGO's and educators who are working to develop solutions to help reduce obesity, particularly among children.

**Testimony of Dr. Danielle Greenberg, PepsiCo Inc.
Before Joint Hearing: Senate Food & Agriculture and Health Committees
April 20, 2010**

We are a company that continues to encourage nutritionists and scientists like me to make a difference; a place where my colleagues and I are proud to work; and a company that seeks to partner in efforts to find common-sense solutions to complex problems like the one on the table today.

We hope that sound science and common sense will prevail as industry, governments, and non-governmental agencies work together to change American habits – not only in what we eat and drink but also in the daily physically activity needed to maintain healthy lifestyles.

In troubled economic times like these, we can understand the appeal of a tax that produces revenues and purports to promote health. However, there is no scientific or medical evidence that a beverage tax will be effective in reducing obesity. And there is ample reason to believe that such a tax would have dire economic consequences for local retailers and residents, putting hundreds of well-paying local jobs at risk, and saddling middle class, working people with another tax burden at a time when they can least afford it.

Thank you very much.

#

**Written testimony and/or additional material
submitted by:**

**Robert Achermann
Director of Government Relations
California Nevada Soft Drink Association**

**Testimony of Bob Achermann
California/ Nevada Soft Drink Association
Joint Hearing of Senate Food and Agriculture and Health Committees
April 20, 2010**

Good morning Chair and members I'm Bob Achermann of the California/Nevada Soft Drink Association. Our member bottlers produce and distribute a wide variety of non-alcoholic beverages that include carbonated soft drinks, water, juices, teas and isotonic beverages among others. Thank you for the opportunity to provide comments.

A tax on soft drinks, juices and other beverages, in our view, unfairly lays the blame for the complex problem of obesity on the consumption of one particular type of product and perpetuates the myth that taxing those products will make a difference in fighting obesity. The only 2 states with an excise tax are Arkansas and West Virginia. How are those states doing in the fight against obesity? Arkansas has the 10th highest obesity rate and West Virginia has the 3rd highest rate in the United States according to a 2009 report by Trust for America's Health and the Robert Wood Johnson Foundation.

The problem of obesity is an important one for our society to overcome. But this type of tax focuses only on soft drinks as a cause for obesity and not total diet and exercise or other possible factors, be they environmental, behavioral, or genetic, which may contribute to obesity which require much broader response than a beverage tax.

I'd like to briefly tell you about initiatives that the beverage industry has committed to in order to make an impact on the problem of obesity. In 2006, the beverage industry on a national basis changed the beverage landscape in schools over the following three years. We pulled full-calorie soft drinks out of schools. Implementation of the School Beverage Guidelines is nearly complete. We reduced calories shipped to schools by nearly 60%.

As of the beginning of this school year, we completed implementation of our Beverage Guidelines in nearly all of the 123,000 public and private schools in the United States. A final report of the complete implementation of the Beverage Guidelines will be available next month.

In 2008, the beverage industry implemented our Global Marketing Policy. The Policy applies to all our non-alcoholic beverages other than water, juices and dairy-based beverages. Our commitment is not to advertise or market other beverages to audiences primarily comprised (50% or more) of children 12 years and younger.

This advertising and marketing applies to the following media: television, radio, print, Internet, phone messaging and cinema (including product placement). It is the first industry-specific, global marketing standard of its kind.

The School Beverage Guidelines and the Global Guidelines is an example of our commitment to be responsible to our consumers and others. Another example of that is our members' participation in the Healthy Weight Commitment Foundation. The goal of the Health Weight Commitment Foundation is to help reduce obesity, particularly childhood obesity, by 2015 by helping people achieve healthy weight through energy balance – calories in and calories out.

The work of the Foundation focuses on three critical areas where people spend their time – in the marketplace, in the workplace, and in schools. In schools, the Foundation's Healthy Schools Partnership integrates nutrition education and physical education in a school-based curriculum to help children develop healthy habits. The partnership successfully piloted the curriculum in schools in Kansas City, MO and will expand to schools in other cities and tribal communities this year.

Companies in our industry also engaged in numerous health and wellness initiatives with local, state and national non-profit organizations, like the Boys and Girls Clubs of America, and the YMCA. These initiatives are designed to teach children and adults the importance of making good decisions regarding nutrition and health. The companies are also engaged in other initiatives to highlight the

importance of making informed choices, like Coke's front-of-pack labeling and Pepsi's Smart Spot program.

But we continue to step up to be part of the solution to obesity, reaching beyond America's schools. In support of First Lady Michelle Obama's "Let's Move" anti-obesity campaign, America's leading beverage companies have committed to clearly display the calories in all our beverages on the front of the can or bottle as well as on our vending and fountain machines. This Clear on Calories Initiative will provide consumers with clear and easy to understand information on their beverage choices. We are coordinating closely with FDA to implement this initiative, which will go above and beyond what is required by the agency's food labeling regulations. This means that within two years, every time consumers touch one of our beverages they will have the calorie information at their fingertips. In fact, in her remarks at the launch of her "Let's Move" initiative, the First Lady acknowledged our industry, stating, "This is exactly the kind of vital information parents need to make good choices for their kids."

The beverage industry offers a wide range of products, from zero-calorie and low-calorie sodas to 100% juices and beverages with varying ranges of calories. The fact is that today more and more Americans are drinking our no- and lower-calorie beverages than they did just 10 or 15 years ago resulting in a 21 percent decrease in the calories per ounce produced from 1998-2008 – that's across our entire product portfolio. Yet, obesity and overweight continue to be an epidemic in this country.

That's why we as an industry are working to educate consumers about the importance of living an active, healthy and balanced lifestyle. Our industry is committed to being part of the solution to the issues of obesity, particularly childhood obesity. We welcome the opportunity to continue this work with members of the committee and others to propose solutions that educate, inform, and benefit Californians of all ages and backgrounds.

In our view, a soft drink tax is not the solution to combat obesity and will only lead to higher prices for consumers, an additional tax burden for residents of the state, and potentially, lost jobs.

Thank you for the opportunity to discuss these issues with you and I'm happy to answer any questions.

**Written testimony and/or additional material
submitted by:**

**Julian Canete
Executive Director
California Hispanic Chamber of Commerce**

Testimony of Julian Canete
Executive Director, California Hispanic Chambers of Commerce

Before the Senate Food and Agriculture Committee and Senate Health Committee
April 20, 2010

Good Afternoon, Mr. Chairman and members of the Committees. My name is Julien Canete and I am the Executive Director of the California Hispanic Chambers of Commerce.

The California Hispanic Chambers of Commerce (CHCC) represents the interests of more than 720,000 Hispanic-owned businesses in the State of California. With a network of more than 65 Hispanic chamber and business associations, the CHCC is the premier and largest regional Hispanic business organization in the nation. Our members provide hundreds of thousands of jobs across the state – and are acutely attuned to the critical issues of economic development, employment, education and health care in California. The issue of obesity and how we as a state and a nation approach the challenges it presents has implications in all of these arenas.

Our members agree that obesity is a serious issue in the United States. It is one that particularly affects the Latino community.

If we're truly trying to reduce obesity, however, a tax on soft drinks and other beverages in California isn't the answer as it simply won't work. A tax will not make Californians healthier. In fact, it could have an adverse affect on small business. If you consider the two other states that have such an excise tax in place on soft drinks – West Virginia and Arkansas – you'll see that they also have among the ten highest rates of obesity in the nation.

Our communities do not want our elected officials using the tax code to tell them what to eat or drink. A couple years ago in Maine, the government imposed a tax on beverages to pay for the state health care program. Just a few months later, Maine voters rejected the tax by a two-to-one margin.

In today's economy, small business owners and retailers, just like hard-working families, are struggling – working hard to provide food and beverages at an affordable price to their customers. They need to provide food for their families at a price that they can afford. Adding the additional burden of a tax to the items in people's grocery carts hurts businesses and families alike.

Taxing certain products to solve our state's obesity problem will do nothing to teach us how to live a healthy lifestyle. A better approach to solving this problem is through widespread education about balancing our calories and getting regular exercise. The Hispanic Chambers support the work of the beverage industry in developing and implementing the national School Beverage Guidelines as part of a broader effort to teach kids about the importance of a balanced diet and exercise. The guidelines remove full-calorie sodas from all schools and provide more lower-calorie, nutritious and smaller-portion beverages. The results are reverberating through communities nationwide. In the past five years, beverage calories available in schools nationwide have dropped by 88%, while shipments of full-calorie soft drinks are down 95% in all schools.

These are the quantifiable results have a meaningful impact in schools, kids and families across the country. As California policymakers seek solutions to the obesity issue, we at Hispanic Chambers of Commerce encourage them to adopt policies that are based in both science and common sense. We simply cannot tax our way to better health.

Thank you for the opportunity to speak with you today about the serious issue of obesity.

Julian Canete

Executive Director, California Hispanic Chambers of Commerce

**Written testimony and/or additional material
submitted by:**

**Samantha Dabish
Vice President
Neighborhood Market Association**

Testimony of Samantha Dabish
Vice President of Government Relations & Community Outreach
The Neighborhood Market Association

Before the Senate Food and Agriculture Committee and Senate Health Committee
April 20, 2010

Good Afternoon, Mr. Chairman and members of the Committees. My name is Samantha Dabish, Vice President of Government Relations & Community Outreach at the Neighborhood Market Association.

The Neighborhood Market Association (NMA) is a non-profit trade association dedicated to empowering independent retailers throughout the west coast and specifically representing over 2,000 retailers in the state of California.

NMA provides representation, education, leadership, community outreach, buying power, and support to our members in order to improve their quality of life and facilitate prosperity in the neighborhoods they serve.

NMA serves all independent retailers and strongly believes that they are the cornerstones of the entrepreneurial spirit, and the backbone of the economy. This innovative spirit and drive comes from many family operated independent businesses that employ over 20,000 people. NMA proudly represent them and we do everything we can to help them succeed. That is why I am here to speak to you today, as the voice of the independent retailer.

Taxing soft drinks, juices and other non-alcoholic beverages alone will not solve the very serious and complex issue of obesity. However, in this very tough economic climate, it will only make it tougher for consumers and retailers to make ends meet. Not to mention, we are concerned with any tax proposal that would require retailers and the clerks they employ to calculate a tax based upon the amount of sugar in a beverage – a nearly impossible task that would be both burdensome and costly.

Many consumers rely heavily on the members of the Neighborhood Market Association to provide them with their basic food staples and refreshment beverages that they enjoy. A beverage tax will increase costs to retailers and consumers and cause severe economic hardship to independent retailers across the state, which can very realistically result in the loss of jobs. Further, consumers can't afford another tax on beverages on top of the CRV and sales tax.

The majority of our customers don't want a tax on their juice drinks, soda and teas. They understand that we can't tax our way to better health, and nobody wants government in their grocery cart or at their kitchen table. California families already are struggling in this difficult economy. There could not be a worse time to ask them to pay more for the products they consume by raising their grocery prices.

If we as Californians really want to have a significant effect on the state's obesity rates and financial challenges, we need to look at comprehensive solutions that will truly have an impact on our citizens, not simplistic approaches targeting one portion of the items in our grocery cart for additional taxation.

On behalf our California's independent retailers, thank you for the opportunity to express our concerns about addressing obesity without causing unnecessary harm to our industry.

Sincerely,
Samantha Dabish

**Written testimony and/or additional material
submitted by:**

**Michael I. Goran, PhD
Professor of Preventive Medicine, Physiology
& Biophysics and Pediatrics
The Dr. Robert C. and Veronica Atkins
Endowed Chair in Childhood Obesity
and Diabetes
Keck School of Medicine
University of Southern California
Director of USC Childhood Obesity Research
Center**

Main Points for the Senate Health Panel Hearing (biological/metabolic effects of sugar)

1. Consumption of sugar is high in obese Hispanic youth – higher sugar intake in this population is the unique dietary variable associated with poor beta-cell function in the pancreas
2. Sugary drinks are the “delivery vehicle” for large doses of sugar, ~50g of sugar per typical beverage; liquid candy
3. The increased consumption of sugar is constantly challenging the pancreas to secrete more and more insulin;
4. Insulin response to glucose increases exponentially with progressive insulin resistance; obesity, puberty, ethnicity are all additive effects that make you more insulin resistant and therefore require exponentially more insulin to be released from the pancreas
5. High sugar intake and fructose in particular leads to fat accumulation in liver and associated with other metabolic complications like dyslipidemia
6. Epidemiological studies support the notion of a link between increased sugar/soda and increased body weight
7. Controlled feeding studies under laboratory conditions (clinical trials) are the best way to examine the effects of diet and diet components as opposed to relying on observational studies because of the difficulty in determining diet and sugar content of foods
8. In controlled feeding studies you can basically reproduce the “metabolic syndrome” in humans (ie insulin resistance, higher Tgs and lipids, and fatty liver) by increased sugar consumption and fructose in particular
9. Fructose is metabolized very differently than glucose – different site and mechanism of absorption in the GI system, does not elicit an insulin response (so less well regulated) and metabolized almost entirely in the liver where it can be converted to new fat production. Also affects different parts of the brain – fructose does not seem to affect appetite regulation in the same way (may explain poor satiating effects of sugar)
10. There is substantially more fructose in sugary beverages (~30g) than in fruit (~15g in an apple) and fruit delivers other health benefits like fiber, antioxidants etc.
11. The fructose content of foods/drinks has gone unchecked because it is not disclosed on food label. The label says “sugars” which is a generic term. Like grouping all fats together. Doesn't make sense since the different sugars are handled differently and have different effects on the body.
12. The negative effects of high sugar are reversible but difficult to achieve with education and behavioral intervention alone. In our prior study at USC CORC in obese Hispanics – after 16 weeks of weekly nutrition education focused on sugar reduction there were no overall effects on sugar reduction or metabolic outcomes. So even with the best knowledge and nutrition education, its not enough. Too many other overpowering factors at play such as intrinsic motivation to change, peer pressure, marketing, availability
13. However, even though there was no overall group change, there was a subset that did reduce sugar by an average of 50g/day (1 soda), those were the ones who got the metabolic improvements, including a reduction in insulin response to a standard glucose challenge. Almost like if you reduce your sugar consumption you can dampen down the revved up pancreas.
14. Summary – sugar has detrimental effects on the body at various organs (pancreas, liver) with fructose having more damaging effects. The effects are reversible but difficult to achieve through behavior and education alone.

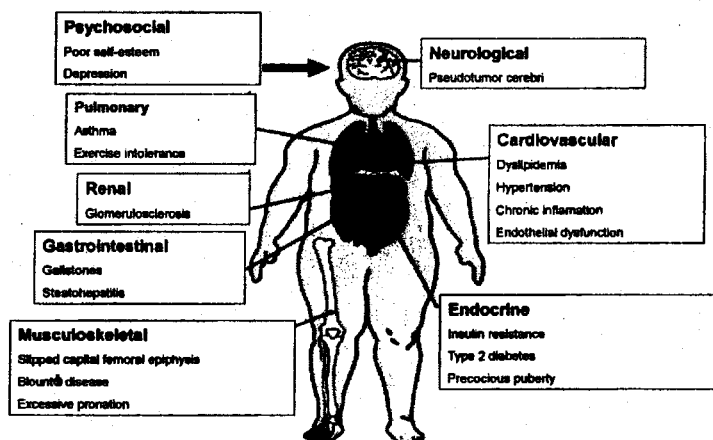
Metabolic Outcomes and Health Risks Associated with Sugar Intake

Michael I Goran, PhD

Professor, Preventive Medicine; Physiology & Biophysics; and Pediatrics
Director, Childhood Obesity Research Center
Endowed Chair in Childhood Obesity & Diabetes
Keck School of Medicine, USC

www.GoranLab.com

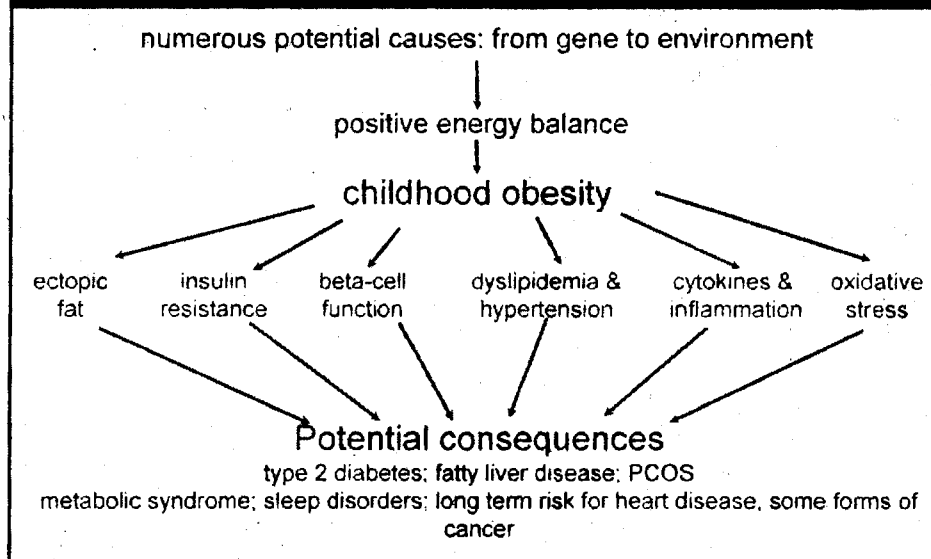
Obesity Affects Multiple Organ Systems - Even in Children



KECK
SCHOOL OF MEDICINE
USC

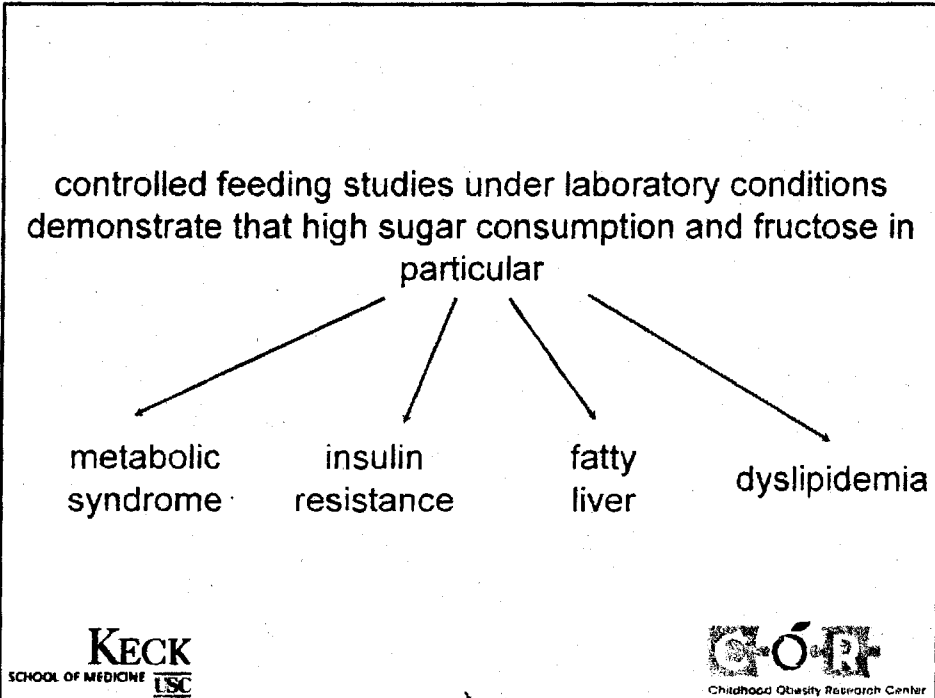
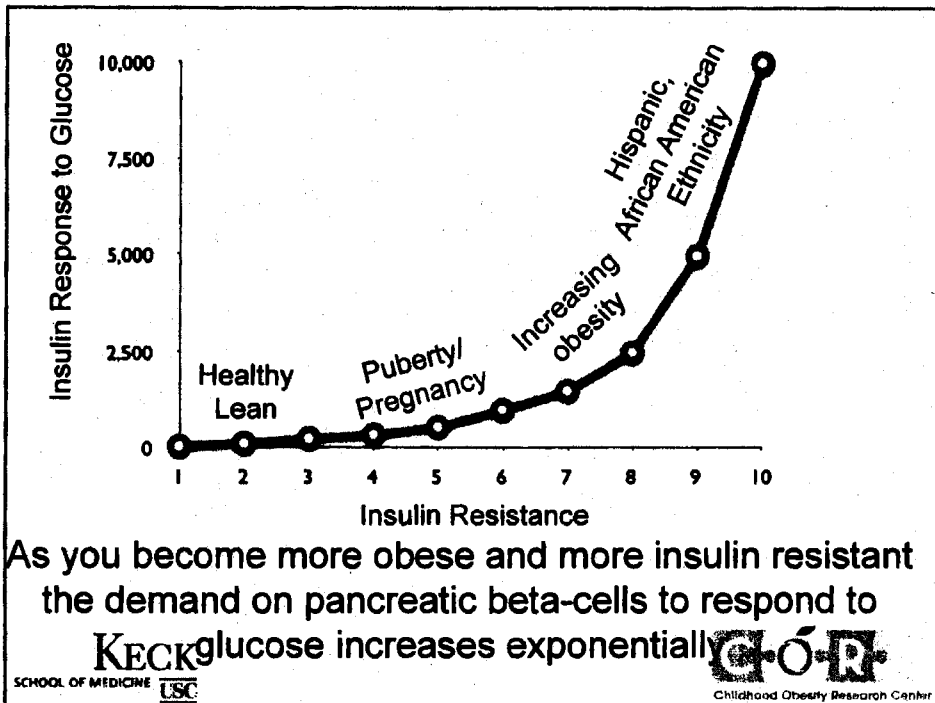
CO-R
Childhood Obesity Research Center

Consequences of Childhood Obesity

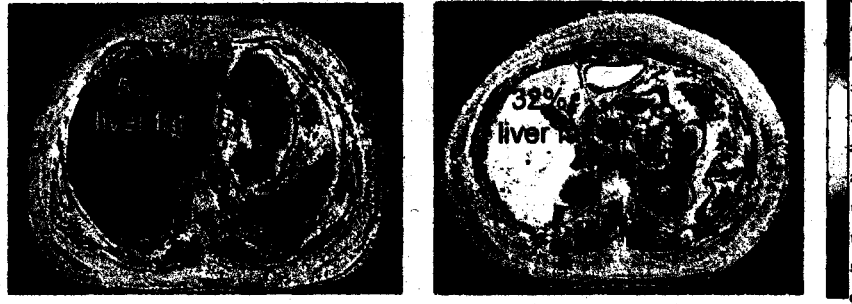


Sugar Intake in Hispanic Children

- Very high
- The sole dietary variable associated with poor beta-cell function, a marker of increased risk for type 2 diabetes



Increased Body Fat Comes in Many Shapes and Sizes: Liver Fat in 2 Obese Hispanic Females



KECK
SCHOOL OF MEDICINE USC

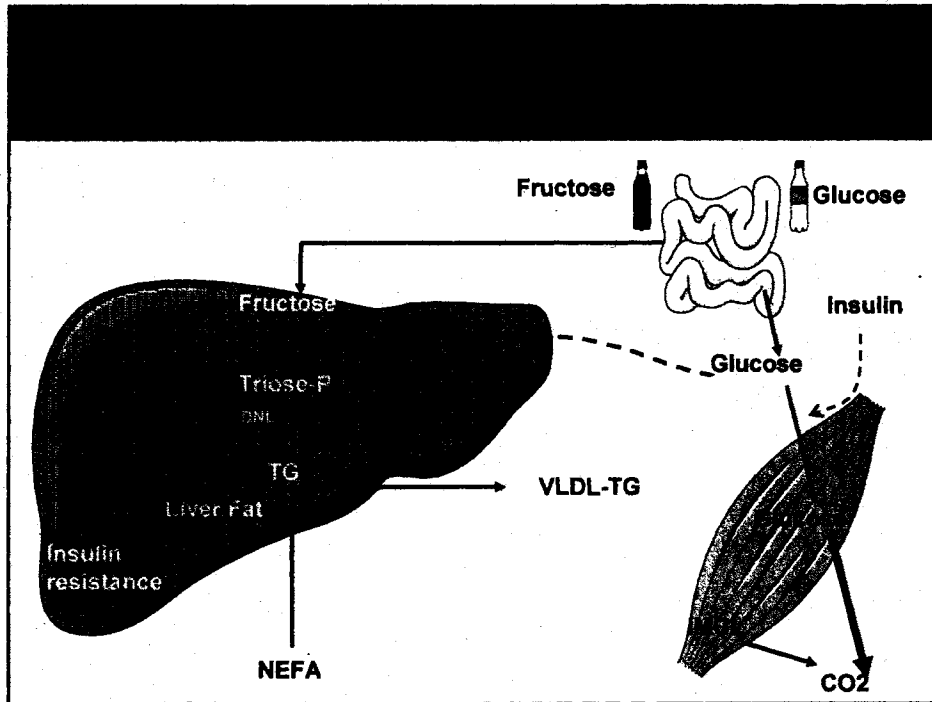
COB
Childhood Obesity Research Center

Fructose versus Glucose

- Glucose and fructose are structurally very similar but functionally very different sugars
 - Fructose is much sweeter
 - has a specific absorption in the gut; in high doses can get fructose malabsorption with GI symptoms
 - it is metabolized almost entirely in the liver where it can be a substrate for new fat synthesis in the liver
 - does not stimulate insulin release therefore less well regulated

KECK
SCHOOL OF MEDICINE USC

COB
Childhood Obesity Research Center



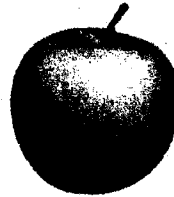
Fructose versus Glucose

- Sucrose (table sugar) is a glucose connected to a fructose
- High fructose corn syrup is a processed mixture of glucose and fructose that has been converted to fructose - typically 55% fructose/45% glucose; the actual content in foods is not on label
- Our studies show that popular soft drinks are 65% fructose/35% glucose

Fructose versus Glucose in Foods



50g sugar
28g Fructose/22g Glucose
or in 65:35 HFCS
33g Fructose/17g Glucose



15g Fructose
+
other dietary benefits
fiber, antioxidants

KECK
SCHOOL OF MEDICINE **USC**

C-O-R
Childhood Obesity Research Center

actual fructose content in foods has gone unchecked
because it is not disclosed on the label

Label says "sugars" which is a generic term which is not
true since not all sugars are equal

Like saying "fat content" without breaking down fat into its
different types

KECK
SCHOOL OF MEDICINE **USC**

C-O-R
Childhood Obesity Research Center

Are Effects of Sugar Reversible?

- Yes, but difficult to achieve with education and behavioral intervention alone
- No overall improvement after 16 weeks of intense intervention focussed on sugar reduction in obese Hispanic teens
- Too many other overpowering factors at play: motivation, peer pressure, marketing, availability, cultural norms
- In a sub-set that did improve sugar intake there were notable metabolic/health benefits

Summary

Sugar has detrimental effects on the body at various organs
(pancreas/liver)

Fructose has more damaging effects and levels of its intake are
very difficult to determine

Effects of high sugar intake are reversible but difficult to achieve
through behavior and education alone

Need a combined approach involving education, policy,
environment etc

**Written testimony and/or additional material
submitted by:**

**Lisa Katic
Registered Dietician
Principle
K Consulting**

Good afternoon ... My name is Lisa Katic. I am a registered dietitian and Principal of K Consulting – a practice based in Washington, DC specializing in food policy, communications and education. I have been working with the food and agricultural industries for over 15 years on complicated issues like overweight and obesity. I have thought about and been involved in developing strategies to combat obesity for most of my career.

What I have seen happen over the past five years with respect to new programs and strategies being implemented to address this problem is remarkable. Government, industry, schools, communities and health professionals are all collaborating on these programs to curtail the rates of obesity in America.

The most recent program we all now know about is First Lady Michelle Obama's *Let's Move* anti-obesity campaign. Launched in February 2010, this program has all the right parts and appropriately targets all the right audiences. Mrs. Obama's campaign does what no other comprehensive government program has done before. It involves every one of the President's cabinet members from the US Departments of Agriculture and Health and Human Services to Housing and Urban Development and even the Department of Labor. To me, this was the first clue that Mrs. Obama and the President really understand the complicated nature of the obesity problem and are enacting serious initiatives to try to solve it.

The First Lady has also involved for the first time **PARENTS**. The First Lady knows that parents play a key role in making healthy choices for their children and teaching their children to make healthy choices for themselves. But in today's busy world, this isn't always easy. So the **Let's Move** campaign will offer parents the tools, support and information they need to make healthier choices for their families.

The Administration, along with partners in the private sector and medical community, will:

Empower Consumers:

By the end of this year, the Food and Drug Administration will begin working with retailers and manufacturers to adopt new nutritionally-sound and consumer-friendly front-of-package labeling. This will put us on a path towards 65 million parents in America having easy access to the information needed to make healthy choices for their children.

Already, the private sector has responded:

In February 2010, The American Beverage Association announced that its member companies will voluntarily put a clear, uniform, front-of-pack calorie label on all of their cans, bottles, vending and fountain machines within two years. The label will reflect total calories per container in containers up to 20 ounces in size. For containers greater than 20 ounces, the label will reflect

a 12 ounce serving size. While more work remains to be done, this marks an important first step in ensuring parents have the information they need to make healthier choices

A second cornerstone of the ***Let's Move*** Campaign is:

Provide Parents with a Rx for Healthier Living:

The American Academy of Pediatrics, in collaboration with the broader medical community, will:

- educate doctors and nurses across the country about obesity,
- ensure they regularly monitor children's BMI,
- provide counseling for healthy eating early on, and,
- for the first time ever, will even write a prescription for parents laying out the simple things they can do to increase healthy eating and active play.

These are just a couple of examples of what our First Lady is doing to address our current obesity problem.

Another landmark initiative that was been launched in February 2009 was the ***Alliance for a Healthier Generation'*** healthcare initiative. As you may recall, the ***Alliance for a Healthier Generation'*** was launched in May 2005 and is partnership between the William J. Clinton Foundation and the American Heart Association to create a healthier generation by addressing

one of the nation's leading public health threats -- childhood obesity.

The **Alliance** has partnered with the healthcare community including hospitals, insurance companies and medical professionals to take major steps to provide a holistic approach to reduce childhood obesity in the US. This **Initiative** will:

- enable healthcare providers to be an active part of the solution to the obesity epidemic by providing children with primary-care visits and visits to registered dietitians (RDs) as part of their health insurance benefits. This is what the American Dietetic Association and members like myself have been advocating about for years, which is direct access and reimbursement for dietitians services and we are finally seeing some progress
- During the first year of the Alliance's Healthcare Initiative, nearly one million children had access to this benefit. The long-term goal of the **Alliance's** Healthcare Initiative is that within the first three years, 25 percent of all overweight children (approximately 6.2 million) will have access to this care.
- The **Alliance** will continue to recruit health insurance companies and employers to participate in this effort.

I spent a most of my time today talking about these programs because for the first time since we have been discussing ways to combat obesity we are seeing implementation of real programs

that are delivering meaningful results. This program will allow children and their families to have access to these preventive medical services in most regions of the country. Looking forward, the *Initiative* represents a tremendous opportunity to bring the best science behind what is effective in the prevention and treatment of childhood obesity to those in greatest need.

I should also mention that one of the pillars of the Alliance's efforts is the Healthy Schools initiative, which was the first to be implemented. I am sure you are aware of the School Beverage Guidelines, which was a landmark partnership between the Alliance and the beverage industry. When everyone first heard of this agreement they didn't believe it would happen. Now the industry has stood by their commitment with the *Alliance* and removed full calorie soft drinks from all schools. The industry has changed the landscape in schools across the country and decreased available calories in schools by 88%. Dietitians across the country were in disbelief, but fully congratulated the industry for standing by their commitment and helping children to have lower-calorie options and smaller portion sizes in beverages served in schools.

In closing, I want to say that I am encouraged for our future and the health of this country when I read about and am involved in so many efforts that are addressing our health problems. I only scratched the surface today and focused on programs that are showing real results, but there are several more. In order to turn the tide on this serious and complex problem, let's do what we

know works and that is focus on families, provide access to effective care and move away from banning and/or taxing food for the sake of trying to say we are doing something. Sometimes doing just anything can have real unintended consequences and takes our attention away from achieving real results.

When families work together to improve health and have access to the right tools to do so, everyone benefits.

Thank you for the opportunity to share this information with you today and I look forward to answering any questions.

**Written testimony and/or additional material
submitted by:**

**Katie Woodruff, MPH
Deputy Director
Berkeley Media Studies Group**

**THE JOINT HEARING OF THE SENATE FOOD AND AGRICULTURE
COMMITTEE AND THE SENATE HEALTH COMMITTEE
“Food & Beverages: Strategies to Recoup the Health Costs of
Excessive Sugar Consumption”
April 20, 2010**

**TESTIMONY OF
KATIE WOODRUFF, MPH
DEPUTY DIRECTOR, BERKELEY MEDIA STUDIES GROUP
A project of the Public Health Institute**

For the last 16 years, we at the Berkeley Media Studies Group have been studying public health issues in the media. Over the last several years we have been investigating how soda and other sweetened beverages are marketed, especially to young people -- and our concerns are growing.

Sweetened beverage companies would have you believe that their products should not be singled out from all the other foods and beverages that we Americans consume every day. But in fact, in addition to the research you heard earlier on soda's unique contribution to diabetes and obesity, I'm here to testify to the soda companies' extensive marketing expenditures and practices, which put them far beyond any other food or beverage category in terms of aggressive, incessant promotion of their products to young people.

Much of this marketing happens outside of parental control or even awareness, which makes it even more problematic.

I've been asked today to give an overview of beverage marketing practices, to describe how sugar-sweetened beverage companies target young people, and to show just a few examples of their marketing campaigns.

MARKETING OVERVIEW

To understand the impact of marketing of sugar-sweetened beverages on youth, we need to understand how broad the marketing function is. Marketing includes not just promotions, such as TV ads and digital campaigns, but the development and packaging of new products, the pricing of those products, and the ubiquitous access to places where those products can be purchased. In all these areas, the landscape has changed significantly in recent years.

First, the range of product offerings has broadened significantly. Where formerly beverage companies sold a few flagship soft drinks, now their offerings have expanded to include a wide variety of sweetened beverages: energy drinks, sports drinks, sugared teas, flavored waters, and more -- truly, as Coca Cola notes, a “world of choices.” These

alternatives are on the rise, but full-sugar sodas like Coke and Pepsi are still the giants, accounting for 70% of the US non-alcoholic beverage market.

Secondly, these companies have increased their portion sizes dramatically. A single soda serving used to be 8 oz; now 20 oz is the default. People tend to consume whatever's in the single container, so as the package volume has gone up, sugar intake has gone up.

At 7-11's soda fountain, the *smallest* cup size now available is 16 oz., double the recommended 8 oz serving size. And that 16 oz cup looks downright modest next to the Big Gulp, Super Big Gulp, Extreme Gulp, and the Double Gulp, which when filled with full-calorie soda contains 48 teaspoons of sugar.

On the pricing front, research has shown that over the last couple of decades the price of soft drinks hasn't changed much, while many other food categories, including fruits and vegetables, have become relatively more expensive. At my local Safeway, Coke products are on sale for \$3.33 for a 12-pack – that's \$0.28 cents a can, ounce for ounce, cheaper than the cheapest milk.¹

Does this low pricing encourage soda consumption? Certainly the industry seems to think so; an industry trade publication reported that when prices of Coca-Cola increased by 12%, sales dropped by 14.6%.² This and other research on the price elasticity of soda indicates that demand for sweetened beverages is quite price sensitive; this suggests that the proposed excise tax on sugar-sweetened beverages may be one of the most effective population-wide obesity prevention strategies we could enact.

And then there are the promotions, which are aimed at developing and reinforcing positive associations with the brand. It's important to realize that the purpose of corporate marketing is not only to sell products now, but to develop customers for life. Marketing may influence children to develop positive feelings about a branded beverage before they even get a chance to taste it, leading to the industry's dream achievement: "cradle-to-grave brand loyalty." To make this happen, beverage marketers reach out to children constantly, starting when they are very young.

MARKETING TO YOUTH

Our brief goes into significant detail about beverage marketing to youth, so I'll just highlight a few key facts for you.

- **Sugar-sweetened beverage companies lead the food and beverage industry in marketing to youth. They spent almost \$500 million dollars marketing just to children and adolescents in 2006 alone. That's half a billion dollars, well over 1 million dollars a day, targeting youth in one year.**³

¹ (2.3 cents per ounce vs 2.4 cents per ounce for milk.)

² "Elasticity: big price increases cause Coke volume to plummet." Beverage Digest. November 21, 2008:3-4.

³ Marketing Food to Children and Adolescents, a Federal Trade Commission study of expenditures and activities by 44 F&B companies, released July 2008. Covers only 2006, the year before soda companies announced self-regulatory agreements.

- Beverage companies spend far more on “new media” – digital marketing – than any other food or beverage category does. These are **their fastest-growing marketing techniques.**

What do all these marketing dollars go towards? Well, TV ads are still the single biggest marketing expense for the sweetened beverage industry.

However, beverage companies are buying less air time: they spent less on TV ads in 2007 than in 2006⁴, and we expect this trend to continue.

In recent years, the beverage companies have pledged that they won't advertise their sweetened products on children's TV, by which they mean programs where kids under age 12 make up half or more of the audience. But this doesn't mean they've given up much ground. SpongeBob SquarePants is the #1 children's TV program, and Coke won't advertise during it; but almost twice as many children watch American Idol as watch a typical episode of SpongeBob.

Coke pays \$35 million a year just to co-sponsor American Idol and get their Coke cups on the Idol judges' table. That kind of product placement is outside the traditional TV advertising budget and doesn't violate the industry's narrow self-regulatory guidelines on marketing to kids, but clearly they are reaching millions of American children this way.

DIGITAL MARKETING

At the same time as they reduce TV ad buys, sugar-sweetened beverage companies are dramatically *increasing* their efforts in the digital marketing realm. These are interactive promotions on web sites and via cell phones and text messages, which are far cheaper than buying TV time. Again, given the unique contribution of sweetened beverages to diabetes and obesity, this industry's efforts to reach young people through ubiquitous digital marketing is alarming. Just a few examples:

- Coca Cola's TWIST/TXT/GET program places a reward code inside every bottle cap of Coke and Sprite. People text the code to the company and in return get “rewards” – ringtones, screensavers, video clips, etc – sent to their cell phone. Of course, the company gets a database of cellphone numbers of their customers, who they can and do contact several times a month. The company says this is a critical part of their effort to QUOTE “establish an omnipresent, on-the-go, everywhere relationship with teens.”⁵
- Mountain Dew's DEWMOCRACY campaign has encouraged its youthful fan base to become co-creators of the brand, and to promote it among their friends. Touted as a participatory form of consumer empowerment, the campaign

⁴ TNS Media Intelligence

⁵ Mark J. Greatrex, senior vice president for marketing communications and insights at Coca-Cola. Quoted in NYT June 2007 <http://www.nytimes.com/2007/06/07/technology/07sprite.html>

mobilizes fans to vote for the next new product flavor, as well as put their creative talents toward creating user-generated ads that will play on the website.

- Taking the voting theme in a different direction is Pepsi's "Refresh Everything" campaign, which awards grants of between \$5000 and \$25,000 to projects "that will have a positive impact" in their community. Winning projects are chosen based on user votes, and people are encouraged to come back online to support their favorites up to 10 times a day. This kind of cause marketing can help companies increase their social networking engagement with consumers -- Pepsi says it doubled its Facebook fans in a single this year, as a result of the campaign. And since you have to register an email address in order to vote, it's a great way to build databases of interested consumers. As an article in *Advertising Age* points out, this type of social engagement effort is effectively "free market research that results in more effective advertising campaigns": by tallying votes on the proposed projects, Pepsi is "encouraging consumers to define what's meaningful to them, so marketers can reach out to them in meaningful ways."⁶

Again, Pepsi has claimed to reduce its advertising to youth but certainly this campaign is engaging millions of young people, both in generating ideas and in voting for them, as this ad shows.

- Increasingly, digital campaigns and TV campaigns are interwoven. Take this "Happiness Factory" ad for Coke.

Let's set aside the implication that Coke is a healthy way to combat a sleep deficit. What many people watching this may not realize is that it's more than just a TV ad -- it ties in to an entire immersive online environment, where you can play interactive animated games, download music from popular artists, get free stuff and invite your friends to join in the fun.

Indeed, with digital marketing, unlike traditional TV ads, kids are actively engaged for many minutes, even potentially hours at a time, with the brand. These experiences are intensive, interactive, and socially stimulating for youth. We are only just beginning to explore the impact of this kind of immersive marketing, but the implications are troubling.

And of course, every click of the mouse, every text message received from a teen, gives the companies more valuable data about their target market. It's ironic and disturbing that these corporations are collecting vast amounts of data on our children while parents may have no awareness this is even going on, and certainly have limited ability to monitor or control the marketing messages their children receive.

Again, remember, sweetened beverage companies are devoting far more resources to these forms of digital marketing than any other food or beverage category does.

⁶ Branding consultant Simon Mainwaring quoted in *Ad Age* "Cause Effect: Brands Rush to Save World One Deed at a Time," March 1, 2010.

ETHNIC TARGET MARKETING

One final area of concern is ethnic target marketing. Beverage companies are intensively driving their promotions to the populations that suffer the most from the health problems associated with sweetened beverage consumption. Just as the tobacco and alcohol industries did before them, beverage companies are reaching out to the African American and Latino communities, fashioning products to fit their tastes, creating price points favorable to these groups, and saturating these communities with targeted ads for their products.

Youth of color are a particularly attractive market segment for soda companies, as they tend to be early adopters of new media technologies – they use text messaging at a far higher rate than the overall teen population -- and they tend to be influential over the broader youth culture.

The companies know this, as seen when Coca Cola unveiled its 2007 Sprite Yard program for mobile phones, aimed at its mostly African American youth target audience. This program used the slang conception of a “yard,” a place where everyone hangs out, to characterize a virtual space. As the Sprite brand director said, “We know that when it comes to reaching teens, mobile is the medium. This program will enable us to connect with teens by putting Sprite both in their hand and in their phone.”⁷

Just a couple of other examples of ethnic target marketing by the soda companies:

- This last ad I’ll show features P. Diddy; it both pokes fun of and reinforces the way hip-hop culture is used to sell products to the broader American public. You can see how the trend starts with Diddy and trickles down to, shall we say, less hip demographics.
- Finally, just last fall Pepsi launched PepsiWeInspire.com – an online blogging community that targets African American moms. You know, the soda companies claim to be concerned about improving children’s health but then they do something like this, which is aimed directly at influencing the nutritional gatekeepers of the kids with the highest rates of diabetes and obesity in our country.

Soda companies make so much money off these communities, and then they leave a public health disaster in their wake -- it’s simply unfair to target these communities in this way.

⁷ Denis Sison, Sprite Global Brand Director, The Coca-Cola Company quoted in company press release, http://www.thecoca-colacompany.com/presscenter/nr_20070606_sprite_yard.html More of the quote: ““We can provide them ‘instant gratification’ through ever-changing content and the ability to immediately receive new information and entertainment.”

OTHER

There are many other problematic beverage marketing practices including:

- viral videos and peer brand advocates;
- sponsorship of sports and cultural events; and
- philanthropic donations for health research and health education campaigns.

More details are available in our framing brief and at our online site, digitalads.org.

Finally, I'd like to note that I come to you not just as a public health professional but as a mom of two young children. We all know that parents have primary responsibility for setting the dietary directions of their children – but parents need help. And against the deluge of marketing I've been describing to you, parents really don't stand a chance.

My children are just three and five; they live in a soda-free home, and they watch virtually no commercial TV. But they recognize soda logos, they are attracted to them, and my 5 year old in particular has a strong preference for the Coke logo over the Pepsi logo. That tells me that marketing works, that something far beyond my power to control as a parent is at work here. This is why I'm so concerned, not just for my children but for their whole generation.

I thank you for investigating this critical issue.

Marketing of Sugar-Sweetened Beverages to Youth

**Katie Woodruff, MPH
Berkeley Media Studies Group
April 20, 2010**

Marketing's Four Ps

- **Promotion**
- **Products (including packaging)**
- **Price**
- **Place**

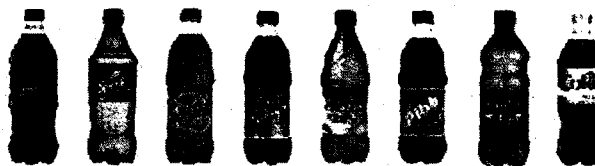


sparkling

[view official site](#) 

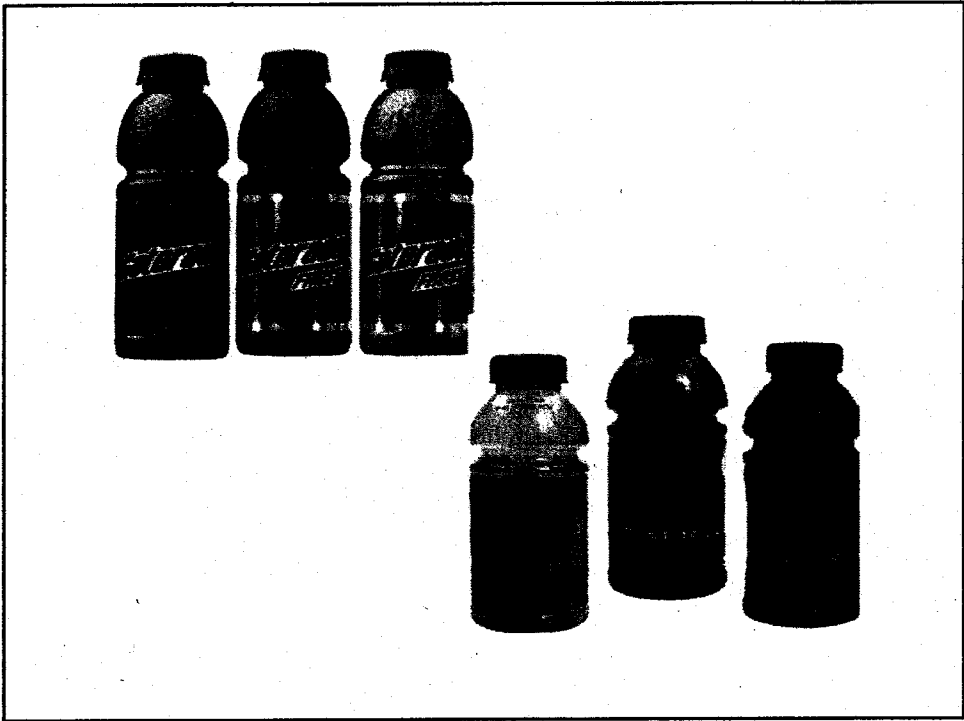
Coca-Cola

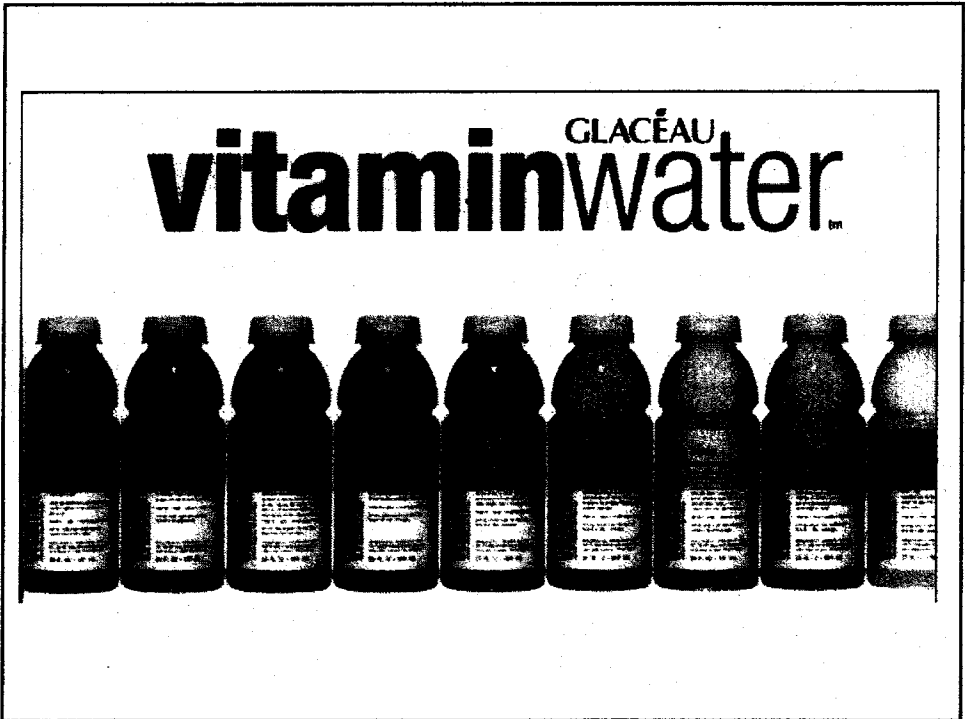
Coca-Cola is the most popular and biggest-selling soft drink in history, as well as the best-known product in the world. Created in Atlanta, Georgia, by Dr. John S. Pemberton, Coca-Cola was first offered as a fountain beverage by mixing Coca-Cola syrup with carbonated water. Coca-Cola was introduced in 1886, patented in 1887, registered as a trademark in 1893 and by 1895



[NEXT CATEGORY](#) 









something for everyone

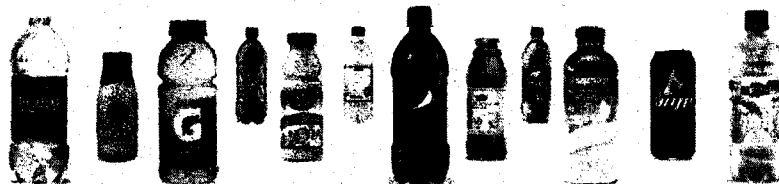
HOME

ABOUT OUR PRODUCTS

ABOUT OUR INGREDIENTS

BRAND SITES

STORE LOCATOR



The Coca-Cola Company

Beverage Lineup

Coca-Cola Stories

Refreshing Ideas

Live Positively

Promotions

Coca-Cola Links

a world of choices



sparkling (7)



energy (7)



water (7)



Coca-Cola (7)



tea & coffee (7)



sport (7)



other (7)



Refreshing Ideas

Get up & fresh up today with these simple, delicious recipes.

Get Inspired



Be Connected

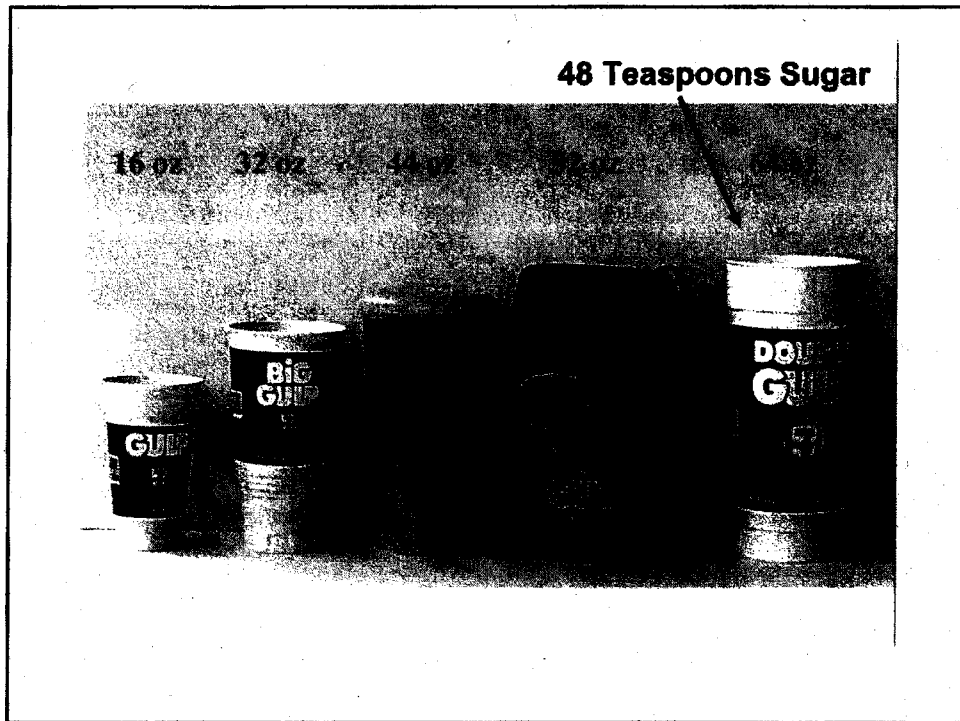
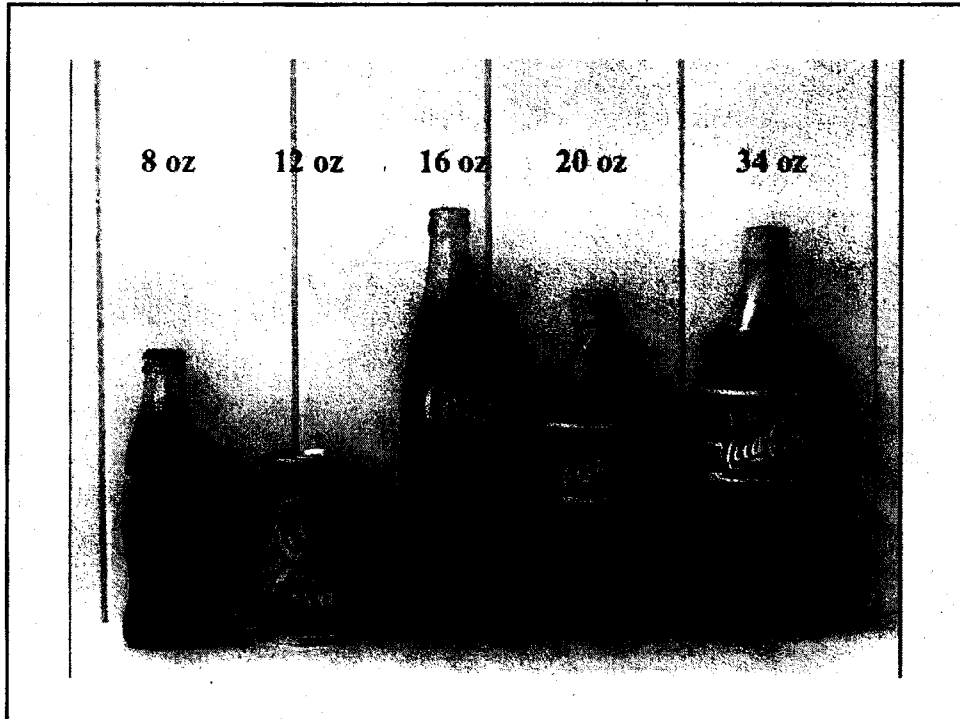
Get up & fresh up today with these simple, delicious recipes.

Share | Sign Up



Live Positively

Get up & fresh up today with these simple, delicious recipes.



Pennies an ounce



2.3 cents per ounce



2.4 cents per ounce

HOME FLAVORS THE FANTANAS FANTANA FUN

DANCE WITH THE FANTANAS

INSTRUCTIONS CHOOSE A BACKGROUND. CHOOSE A FANTANA DANCE. **START!**
UPLOAD YOUR PHOTO. SHARE WITH YOUR FRIENDS.

DOWNLOADS

YOUR PHOTO HERE

REPLAY

Privacy Policy | Terms of Service | DMCA | Rules | Gameplay | Contact Us

© 2009 The Coca-Cola Company. "Fantana" and "Fantanas" are registered trademarks of The Coca-Cola Company.

Sugar Water Gets a Facelift: What Marketing Does for Soda

SEPTEMBER 2008

Consumers want their water consumption choices to be healthier and more flavorful. In fact, they are. However, their choices are being shaped by the drive to maximize the production margins of the industry. From the sleek, sleek Cola, Pepsi, and the new "Schwappys" to the new "Fruit" sodas, the industry is working hard to make sure that "You can still get it" (The Coke Side of Life for "Thank Pepsi, Get Real" for the health and get double the amount of brandable flavors and other "add-ons").

Without marketing, soda would be known only for the standard brand on their bottle: no name, just a clear or brownish liquid. The drive to maximize the production margins of the industry is what has led to the Cola, Pepsi, and the new "Schwappys" to make sure that "You can still get it" (The Coke Side of Life for "Thank Pepsi, Get Real" for the health and get double the amount of brandable flavors and other "add-ons").

It is not only the drive to maximize production margins that has led to the Cola, Pepsi, and the new "Schwappys" to make sure that "You can still get it" (The Coke Side of Life for "Thank Pepsi, Get Real" for the health and get double the amount of brandable flavors and other "add-ons").

It is not only the drive to maximize production margins that has led to the Cola, Pepsi, and the new "Schwappys" to make sure that "You can still get it" (The Coke Side of Life for "Thank Pepsi, Get Real" for the health and get double the amount of brandable flavors and other "add-ons").

It is not only the drive to maximize production margins that has led to the Cola, Pepsi, and the new "Schwappys" to make sure that "You can still get it" (The Coke Side of Life for "Thank Pepsi, Get Real" for the health and get double the amount of brandable flavors and other "add-ons").

It is not only the drive to maximize production margins that has led to the Cola, Pepsi, and the new "Schwappys" to make sure that "You can still get it" (The Coke Side of Life for "Thank Pepsi, Get Real" for the health and get double the amount of brandable flavors and other "add-ons").

It is not only the drive to maximize production margins that has led to the Cola, Pepsi, and the new "Schwappys" to make sure that "You can still get it" (The Coke Side of Life for "Thank Pepsi, Get Real" for the health and get double the amount of brandable flavors and other "add-ons").

It is not only the drive to maximize production margins that has led to the Cola, Pepsi, and the new "Schwappys" to make sure that "You can still get it" (The Coke Side of Life for "Thank Pepsi, Get Real" for the health and get double the amount of brandable flavors and other "add-ons").

SSB Marketing to Youth

- \$492 million spent to market sugar-sweetened beverages to children and adolescents in 2006

Source: Federal Trade Commission, 2008

SSB Marketing to Youth

- \$492 million spent to market sugar-sweetened beverages to children and adolescents in 2006
- = \$1 MILLION+ PER DAY

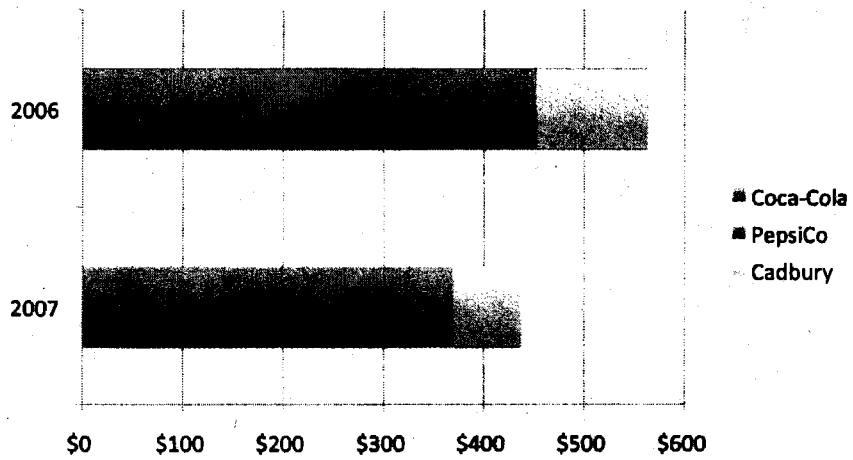
Source: Federal Trade Commission, 2008

Digital Marketing of SSBs

- \$21 million spent on digital marketing/"new media" in 2006
- More than any other F/B category
- Fastest-growing marketing techniques

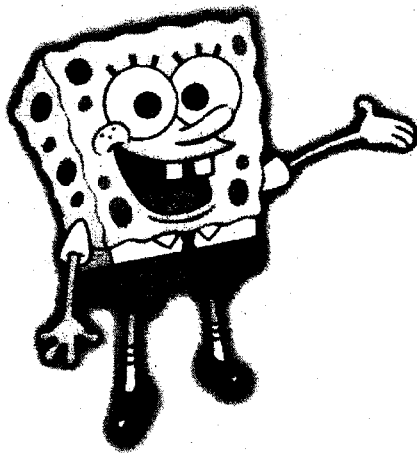
Source: Federal Trade Commission, 2008

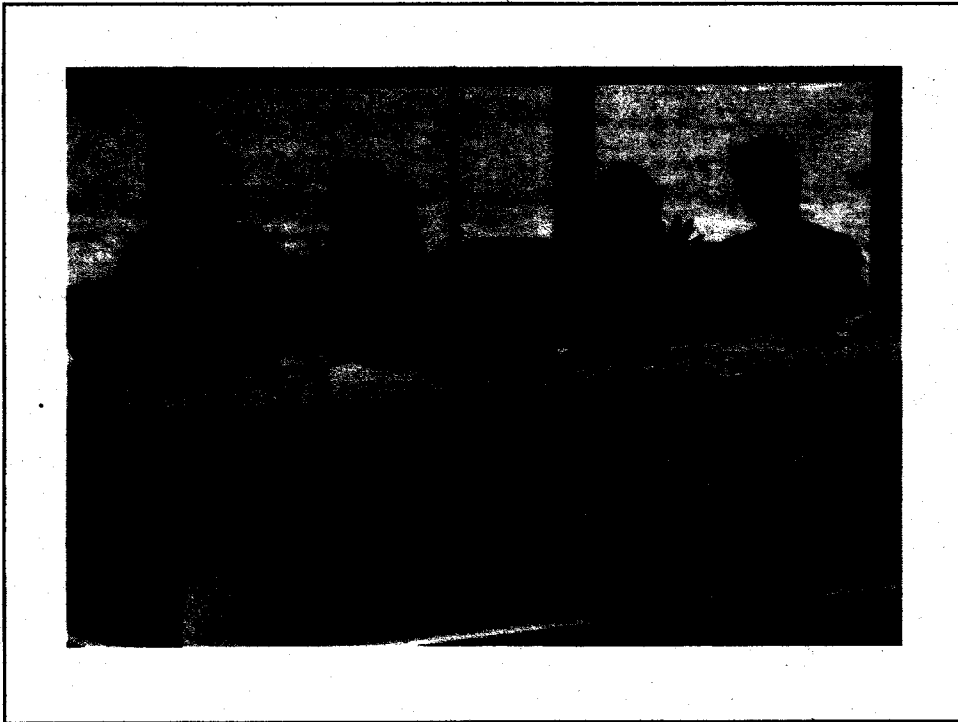
TV ad buys for SSBs declining



In millions. Source: TNS Media Intelligence

Nickelodeon's SpongeBob SquarePants





COKE

TWIST.TXT.
you could
WIN **10000**
of COKE

HOW TO PLAY OFFICIAL RULES FAQS PRIZES WINNERS LIST

REDEEM

EA SPORTS

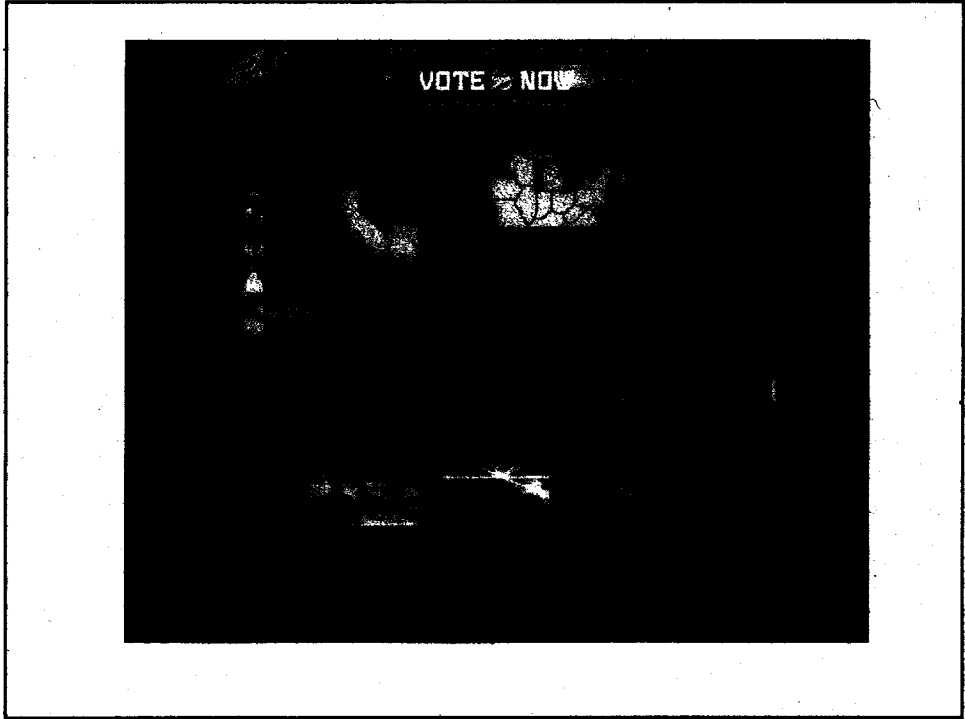
GRAND PRIZE
4 EA SPORTS GAMES
and GAME CONSOLE.
It's game time.

1st PRIZE
A PAIR OF MOVIE PASSES

2nd PRIZE
\$20 COKE CASH CARD

3rd PRIZE
\$20 LIVE NATION[®]
CONCERT CASH

SEE ALL PRIZES



VOTE NOW!

Round up giving away millions to fund great ideas. Voting for the new ideas is now open. Find your favorite ideas and come back to support them all month. See who got the most support.

ALL CATEGORIES

- HEALTH
- ARTS & CULTURE
- FOOD & SHELTER
- THE PLANET
- NEIGHBORHOOD
- EDUCATION

CURRENT GRANT CYCLE

Days left to vote: **12**

Start in the survey: **11:38**

Project announced: **May 1st**

Funding available: **\$1,300,000**

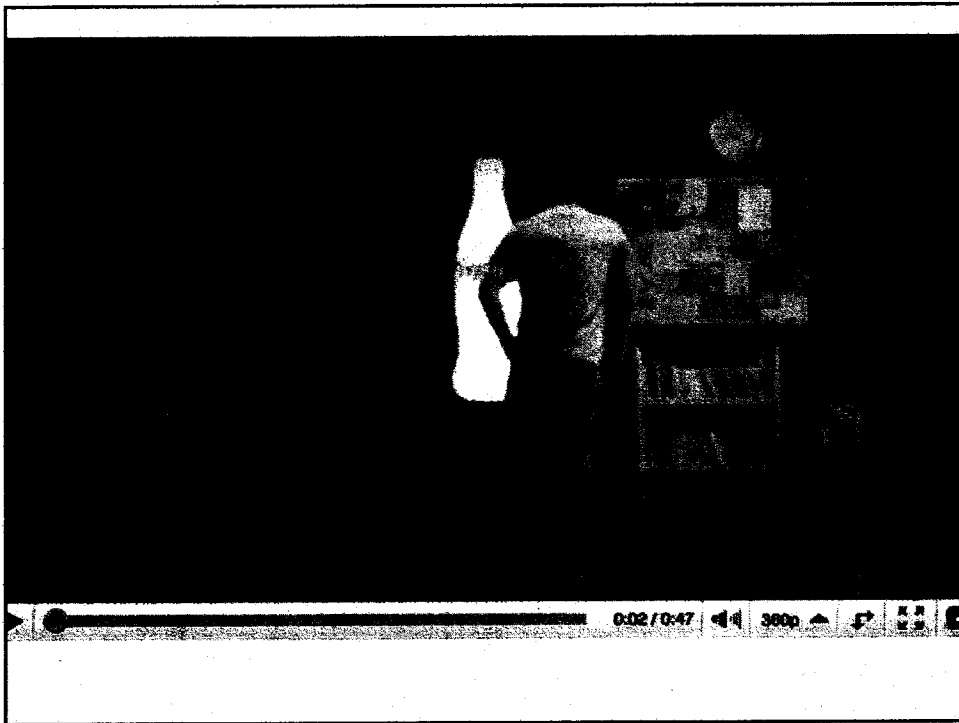
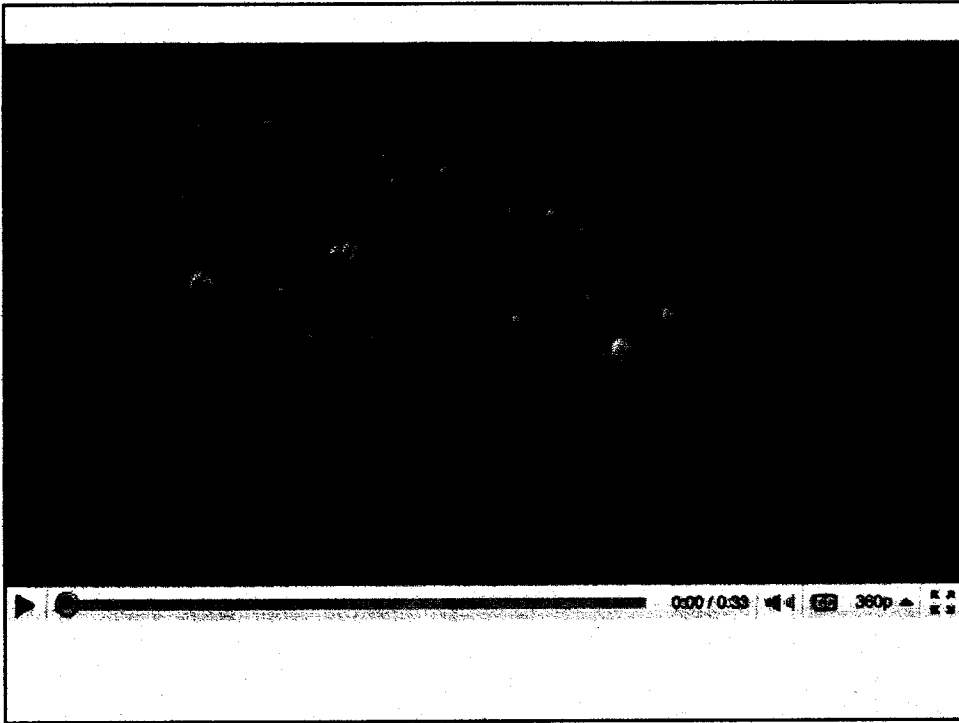
VOTE FOR \$100,000 \$25,000 \$50,000 \$200,000

95 **Take the Foothill Elem. 6th Grades to Outdoor School in Yosemite**

Project description and details.

112 **Provide economically vulnerable women with medical assistance**

Project description and details.





Reaching youth of color

- “We know that when it comes to reaching teens, mobile is the medium. This program will enable us to connect with teens by putting Sprite both in their hand and in their phone.”

– Denis Sison, Sprite Global Brand Director, The Coca-Cola Company, company press release on the launch of the “Sprite Yard” campaign, June 6 2007



Home | Search | Log Out | Account



Welcome to Pepsi We Inspire! Join us in sharing the simple pleasures in life that motivate and inspire you by clicking the "share your inspiration" button.

Latest Inspiration [Share your inspiration](#) [View all inspiration](#)

A row of six black and white portrait photographs of diverse women. Below the photos is a paragraph of text: "Welcome to Pepsi We Inspire! Join us in sharing the simple pleasures in life that motivate and inspire you by clicking the 'share your inspiration' button." At the bottom, there is a navigation bar with the text "Latest Inspiration" followed by two buttons: "Share your inspiration" and "View all inspiration".

**Written testimony and/or additional material
submitted by:**

**Genoveva Islas-Hooker, MPH
Regional Coordinator
Central California Regional Obesity
Prevention Program**

TESTIMONY - April 20, 2010

Purpose: *Testify on the over consumption of sugar from sweetened drinks, such as soda, and its link to obesity.*
7-Minutes

Good Afternoon

My name is Genoveva Islas-Hooker. I am a board member for the Latino Coalition for Healthy California as well as a board member for California Food Policy Advocates. I live in Tulare, California and I am involved in creating healthier food and physical activity environments through the Central California Regional Obesity Prevention Program (CCROPP). We are a partnership between public health departments, community based organizations and grassroots community members including youth in Fresno, Kern, Kings, Madera, Merced, San Joaquin, Stanislaus and Tulare Counties.

It's an honor to be here with you all. I've been asked to testify on the over consumption of sugar from sweetened drinks, such as soda and it's link to obesity. To that end I would like to provide you some context...

Central California is the fruit and vegetable bowl of our state, nation and perhaps the world. It's everything that you can imagine a rich agricultural valley to be; picturesque, green and rural.

And it's many things that you may not imagine it to be... In this place of bounty there is hunger and poverty. ~~The central valley is home to the poorest congressional districts, and much of that poverty stems from the super-exploitation of farm laborers.~~

This context is important to understanding why obesity is such a prevalent health issues in our area. (Central California has the highest rates of obesity and obesity related diseases such as hypertension, heart disease, diabetes and cancer. 1 in 3 children are obese. In some of our counties 70% of our adult population is overweight or obese.)

In a nutshell,

- poverty,
 - the over-availability of cheap unhealthy foods like soda
 - the under-available of healthy affordable foods and beverages like water
 - the omnipresent targeting, promotion, and marketing of unhealthy choices like soda and other sugar sweetened beverages
 - as well as the limited physical activity resources and infrastructure ;
- are predisposing us to an increased incidence of obesity.

In my region, community members are making choices about their food and beverages that are driven by their economic means and by what is available.

Sodas have become the default beverage choice because they are cheap and they are readily available. (As documented in the report "Bubbling Over, Soda Consumption and it's Link to Obesity in California", Central California also has some of the highest rates of soda consumption in our state.)

"In this photo I see a food store and near the door there are seven soda machines and only one water machine.

People are more likely to buy soda especially because some sodas cost twenty-five cents while the water is more than a dollar.

This affects the community because people are more likely to consume soda.

This challenge exists because by selling soda at such a low price it attracts more people than water which is more expensive.

The food store can help by having more water machines and lower the price of water.

This photo makes me feel like food stores are advertising soda more than water."

-Jasmine, Sunnyside High School student in Fresno, CA

Food store can help but there are many other opportunities to change the food/beverage and physical activity environments in order to support better health.

For example, in many Central Valley communities, water is un-drinkable... Community members are having to pay a bill for water that they can not drink. Then they are having to pay for bottled water. So if you are struggling, and you are trying to stretch your dollars what do you do? You look for the most affordable options like soda and other sugar sweetened beverages. ...free, safe drinking water often times can not be found, especially in many rural unincorporated communities. I have a quotes to highlight this issue.

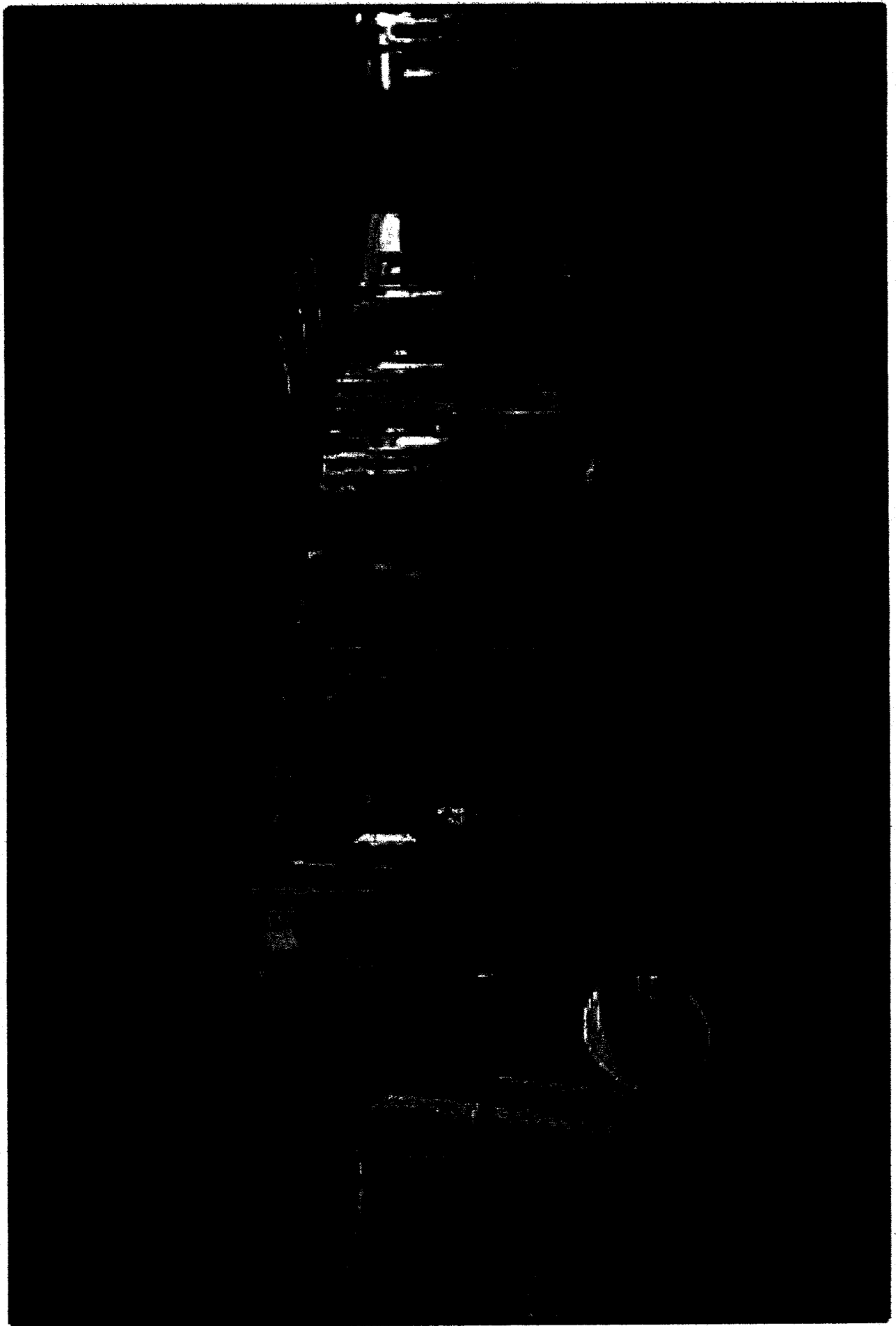
"It's not fair that I don't have safe drinking water at home and that when I go to school I have to deal with the same thing".

-Jessica Sanchez, student at Orosi High

Revenues generated from the tax are desperately needed to turn the tide on the obesity epidemic. This resources could help to change the food/beverage and physical activity environments in many under-resourced communities. We need things like...

- increased access to safe drinking water
- support for increased access to healthy foods
- more physical activity environment and resources, parks and recreation, improved community design
-

In closing, I applaud Senator Florez who is leading a penny per teaspoon tax on sugar sweetened beverages; thank you Senator Florez for legislating for the health of our communities.



**Written testimony and/or additional material
submitted by:**

**Dana Richardson
Representative of the Healthy Eating Active
Communities Initiative**

Greetings Members of the CA State Legislature Food & Agriculture and Health Committees;

My name is Dana Richardson; I am a resident of Southern California – in the City of National City, and I also represent the Healthy Eating Active Communities Initiative in Chula Vista (located just 10 minutes north of the U.S. / Mexico border at Tijuana). Since March 2005, our project has worked to reduce childhood obesity and promote healthy lifestyles by improving food and physical activity environments, particularly in the western Chula Vista area.

Clearly, there are connections between **health** and **place**, which have become increasingly prominent as communities struggle with alarming levels of asthma, obesity, heart disease and diabetes (particularly) in low-income, communities of color. Neighborhood environments are critical factors, which either can support or undermine **any** local communities' ability to engage in physical activity, and adopt healthy lifestyles.

A white paper written by one of my colleagues, Mary Lee of the Policy Link organization states that ...

“... Environmental conditions, along with social and economic factors, play a **much larger** role in determining the health outcomes of a community. To this end, it is becoming increasingly clear that **where you live affects your health**, and that **the health of individuals depends on the neighborhood in which he or she lives**. **Economics** and the **zip code of residence** tell us much more about the key factors that shape negative or positive **health behaviors** and **health outcomes** than does their physiology.”

So, how do we set our youth and families up for success and healthy lifestyles? **Answer:** WE CREATE NEIGHBORHOOD ENVIRONMENTS THAT FACILITATE ACHIEVING THESE GOALS and we use policies with a broad focus on health to address the underlying issues that fuel unhealthy habits and lifestyles, and provide populations with the resources and tools to avoid what is ailing them in the present day.

Many poor lifestyle decisions are made in the context of the community environments. As I stated earlier, where you live affects your health. And, a Physical Environment that **supports** health obviously does **NOT** contain what I currently experience in my own community, which includes:

(1) a prevalence of alcohol outlets (literally on every corner) and numerous billboards with alcohol advertisements targeting predominant Latino/Black populations, (2) a prevalence of fast food outlets and a limited number of healthy food options (for example, the only major chain grocery store in my community is in the process of closing as we speak), and (3) areas of our community with no sidewalks and little to no access to parks and open space. This is just my short list. There is actually a much longer list of community conditions that I can share with you which contribute to childhood obesity and community-based violence.

Conversely, neighborhood environments that **do** support health provide a balance of healthy and accessible food and beverage options for the community, and also match these strategies with long-range plans to incorporate ample public transportation, affordable, well-maintained housing, schools, parks, complete streets for all users, thriving businesses, new employment opportunities, and accessible, safe public play and recreation facilities, to name a few.

Senator Flores' legislation is definitely a step in the right direction – **to strategically address** poor health outcomes as it relates to the consumption of sugar, sweetened beverages, particular in low-income, vulnerable populations. The research has proven that this is a key strategy for addressing this immediate concern, and achieving better health outcomes. Yet, from an infrastructure standpoint the legislation **is also visionary**, because it proposes to contribute **RESOURCES** toward creating healthier environments by improving nutrition in schools and ensuring access to safe, quality parks and open spaces. This legislation provides us a short-term immediate strategy, yet also recognizes the long-range, sustainable elements that communities need to achieve health.

Therefore, I encourage this committee to **SUPPORT** this policy today. Together, we can begin to **“turn the tide”** on poor health outcomes that persist in vulnerable populations, and **CREATE COMMUNITIES OF OPPORTUNITY**.

Thank you for allowing me to speak.

**Written testimony and/or additional material
submitted by:**

The Coca-Cola Company

Sen. Florez Hearing – Statement by The Coca-Cola Company

April 19, 2010

We at The Coca-Cola Company share the Legislature's concern over the continued epidemic of overweight and obesity in California and in the United States. Our Company is committed to doing our part to help Californians live a healthy lifestyle, including offering an ever-increasing range of beverage options that fit within a balanced diet and supporting programs that encourage physical activity. We introduced TaB, our first calorie-free cola, in 1963 and diet Coke in 1982. And over the last 40 years, The Coca-Cola Company has introduced more than a hundred low- and no- calorie beverages to complement our line of regular soft drinks, providing consumers a choice as to the caloric content of their beverages.

People consume many different foods and beverages, so no one single food or beverage alone is responsible for people being overweight or obese. All calories count, whatever food or beverage they come from, including those from our caloric beverages. But according to data from the National Cancer Institute, sodas, energy drinks, sports drinks, and sweetened water beverages combined now comprise 5.5% of all calories consumed by Americans – so nearly 95% of our calories come from other sources. (Bosire et al., 2009).

In light of these facts, focusing primarily on reducing the consumption of sugar-sweetened beverages in California is not the answer to resolving the overweight and obesity epidemic in the state. Although it is certainly true that adding calories to a stable diet, whether those calories come from sugar-sweetened soft drinks or from other foods, can contribute to weight gain if not balanced by an increase in physical activity, our body's energy balance is extremely complex. As noted by the Institute of Medicine in its 2005 report on childhood obesity, "Although 'energy intake = energy expenditure' looks like a fairly basic equation, in reality it is extraordinarily complex when considering the multitude of genetic, biological, psychological, sociocultural, and environmental factors." Given the complexity of this system, it is not surprising that, after reviewing extensive scientific evidence regarding sugar-sweetened beverages and

obesity, the American Heart Association concluded in its Scientific Statement on "Dietary Sugars Intake and Cardiovascular Health," issued in August of 2009, that "[b]ecause overweight and obesity are complex metabolic conditions, it is unlikely that a single food or food group is primarily causal." (Johnson et al., 2009).

In other words, although regular soft drinks have calories, we cannot blame overweight or obesity on soft drinks or any other single food or beverage alone. A number of large-scale studies have shown that consumption of sugar-sweetened soft drinks has little impact on long-term weight status. (Schulze et al., 2004; Palmer et al., 2008; Kvaavik et al., 2004). Indeed, while consumption of sugar-sweetened soft drinks has decreased substantially in the last 10 years, rates of overweight and obesity over that same period have continued to increase or remain stable. (Popkin, 2010; Flegal et al., 2010).

Furthermore, suggesting that sugar-sweetened beverages are driving an increase in diabetes is similarly inaccurate. The American Diabetes Association has identified the risk factors for diabetes, including obesity and a lack of physical exercise. However, the ADA does not identify the sugar used in soda, or any other single food or ingredient, as a specific risk factor for the disease. In fact, a 2003 study concluded that "intake of sugars does not appear to play a deleterious role in primary prevention of type 2 diabetes." (Janket et al., 2003). Others have also found that sweetened beverage consumption showed no consistent association with the incidence of type 2 diabetes mellitus. (Paynter et al., 2006). These studies reinforce the ADA guidelines that a moderate amount of sugar can be incorporated in a healthy diet.

We must not forget that energy balance involves both energy consumption *and* energy *expenditure*. Lack of physical activity must be addressed when discussing obesity. For example, a study published in Obesity Reviews examined associations between obesity and certain dietary and physical activity patterns in over 130,000 children in 34 countries and did not find an association between soft drink intake and overweight. This study did find low levels of physical activity were associated with increased Body Mass Index (or BMI). (Janssen et al., 2005).

Suggesting that sugar-sweetened beverages are disproportionately responsible for obesity may be convenient and simple, but it disregards the complexity of the science. The fact is that we cannot blame overweight or obesity on soft drinks or any other single food or beverage alone. If the goal is to create sound health policy that addresses the obesity rates in California then it must be solved with a multi-faceted approach and through partnership among consumers, government, business and medicine. That means together we must provide consumers with options, give them meaningful opportunities to stay active and help them understand how to live a healthier lifestyle.

As part of the food and beverage industry and a member of the California community, The Coca-Cola Company is committed to doing our part to help Californians live healthy lives and maintain healthy weight. In our almost 125 year history, we have been innovators, consistently evolving our business, and developing products and packaging to offer many beverage options supportive of a healthy lifestyle. We are now the world's leading beverage company. And we are a total beverage company. We offer sparkling beverages, water, juices, tea, coffee, and energy drinks. We produce the Minute Maid line of products, Simply Orange and other Simply juice products, Gold Peak tea, and Odwalla juices and juice drinks. Our Odwalla business is based in California.

We are also offering equipment that provides consumers with options. In Southern California, we are currently testing a new fountain dispenser, Coca-Cola Freestyle that offers consumers the selection of more than 100 beverages - more than half of which are low and no-calorie. We are proud of these innovations and are always working to offer consumers new beverage options.

We also recognize the importance of providing consumers with options for portion control. To that end, we recently introduced smaller 90-calorie cans of Coca-Cola and other beverages that will soon be available in California and nationwide.

And in 2010, Coca-Cola is supporting an initiative developed and led by the First Lady of the United States, Michelle Obama, to end childhood obesity in a generation. Coca-Cola has joined the U.S. beverage industry in supporting Mrs. Obama's initiative,

called "Let's Move," with a "Clear on Calories" commitment. This commitment includes the following:

Product Labels: As part of the Clear on Calories initiative, we will display total calorie counts on the front of nearly all containers. Containers up to and including 20 fluid ounces will state calories for the full package; larger, multi-serving packages will be labeled using a 12-fluid ounce serving (as well as the number of servings per package) for nearly all beverages. One hundred percent juices will continue to use an 8-fluid ounce serving size.

Company-controlled Vending Machine Labels: Total calorie counts per package will be displayed on selection buttons.

Company-controlled Fountain Equipment Labels: Total calorie counts will be displayed prominently.

This commitment builds on the leadership role we established in the global industry last September (2009) when we announced our goal to provide front-of-package calorie labeling for nearly all of our products globally by the end of 2011.

We make the same commitments with our programs. For example, there are 113 California chapters of the Boys & Girls Clubs of America that offer Triple Play, which is designed to provide young people with the basic knowledge on nutrition, exercise and teamwork. Results of a two year study released earlier this year found that children who participated in the program increased in their level of physical activity, made smarter nutrition choices and improved their sense of self mastery.

Beyond that, we are proud to be industry partners on the School Beverage Guidelines. Since the adoption of these guidelines, as a result of increasing the availability of no- and low-calorie beverages in schools all across the country, the U.S. soft drink industry has achieved an 88 percent reduction in total calories from beverages

delivered to schools in the first half of the current school year as compared to the first half of the 2004-05 school year. Over the same period, there has been a 95 percent decline in shipments of full calorie beverages. Overall, 98 percent of schools are now in compliance with the guidelines.

And in October of 2009, we joined with other food and beverage industry leaders in committing to actions designed to help Americans live active, healthy lives and make informed choices about what they eat and drink. The Healthy Weight Commitment Foundation aims to bring a common sense approach to helping reduce obesity by 2015, with a particular focus on childhood obesity.

In total, Coca-Cola is committed to helping address obesity by supporting and encouraging active healthy living through our products, programs and policies.

**Written testimony and/or additional material
submitted by:**

American Beverage Association



April 20, 2010

Senator Dean Florez
State Capitol, Room 313
Sacramento, CA 95814

Re: Informational Hearing on Soft Drinks

Dear Senator Florez:

The American Beverage Association (“ABA” or “the Beverage Association”) is the trade association for America’s non-alcoholic refreshment beverage industry, representing hundreds of beverage producers, distributors, franchise companies and support industries. Our industry employs nearly 220,000 people nationwide, generates more than \$112 billion in sales per year and has a direct economic impact of more than \$136 billion. ABA members market hundreds of brands, flavors and packages, including carbonated soft drinks, ready-to-drink teas and coffees, bottled waters and water beverages, sports drinks, 100 percent juice, juice drinks, and energy drinks.

The ABA welcomes the opportunity to provide information that would make the greatest impact on consumers’ health - especially when it comes to reducing obesity. Issues related to weight gain, obesity, and overall good health are incredibly complex. According to the National Institutes of Health (“NIH”), the increase in obesity has been fueled by a complex interplay of environmental, social, economic, and behavioral factors, acting on a background of genetic susceptibility.

In order to help reverse the obesity trend and reinforce overall good health, consumers need to be educated on the importance of overall diet and energy balance; that is, all calories consumed from all foods and beverages must be balanced by all calories expended through all forms of physical activity, including occupational and leisure time physical activity.

Attached are two documents: 1) a PowerPoint presentation that was presented previously at a hearing called by Senator Alex Padilla on November 5, 2009 and 2) a summary of studies that discuss several topics related to soft drinks.

American Beverage Association
April 20, 2010
Page 2

The PowerPoint presentation begins by examining the trends in the U.S. food supply and the available calories made from production of commodities, such as added fats and oils, grains, and sugars. These data shown in these slides are all collated and published online by the USDA Economic Research Service. Consumer demand and innovation by the industry have produced more low-calorie and no-calorie options. Hence, calories per ounce produced have declined as shown in Slides 7 and 8. Data clearly show that total calorie consumption has increased since the 1970s; yet, occupational and planned leisure-time physical activity has declined or remained flat (Slides 10 and 11). Calorie content of individual foods and beverages are shown in Slides 13 and 14. These data are from the USDA Nutrient Data Laboratory whose database is publicly available online. The pie charts shown on slides 16 and 17 are taken from an analysis conducted by the National Cancer Institute. These data were presented to the 2010 Dietary Guidelines Advisory Committee in April, 2009, and the tables of data are publicly available online at the USDA website www.dietaryguidelines.gov. Finally, epidemiological studies (a few of which are presented here) indicate there is no unique causal relationship between soft drinks and obesity. These studies are published in the peer-reviewed literature and were funded by the National Institutes of Health or the World Cancer Research Fund.

The establishment of discriminatory taxes or fees on soft drinks or other sugar-containing foods or beverages is not supported by the science; is economically harmful and punishes those who can least afford another California government-imposed tax or fee; and most importantly, will have no meaningful impact on the complex obesity issue.

Furthermore, a focus on soft drinks and a discriminatory tax or fee not only will fail to solve obesity, it will distract us from putting effective educational and informational programs into place. Programs that encourage citizens to understand how many calories they need to achieve and maintain a healthy body weight; to read food labels for calorie content; to monitor portion sizes and how much they are eating; and more importantly, to be more physically active to improve cardiorespiratory fitness, which is strongly linked to lower morbidity and mortality. That will achieve the goal we all seek.

Respectfully submitted,

Maureen Storey, PhD
Senior Vice President, Science Policy
American Beverage Association

Perspectives on Diet and Obesity

Maureen Glynn, PhD
Senior Vice President
Science Policy



Bottom Line

...and, in any case, imposing a tax on people who do not solve the problem.

- **Epidemiological evidence suggests that the consumption of beverages of any type is not associated with a subsequent weight gain and obesity, although the results are inconsistent;**
- **Education and information, not taxation, are the keys to solving obesity.**

Outline

...ations in the food supply (average caloric intake per person, lower calories per ounce produced) (Beverage Marketing Corporation data);

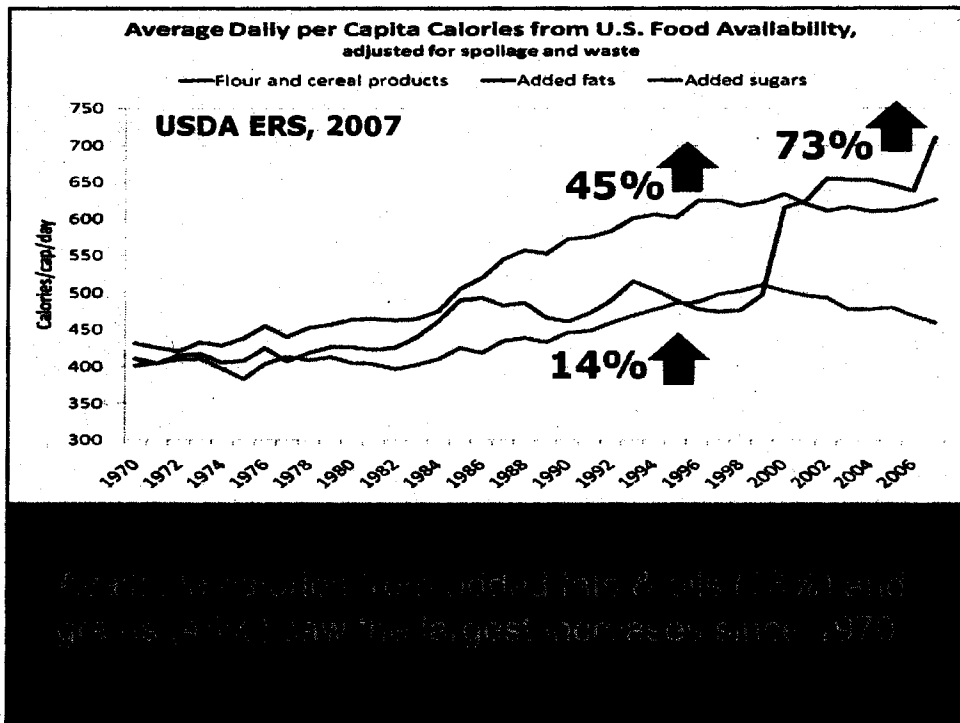
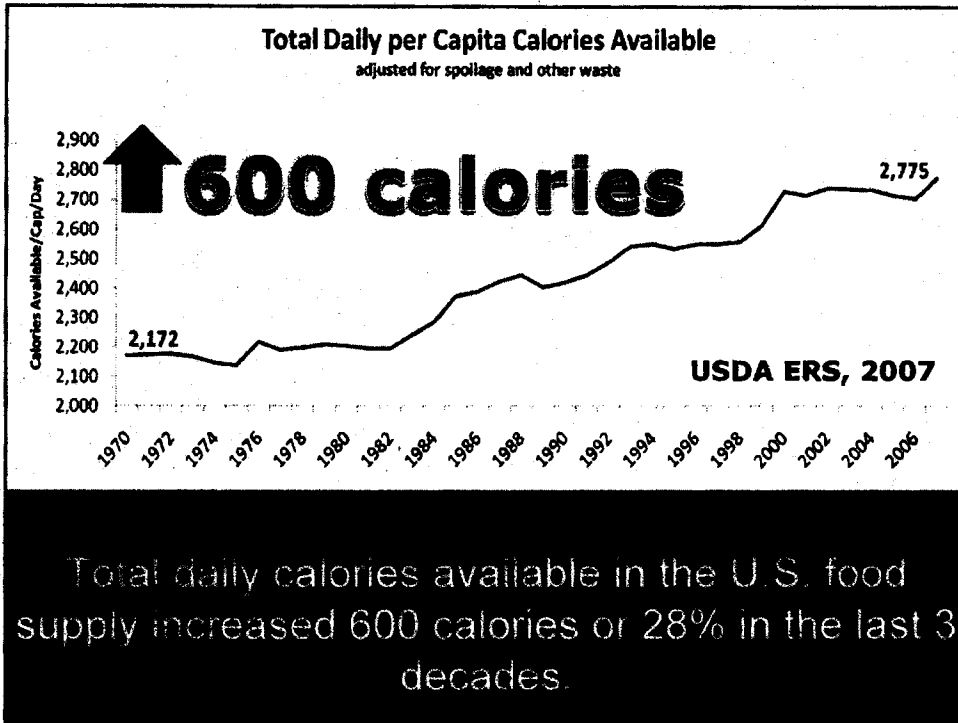
- **Food Consumption**
 - Increased total calorie consumption (Kant & Graubard study);
- **Sub-Macro Level of Individual Foods**
 - Calorie content of popular foods and beverages (USDA data);
 - Sources of calories in the food supply (National Cancer Institute analysis)
- **Epidemiological studies**

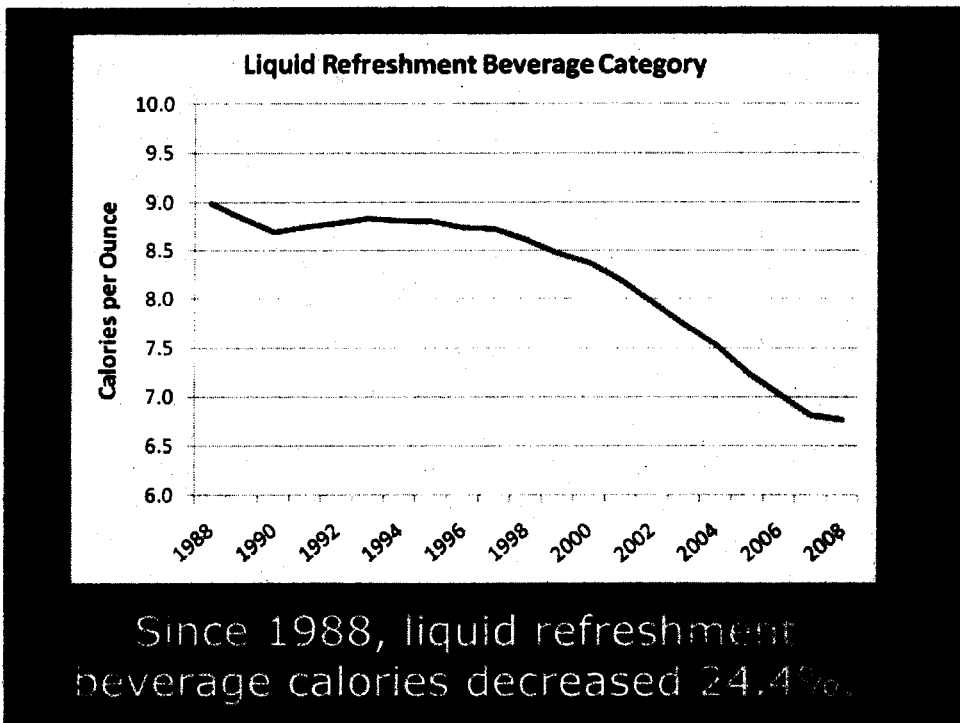
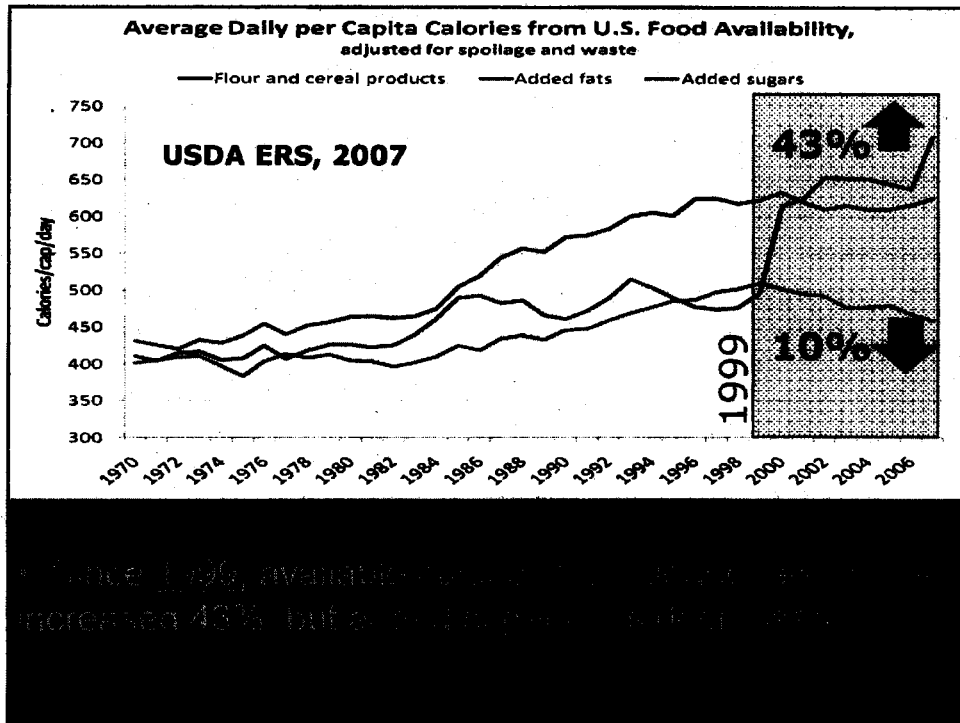
TRENDS IN U.S. FOOD SUPPLY 1970 – 2006

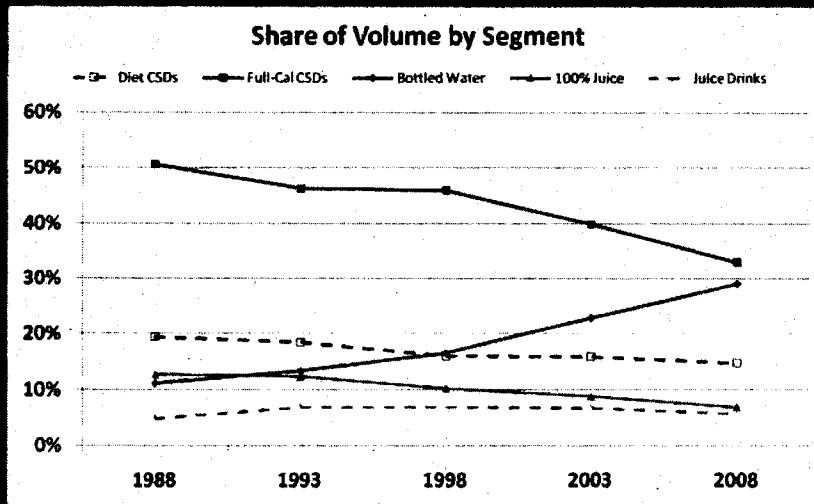
Data Source:

USDA Economic Research Service







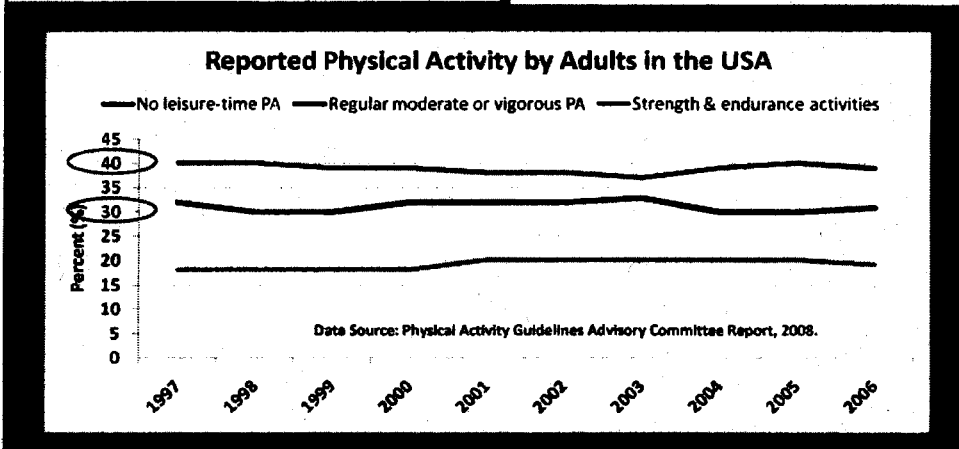
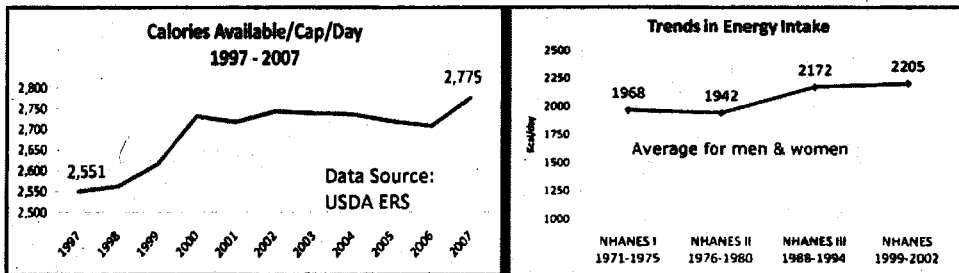
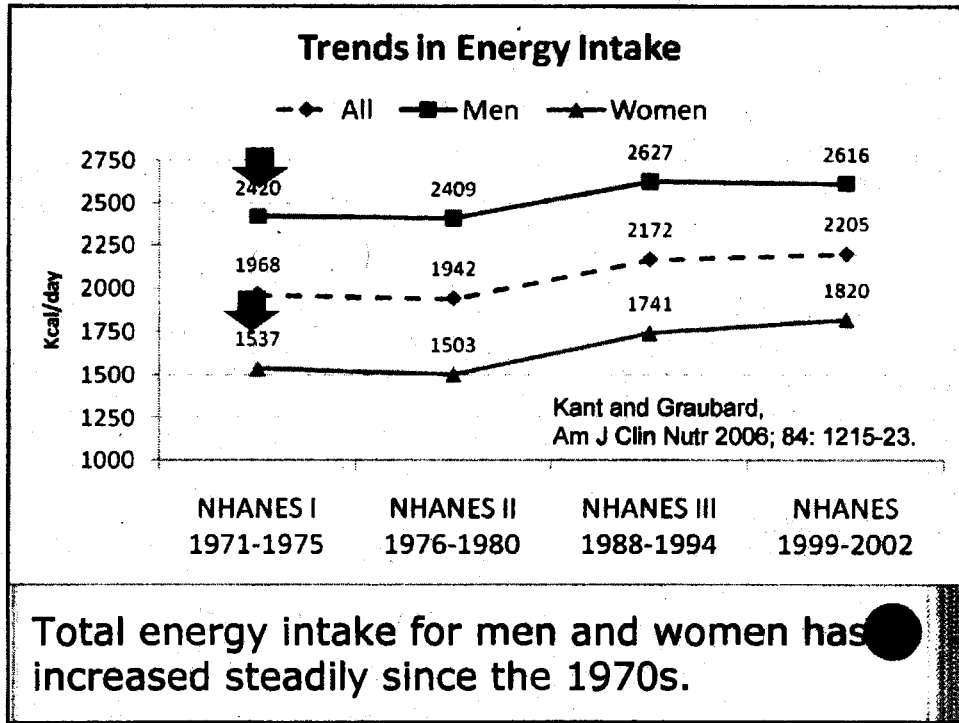


Since 1988, full-calorie CSDs has declined from 50% to 33% of market volume.

TRENDS IN CALORIE INTAKE

*Analysis published by Kant and Graubard,
Am J Clin Nutr 2006; 84: 1215-1223.*



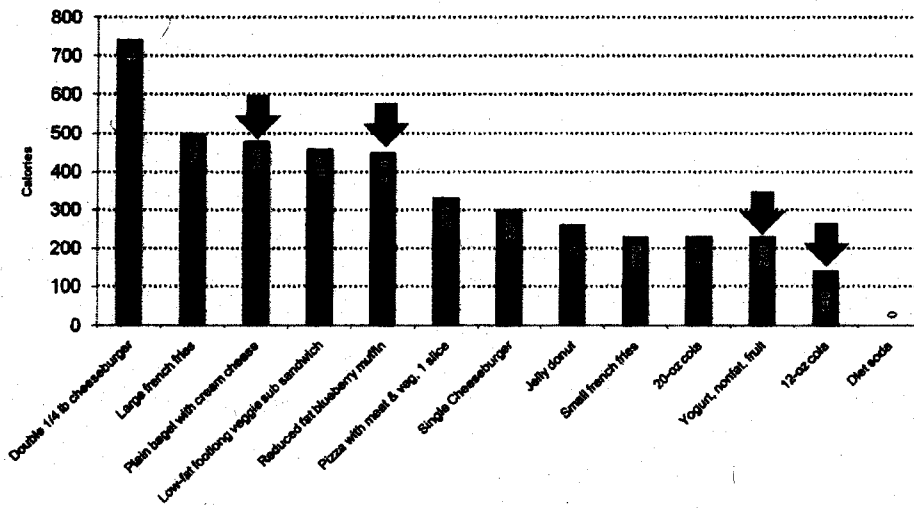


CALORIE CONTENT

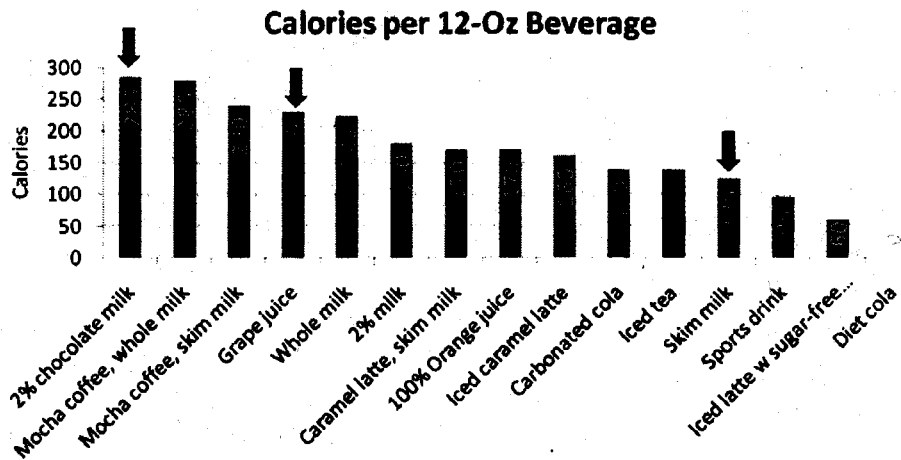
Data were compiled from the following websites: USDA Agricultural Research Service, Nutrient Data Laboratory and other publicly available websites)



For Obesity - All Calories Count!



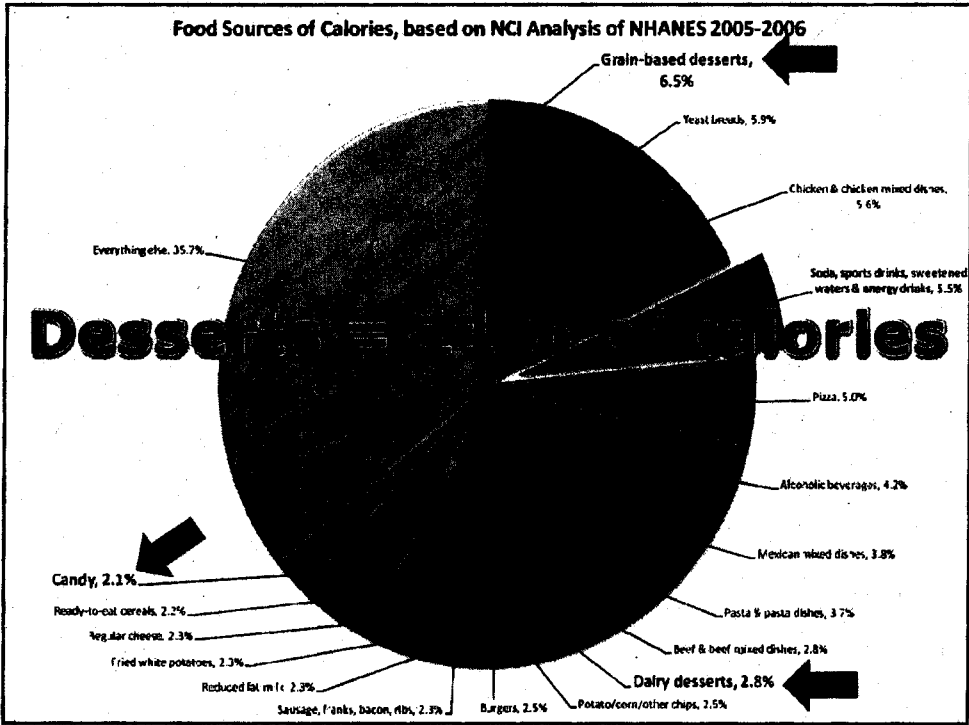
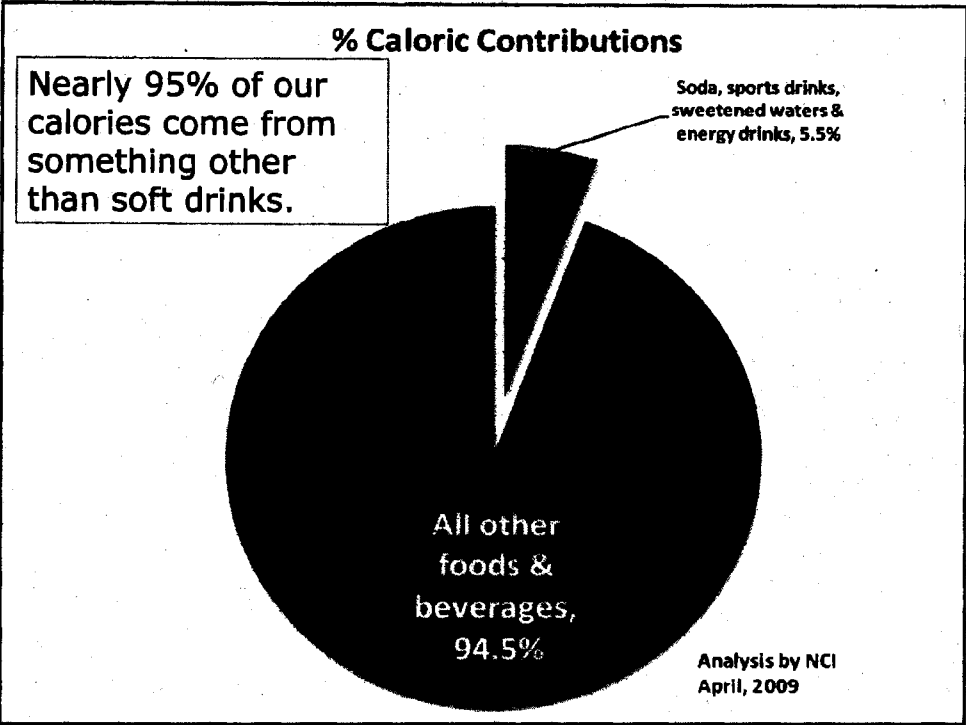
For Obesity - All Calories Count!



CALORIE SOURCES IN THE U.S. FOOD SUPPLY

Analysis conducted by the National Cancer Institute of the National Institutes of Health and presented to the 2010 Dietary Guidelines Committee in April, 2009.







**A SMALL SAMPLE OF STUDIES
AND REVIEWS**



The **NEW ENGLAND
JOURNAL of MEDICINE**

ESTABLISHED IN 1812

FEBRUARY 26, 2009

VOL. 360 NO. 9

**Comparison of Weight-Loss Diets with Different Compositions
of Fat, Protein, and Carbohydrates**

Frank M. Sacks, M.D., George A. Bray, M.D., Vincent J. Carey, Ph.D., Steven R. Smith, M.D., Donna H. Ryan, M.P.H.,
Stephen D. Anton, Ph.D., Katherine McManus, M.S., R.D., Catherine M. Champagne, Ph.D., Louise M. Bishop, M.S., R.D.,
Nancy Laranjo, B.A., Meryl S. LeBoff, M.D., Jennifer C. Rood, Ph.D., Lillian de Jonge, Ph.D., Frank L. Greenway, M.D.,
Catherine M. Loria, Ph.D., Eva Obazekan, Ph.D., and Donald A. Williamson, Ph.D.

In conclusion, diets that are successful in causing weight loss can emphasize a range of fat, protein, and carbohydrate compositions that have beneficial effects on risk factors for cardiovascular disease and diabetes.^{29,40} Such diets can also be tailored to individual patients on the basis of their personal and cultural preferences and may therefore have the best chance for long-term success.

CS Berkey; HRH Rockett; WC Willett; GA Colditz

Milk, Dairy Fat, Dietary Calcium, and Weight Gain: A Longitudinal Study of Adolescents

Arch Pediatr Adolesc Med, Jun 2005; 159: 543 - 550.

"Children who drank the most milk gained more weight."

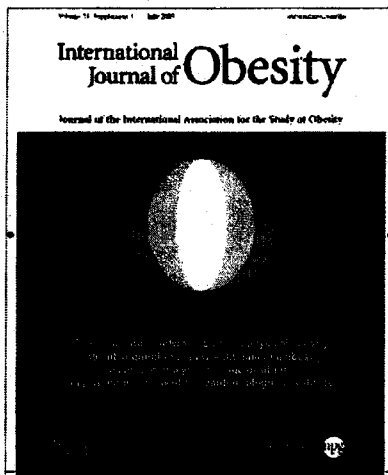
"Our analysis of a very large prospective cohort of children from all 50 states ... suggests that high intakes of milk, including skim and 1% milk, may provide some children with excess energy that results in an increase in body weight."

ARCHIVES
PEDIATRICS &
ADOLESCENT MEDICINE



JAMA
ARCHIVES

**FUNDED BY THE WORLD CANCER RESEARCH FUND
PUBLISHED 2009**



"the epidemiological evidence suggests that the consumption of beverages of any type is not associated with a subsequent weight gain and obesity, although the results are inconsistent."

SUMMARY

- Availability of added sugars calories has declined, since 1999, but obesity continued to rise;
- When addressing obesity, all calories count and 95% of calories come from something other than the category of soft drinks;
- Total calorie intake has increased while total energy expenditure has flat or declined over several decades;
- Epidemiological studies indicate there is no unique causal relationship between soft drinks and obesity.

CONCLUSION

It's Energy Balance!
All Calories Count – In and Out

Joint Informational Hearing

SENATE COMMITTEE on FOOD and AGRICULTURE

Senator Dean Florez, Chair

and

SENATE COMMITTEE on HEALTH

Senator Elaine Kontominas Alquist, Chair

Food and Beverages: Strategies to Recoup the Health Costs of Excessive Sugar Consumption

April 20, 2010

Sacramento, California

SENATOR DEAN FLOREZ: It's a busy day here at the Capitol with regular voting occurring, and so, I will tell you ahead of time that we may make a pause here and there so I can vote in the numerous committees that are going on. I'm going to try to make it logistically easier for folks by giving some folks some notice of when we're going to leave.

Thank you for coming. This is the Senate Food and Agriculture Committee along with the Senate Health Committee. Today's topic is "Strategies to Recoup the Health Costs of Excessive Sugar Consumption." Obviously, if anybody gets really thirsty, just come on up and grab whatever your choice is in front of you in the room.

I would like to thank those who have traveled here to the State Capitol today. Obviously, today we're with medical professionals, soda industry representatives, and community health advocates to address the role that sugar sweetened drinks, like sodas and energy drinks, sports drinks, sweetened teas, etc. play in our overall diet, and, of course, we're going to be talking about its link to obesity.

I would like to thank my colleagues Senator Padilla and Senator Alquist. This is very much part of a continuing discussion that we began in Los Angeles. Today, we'd like to carry that discussion on a little further to talk about the effects of raising revenue to offset what is a \$41 billion health care cost to the State of California and to talk about diabetes and type 2 diabetes as well today.

At the hearing, obviously, we had not introduced Senate Bill 1210, which is our bill coming up in a few weeks, to tax sugar sweetened beverages at a rate of one penny per teaspoon of sugar to fund programs. Some of those programs, of course, deal with physical education and nutrition programs in the State of California.

A lot has transpired since the L.A. hearing. Of course, President Obama and Michelle Obama's move to get our children more active in the Let's Move campaign, obviously, is very, very important. And, again, adding more calorie content information to the front of beverage containers and vending machines, fountain equipment is an important step as well.

We'd like to recognize PepsiCo announced that it would also remove sugary drinks from primary schools worldwide, and I think that is, obviously, an industry heading in the right direction, so we're very thankful for that.

I can tell you that as we meet, states like New York, New Mexico, Colorado, Mississippi, Kansas, Washington State, and even cities like Philadelphia, are proposing very similar taxes and the question is why would California be any different? And what we're seeking is indeed something somewhat different. We're not seeking this as General Fund relief; what we're trying to do is find the relief in the right measure in terms of additional monies for after school programs and physical education programs.

I can tell you that from my view of the last hearing, the sweetened beverages were and are the single largest source of added sugar in the American diet. It's estimated that Americans consume 300 additional calories, more than it did three decades ago; roughly about have of that coming from sweetened beverages. And, of course, in the district that I represent, Fresno County, we have 53 percent plus children drink one or more sodas a day; in Kern County, 55 percent of children do; and in some counties, over 70 percent of adolescents drink one or more sodas per day. I would be talking about Tulare County. So even in the Central Valley, it seems to be one of the epicenters where we have a lot of childhood obesity, type 2 diabetes. These statistics are very alarming but the goal here is not to wait for these numbers to blossom; it's to have an open, frank discussion about the role of sugary drinks in this, what I would call, epidemic, at least in my district and, of course, in California.

We know that we are going to have some divergent views today. We know that there are various thoughts on how to achieve the goals that we're trying to achieve

here in California. But the purpose of today is the kind of hearing that I like; it is not an up or down vote on anything; it is a discussion. It is an opportunity to provide the consumers and the public with information on our current health crisis and what over consumption of sugary drinks does in terms of contributing to that particular crisis we have in California.

Let's go ahead and begin the hearing. We have some panels. And we'd like to start with Gail Woodward Lopez and Harold Goldstein as panel one. We're going to be speaking about the link between sugar and obesity. I do have some questions for both of you, but I'd like you to be able to make your presentation, and then, we'll hear from the industry and a few folks as well, who would like to make a few comments.

GAIL WOODWARD LOPEZ: Chair Florez, Members, thank you inviting me here today to share findings on the relationship between sweetened beverages and obesity. I'm the associate director of the Dr. Robert C. and Veronica Atkins Center for Weight and Health at the University of California, Berkeley, and author of a recent book on the dietary determinants of obesity.

The Center for Weight and Health is well known for its work in the area of child obesity prevention and particularly for evaluating programs and policies in synthesizing research to inform policy and action.

We all know that we are in the midst of an obesity epidemic that will have catastrophic consequences if not addressed. We also know that calorie intake has increased dramatically over the same time period that obesity rates have risen and this increase in calories alone is more than enough to account for the rise in obesity we have observed in recent decades. But obesity is not merely the result of eating too much of everything, it is also influenced by what we eat. Therefore, it is critical to identify the dietary factors that are contributing most to excess weight gain.

At the Center for Weight and Health we conducted an extensive, systematic literature review and found that sweetened beverages were the single dietary factor with the strongest evidence linking it to obesity.

Let me share with you the four lines of evidence demonstrating the link between obesity and sweetened beverage consumption:

The first line of evidence compares secular trends in dietary intake and obesity. The parallel rise in sweetened beverage intake in obesity is quite striking. Between 1977 and 2002 when obesity rates were climbing most steeply, Americans more than

doubled their intake of sweetened beverages (and this is not a coincidence). Sweetened beverage intake accounted for 43 percent of the increase in overall calorie intake during this time period. By 2004, Americans were consuming between 9 and 13 percent of their total calories just from sweetened beverages alone. Among food and beverage items consumed by Americans in recent decades, soft drinks are the number one contributor to our calorie intake.

The second line of evidence addresses biological feasibility and answers the question, how do sweetened beverages contribute to excess weight gain? How are they different from other foods? Researchers found that when we consume calories in liquid form compared to solid foods, we don't compensate with an equivalent reduction in the intake of other foods and beverages. One analysis of over 40 studies concluded that 91 percent of liquid calories are not compensated for; they're just added to the rest of our intake. Several well conducted studies have found a significant association between sweetened beverage intake and calorie intake. These extra calories will lead to weight gain if there is no equivalent increase in energy expenditure.

The third line of evidence includes over 50 studies that looked at the direct relationship between sweetened beverage intake and some measure of body fat or body weight. The majority of these studies, especially those of more rigorous design, found that higher levels of sweetened beverage intake were associated with higher weight. These results were consistent across all age ranges and ethnic groups examined and were especially strong for children.

The fourth and final line of evidence is the most powerful. These findings are from nine experimental studies where sweetened beverage intakes were either increased or decreased and the resulting change in weight is measured. Studies of strongest design, the majority of which were randomized controlled trials, showed that reducing intake of sweetened beverages resulted in measurable and significant decreases in body fat. Conversely, when sweetened beverage intake was increased, subjects gained weight, up to 3.5 pounds in just ten weeks.

So do sweetened beverages cause excess weight gain? The evidence is quite compelling. There are several well established criteria that must be met to establish causation. The evidence we reviewed meets all of these criteria and therefore supports a causal link between sweetened beverage intake and obesity. Compared to other dietary components, sweetened beverages stand out as a major contributor to the

obesity epidemic. The evidence therefore suggests that reduction in the intake of sweetened beverages alone could have a measurable impact on obesity rates and this impact could be quite large.

An analysis we conducted revealed that the increase in sweetened beverage intake between 1977 and 2002 was equivalent to 43 percent of the total increase in calories over that time period. Given that it is unlikely that more than 50 percent of that increase in calories from sweetened beverages was compensated for by reduction in other foods, we estimate that at least 22 percent of the weight gained over that time period was due to the increase in intake of sweetened beverages.

In conclusion, it is clear that not all calories are equal when it comes to obesity. The same number of calories provided in different foods has a different impact on how full or satisfied we feel and therefore how likely we are to continue eating. Further, most Americans have little room in their diets for empty calories such as those that come from sweetened beverages that do not provide any additional naturally occurring nutrients. These beverages, therefore, are either consumed in addition to healthier options, thereby leading to obesity, or they replace those healthy options, thereby reducing intake of other needed nutrients.

Some may claim that sweetened beverage sales are essential for funding services and programs and institutions such as our schools. However, our studies conducted at over 100 schools found that the profits were less than \$400 per year per vending machine, indicating that the schools are getting a very small portion of the dollars that students were spending on these unneeded and potentially harmful beverages.

So what can we do? Committees convened by the Institute of Medicine and the Centers for Disease Control and Prevention, as well as reviews conducted by our center and others, suggest that alterations in pricing and access are among the most promising strategies for changing dietary intake. Therefore, policies that increase the price and/or reduce access to sugar sweetened beverages are merited.

Thank you very much.

SENATOR FLOREZ: Thank you. We'll also have some questions for both of you.

HAROLD GOLDSTEIN: Mr. Chairman, my name is Dr. Harold Goldstein. I'm the executive director of the California Center for Public Health Advocacy, a non-

partisan, nonprofit organization dedicated to protecting the health of all Californians. Thank you for inviting me to speak here today.

In 1985, when obesity was first measured, less than 9 percent of Californians were obese. Today, that number has skyrocketed to 24.3 percent in just 25 years, almost triple. Overweight obesity and physical activity, as you mentioned, Senator, costs California \$41 billion annually. And as you just heard, sugar sweetened beverages have been the single largest contributor to the obesity epidemic in the United States.

So how did sodas become such a big part of the problem? Well, those little 8 ounce soda bottles from the 1960s have become 12 ounce cans and then 20 ounce bottles and now 33 ounce bottles as if they've been on steroids. Six packs have become 24 packs. Small cold glasses at restaurants have been replaced by free massive refills at every fast food outlet. And until recently, soda was widely available in California public schools, a change that was vigorously opposed for many, many years by the beverage industry.

And remember that one 20 ounce soda has 17 teaspoons of sugar. It's like drinking a piece of chocolate cake every time you're thirsty. Imagine putting 17 little packs of sugar in your coffee; how would it taste?

Now when I was a kid in the 1970s, back then soda was a treat for kids. Today, one-third of children aged two through five, one-third of children age two through five in California drink a soda or more a day. Children consume on average 175 calories of soda a day. The average adult in the United States drinks 50 gallons of sugar sweetened beverages a year—the equivalent of 39 pounds of sugar; that's the amount of sugar sitting right over there.

Having reviewed the overwhelming research linking sodas and obesity, the nation's leading health organizations have called for strategies to reduce consumption; organizations like the Centers for Disease Control, the USDA, the American Academy of Pediatrics, and the American Medical Association. About the only organizations that don't agree are the ones that make money selling these products. The beverage industry has responded with a script that's now very well known, much of which is quite similar to what the tobacco industry said when they were fighting public health interventions.

So I can't completely predict what industry is going to say today, but I'm guessing you're going to hear about ten things: two of which are going to be true, eight of which are going to be false.

First, industry is probably going to say that there are many things that contribute to the obesity problem and that's true, but that's not an argument to do nothing. We have to start where the science is strongest, and the science is without question, strongest on sugar sweetened beverages.

Second, they're probably going to say that just reducing sugar sweetened beverage consumption isn't going to solve the obesity problem. That even here in California there are studies that have shown that half of the people who are overweight don't drink soda. That's true as well. But the half that do drink soda are 27 percent more likely to be overweight. Again, there is no magic bullet. But that doesn't mean that you ignore the biggest culprit.

Then the industry is probably going to suggest that sugar sweetened beverages are not a significant portion of calories that people consume, and that's false. Sodas are the single largest source of discretionary calories in the American diet. They're far and away the biggest source of added sugar. And as Dr. Woodward Lopez just said, they've been the single largest source of new calories in the American diet over the last 30 years.

The industry is probably going to claim that sodas make up something like 5 percent of calories, including food supply data, which isn't what people report consuming (which is more like double or triple that amount).

Now industry, as they said in the Los Angeles hearing, is likely going to say again that a calorie is a calorie. That it's unfair to single out one product. Now that's false in two different ways:

First, unlike other foods, sodas provide no nutritional benefit. We drink soda like it's water, but it's not. And more importantly, there is poor compensation for calories consumed in liquid form. When you go out to lunch, you don't feel more full if you drink a regular Coke instead of a diet Coke like you would if you had an extra half sandwich, say. As mammals, we evolve consuming only two beverages: mother's milk as infants and water as adults and children. So here's the problem: It turns out that our bodies are not biologically equipped to recognize or respond to liquid calories.

Next, industry is likely to falsely claim that the science showing the link between soda consumption and obesity is unclear, as if the Centers for Disease Control and the American Medical Association, the World Health Organization have all been duped. They'll quote a few studies that show contrary results, many of which they paid for themselves, and they'll bring experts to testify who are or have been on industry payrolls.

Next, they're likely going to say that raising the price of sugar sweetened beverages is not going to lower consumption. Now that too is false. A scientific review of the literature has shown that for every 10 percent increase in soda prices, there's a 7.8 percent decrease in consumption. The beverage industry's own data suggest that the relationship is actually even stronger. I provided the committee with a copy of this November 2008 report from Beverage Digest which says, "Industry sources have long said that carbonated sweetened drinks are highly price elastic, meaning that price increases depress volume." The report also showed that consumers are very flexible. When one bottler raise prices and the other one didn't, consumers quickly switched products.

The beverage industry is also fond of citing a recent Rand study showing that current state taxes averaging 4 percent have no impact on children's consumption. But most of these are sales taxes and, of course, sales taxes don't reduce consumption. Consumers don't even see a sales tax until after they make their purchase and even then, they could never figure out how much of the sales tax is due to one product instead of another. As for the paper's conclusion that soda taxes have to be sufficiently high to see an effective consumption, as you well know, SB 1210 would impose a tax of a penny per teaspoon of added sugar; an excise tax averaging about 15 percent, four times the average rate considered in the Rand study.

Next, the beverage industry is probably going to say that soda taxes won't have the desired public health benefit. This too is false. They may point to places like West Virginia and Arkansas as examples where tiny taxes haven't solved their obesity problem. Well, here in California we proved beyond any doubt that taxing a harmful product is a critical component of a successful public health campaign. In 1988, California put an excise tax of 25 cents on each pack of cigarettes. Ten years later, smoking rates have gone down 30 percent, and lung cancer rates have gone down 19

percent—in ten years. The key was for revenues to go right back to schools and communities to fund prevention programs, just as SB 1210 would guarantee.

Next, the beverage industry is likely going to say that consumers don't want a soda tax, especially during a recession. This too is false. A public opinion poll conducted by the reputable Field Research Corporation was released today showing strong support among registered voters—56 percent compared to 43 percent if the monies go to support children's health. And while the beverage industry sometimes sounds like they're fighting soda taxes on behalf of poor people, this public opinion poll showed that low-income Californians support the soda tax by almost 2 - 1.

Next, industry will likely claim that government shouldn't interfere with the market price of their products. It sounds good, but it too is false. The government is already interfering with the price of soda by subsidizing corn and keeping high fructose corn syrup prices, and therefore, soda prices artificially low. You may not know that two-thirds of all high fructose corn syrup in the United States is consumed in beverages. You sure don't hear the beverage industry arguing to end their government subsidies.

Finally, industry will likely argue that efforts to reduce soda consumption are going to hurt business. This too is false. I know of not a single study suggesting that either tobacco or alcohol taxes have ever resulted in job losses. And certainly no studies have shown job losses in places like Arkansas and West Virginia resulting from their soda taxes. Once again, industry is crying wolf. Instead, a tax on sugary beverages will create a market mechanism to encourage people to buy beverages without all that sugar, beverages that are produced by these same companies. On the table on this side, beverages produced by the industry that have calories. On this side, beverages that consumers, given a higher price on one, will lean toward consuming instead; all to a public health benefit; all of which will have no impact whatsoever on industry profits.

In conclusion, for decades Coke and Pepsi have been waging a soda war, competing for customers as if one would win and one would lose. Well, it turns out, they both won. They both got more customers, evidently including a lot of two to five-year-olds. And we've all lost. Their products became the single greatest contributor to the obesity epidemic, and a unique contributor to that epidemic because our bodies are not biologically equipped to compensate for liquid calories.

So a soda tax would accomplish two important things. It would provide needed funding for schools and communities to counteract the harmful effects of sugar sweetened beverages by supporting healthier school food, more PE teachers, and clean drinking fountains. And, it would, once and for all, hold beverage companies accountable for the portion of the obesity epidemic for which they are responsible.

Thank you.

SENATOR FLOREZ: Just a couple of questions. You've pretty much covered a good amount of ground. But maybe you can both give me your perspective on the issue of liquid calories, again, versus, if you will, the solid food. We do have a reference point here where the difference between a slice of pizza or desert versus a Coke or a Pepsi or something sugary and not liquid form. What can you tell us in terms of that debate that we constantly are having here?

MS. WOODWARD LOPEZ: Well, I think the research is fairly clear that liquid calories, especially from sweetened beverages but probably from other liquids also, just are not as filling. To put it in layman's terms; not as filling as a solid. I think we kind of know that all intuitively, but the research has confirmed it. It's probably somewhere between only 9 to 50 percent are compensated for it. The research does vary in terms of the exact amount. But it appears like 50 would be kind of an upper limit for the amount that's compensated. So that's 50 percent of the calories—you're adding them to your normal intake. And we see this—researchers who study what we call satiation and satiety (that's the sense of fullness or soon to resume eating again), show that many different things effect how full you are, so this is not a new concept but has been in the literature for a long time. Things that have protein are more filling, so milk would be more filling than something that doesn't—a beverage that doesn't have protein. It's just an example.

SENATOR FLOREZ: Okay. And how much sugar is recommended as part of a balanced diet for children on a daily basis?

MS. WOODWARD LOPEZ: There is no recommendation for sugar. That's the beauty of eliminating sugar sweetened beverages; you have no biological need to take in sugar that's been extracted from cane or beets. There is a recommendation for discretionary calories, and those are those kind of empty calories—added fats, added sugars, or foods naturally high in those substances. That usually tends to be anywhere from about 200 to 300 calories depending on the age and the energy. Of

course, if you are very physically active you could do more. So that one 12 ounce beverage would take up half of that. For girls, I think that would take up more than half of it, so that means they couldn't have a steak; they couldn't have a hamburger; they couldn't have any foods—or cheese, which is high in fat. So probably sweetened beverages is the worst place to get those extra calories because it's not providing anything else like those other foods I've mentioned which actually do provide some other nutrients. Sugar sweetened beverages provide none.

DR. GOLDSTEIN: I should say, the American Heart Association came out with their report last year that suggested that Americans consume 5 to 9 teaspoons of sugar a day, whereas we're now consuming 22 teaspoons of sugar a day. You subtract 5 from 22, you get 17—that's the soda a day that Americans are consuming.

I should also say that as Dr. Woodward Lopez is describing, calories consumed as liquids contribute differently to weight gain. The evidence of juice consumption is somewhat different. While juice has a lot of sugar in it, the public health research shows that juices are not being consumed at the level, at anything close to the level of other sugar sweetened beverages in large part because they're so expensive.

Is there anything else you want to add to that?

MS. WOODWARD LOPEZ: The research has not shown the relationships. So the lines of evidence I looked at—we actually looked at those for juices. We have not updated it. It was a few years ago. And we only found on—two-year-olds with apple juice was the only juice product that we could find that had any relationship to obesity, so we actually dropped pursuing that. And, we were perfectly open-minded. The evidence just isn't there for juice and it makes sense because juice does have other nutrients. Juice is more expensive and it has other substances that make...

SENATOR FLOREZ: And what is the group that seems to consume the most sweetened beverages—age group?

MS. WOODWARD LOPEZ: That would be teenagers, especially teenage boys. But it is startling; some of the biggest increases have come in the two to five-year-old age range. Although they drink less in absolute quantities, I think it's very startling how much they are drinking; that they're drinking sugar sweetened beverages at all at age two.

DR. GOLDSTEIN: As part of the bubbling over study that we released last year, it showed again that 41 percent of two to eleven-year-olds drink a soda or more a

day. Among two to five-year-olds it's 33 percent. Among Latino two to five-year-olds it's 42 percent who drink a soda or more a day.

SENATOR FLOREZ: Wow! Obviously, the bill doesn't do much on the side of trying to legislate some strict guidelines for the age group of two to five. Which seems to me, that's a lot of sugar if a small kid is downing one Dr. Pepper, for example. And I've seen that. Obviously, all of us have seen that at some point.

What does, from your perspective, the pushing of this tax on sweetened drinks do, and why is it important in California now? I know you both kind of alluded to that. But in the end, that's really the question of the day. Is what will it really accomplish; what will it really do from your on-the-ground perspectives?

DR. GOLDSTEIN: Well, again, I think it does two very important things. I think it uses a market mechanism to shift people from buying the products on the left to the products on the right in a way that is—there is no reason to believe it's going to impact company profits at all. The same company make the same—the whole range of products. And at the same time, it holds the beverage industry accountable for its portion of the obesity epidemic and uses that revenue to address the problem that's been caused. At the very least, all of these calories that our kids are consuming have to be burned off and yet California public schools, by-and-large, are not providing physical education. They don't have the funding for PE teachers; for equipment. At the very least, we should be taking revenue from this tax so that we can hire the PE teachers we need so that kids can burn off all of those soda calories—at the very least.

SENATOR FLOREZ: Just lastly: In terms of the switch, from your perspective, of sugary drinks to another product. For example; Coke is talking about the new formula or the reformula that tastes just like regular Coke—it's a zero product—is that something that you support? I know water would be the preference, correct? More water would probably be the preference.

DR. GOLDSTEIN: Coke Zero.

SENATOR FLOREZ: Yeah, and some of the alternative products that, in essence, may taste exactly the same now. I assume that's the marketing edge here; it tastes just like Coke. I just saw a commercial on it the other day. It tastes exactly like it but with zero effects. Is this something you see that's a positive, the reformulization of some of these items? Just your perspective.

MS. WOODWARD LOPEZ: Well, I would ideally like to see people switch to water, and, especially for children, milk, and that those should be the primary beverages.

In terms of obesity, zero calorie, artificially sweetened drinks may help/don't hurt _____ from the literature. But there are ongoing concerns about artificially sweetened beverages, and especially we're concerned with children. So it's not a highly desirable alternative, but in terms of obesity, it would be neutral..

DR. GOLDSTEIN: As you're suggesting, the beverage industry is coming up with new products all the time; stevia sweetened beverages. My wife and son came home the other day with a mint flavored water. Great! You know, again, I have complete faith in our friends from the beverage industry that they're going to come up products that have no calories; that aren't going to be subject to the tax. And in that way, again, this legislation is going to push the market in a way to help Californians live healthier lives.

SENATOR FLOREZ: Thank you both. Appreciate it. Thank you for your testimony.

Let's have panel 2 come up. That would be the "Industry and Economic Perspective." We have Danielle Greenberg; Robert Achermann; Julian Canete; and Samantha Dabish. Who would like to start?

DANIELLE GREENBERG: I guess I'm first on the list, so I'll start.

SENATOR FLOREZ: Thank you.

DR. GREENBERG: Good afternoon, Chairman Florez, Committee Members. My name is Dr. Danielle Greenberg. I'm a nutritionist with PepsiCo, where I've worked for the past nine years. Prior to joining PepsiCo, I served on the faculty of Cornell University Medical College for 15 years, doing research in obesity and the control of food intake.

I want to thank you for the opportunity to be here today.

We can all agree that obesity is a serious health issue. As a leading food and beverage manufacturer, PepsiCo knows we have an essential role to play in helping to find solutions to this complex problem. We have worked for decades now to reduce calories in our products and help consumers to make healthier choices. Let me give you a few examples:

In the past five years, the average number of calories in the beverages we sell in the United States have decreased by 11 percent. This is a significant achievement and a notable statistic in light of the backdrop of increasing obesity rates and we want to ensure that there's a continued trend towards lower calories. In this line, PepsiCo announced a significant commitment last month, which is that in our global beverage portfolio we are going to reduce sugar content by 25 percent by the year 2020. We also made a similar commitment on our food products, to reduce sodium by 25 percent, and to reduce saturated fat by 15 percent. And, we're going to report the progress on these initiatives on our websites to make sure that we have transparency and accountability.

We've also taken significant steps to help raise consumer awareness about the calories in our beverages. For many years now, as you noted, we have shown the calorie counts for both the full container and the 8 ounce serving on the back panel (that is on the "nutrition facts" panel) for portion sizes smaller than a liter. And, we've provided complete nutrition, ingredients, every information you could possibly want, on our website, *pepsiproductfacts.com*.

Now, in support of the initiative you mentioned, Michelle Obama's Let's Move; we have committed to the Clear on Calories initiative, where we're going to display the calories on the package on the front of all of our beverage containers and on vending machines and on fountain equipment at restaurants. And these sweeping changes are being coordinated with the White House and the FDA and will be implemented, importantly, in the next two years, which for something this large is really a huge commitment.

The groundbreaking Clear on Calorie labeling initiative is a good example of the progress that can be made through collaborations between industry and government. At PepsiCo we strongly believe in the power of such collaborations and we work hard to find opportunities to join with other industry members, government, and non-governmental organizations. For example, as you also mentioned, we collaborated with the American Heart Association and the Clinton Foundation, to completely change the beverages available in schools. Through this effort we have voluntarily removed full-calorie soft drinks from schools throughout the U.S. and now offer a range of lower calorie, smaller portion size drinks which resulted in an 88 percent decrease in the calories available in schools since the year 2006. And in another

example, PepsiCo's chairman and CEO, Indra Nooyi, is playing a pivotal role in the Healthy Weight Commitment Foundation, a collaboration of more than 60 retailers, food and beverage manufacturers, non-governmental organizations, and educators who are working to develop solutions to help reduce obesity, particularly among children.

We are a company that continues to encourage nutritionists and scientists, like myself, to make a difference; a place where my colleagues and I are proud to work; a company that seeks to partner in efforts to find common sense solutions to complex problems like obesity. We hope that sound science and common sense will prevail as industry, governments, and non-governmental agencies work together to change American habits not only in what we eat and drink, but also in the calories we expend in physical activity.

In troubled economic times like these, we can understand the appeal of attacks that produces revenues and purports to promote health. However, there is no scientific or medical evidence that a beverage tax will reduce obesity. There is ample reason to believe that such attacks would have dire economic consequences to local retailers and residents, putting hundreds of well paying jobs at risk and saddling the middle class working people with yet another tax burden at a time when they can least afford it.

Thank you very much.

SENATOR FLOREZ: Okay. Next.

ROBERT ACHERMANN: Mr. Chairman, I'm Bob Achermann on behalf of the California/Nevada Soft Drink Association. Our member bottlers produce and distribute a wide variety of non-alcoholic beverages, including carbonated soft drinks, water, juices, teas, and isotonic beverages among others. We appreciate the opportunity to make comments this afternoon.

A tax on soft drinks, juices and other beverages, in our view, unfairly lays the blame for the complex problem of obesity on the consumption of one particular type of product and perpetuates the myth that taxing those products will make a difference in fighting obesity. The only two states with an excise tax are Arkansas and West Virginia. How are those states doing in their fight against obesity? Arkansas has the tenth highest obesity rate and West Virginia has the third highest rate in the country,

according to a 2009 report by the Trust for America's Health and the Robert Wood Johnson Foundation.

The problem of obesity is an important one for our society to overcome. But this type of tax focuses only on soft drinks as a cause for obesity and not on total diet and exercise or other possible factors, be they environmental, genetic, or others which may contribute to obesity and require a broader response than a beverage tax.

I'd like to briefly tell you about some of the initiatives that the bev industry has committed in order to make an impact on the problem of obesity. Some of those have been discussed already this morning and I won't belabor them. The School Beverage Guidelines, which have been adopted nationwide, have now resulted in the removal of full-calorie soft drinks from schools; replaced them with other beverages that provide healthier alternatives in the school environment. Those guidelines have been implemented this year in all 123,000 public and private schools in the United States. A final report will be issued later this month.

In 2008, the beverage industry implemented our Global Marketing Policy. The policy applies to all non-alcoholic beverages other than water, juices, and dairy-based beverages. Our commitment is to not advertise or market these beverages to audiences primarily comprised (50 percent or more) of children 12 years and younger. This advertising and marketing applies to the following media: television, radio, print, internet, phone messaging, and cinema, including product placement. It is the first industry specific global marketing standard of its kind.

The School Beverage Guidelines and global guidelines are an example of our commitment to be responsible to our consumers and to others. Another example is our member participation in the Healthy Weight Commitment Foundation. The goal of the Healthy Weight Commitment Foundation is to help reduce obesity, particularly childhood obesity, by 2015, by helping people achieve healthy weight through energy balance—calories in and calories out.

The work of the foundation focuses on three critical areas where people spend their time: the marketplace, the workplace, and in schools. In schools, the foundation's Healthy Schools Partnership integrates nutrition education and physical education in a school-based curriculum to help children develop healthy habits. The partnership successfully piloted the curriculum in schools in Kansas City, Missouri, and will expand to schools in other cities this year.

Companies in our industry also engaged in numerous health and wellness initiatives with local, state, and national nonprofit organizations, like the Boy's and Girl's Clubs of America and the YMCA. These initiatives are designed to teach children and adults the importance of making good decisions regarding nutrition and health. The companies are also engaged in other initiatives to highlight the importance of making informed choices, like Coke's front-of-pack-labeling and Pepsi's Smart Spot program.

But we continue to step up to be part of the solution to obesity, reaching beyond America's schools. We now, as earlier indicated, supported First Lady Michelle Obama's Let's Move anti-obesity campaign; committed to fully display calories on our beverage products in a variety of formats, including on the container, on vending machines and fountain machines. The Clear on Calories initiative will provide consumers with clear and easy to understand information on their beverage choices. We are closely coordinating with FDA to implement this initiative, which goes well beyond what's required by the agency's food labeling regulations. It means within two years, every time consumers touch one of our beverages they will have the calorie information at their fingertips.

In her remarks in at the launch of her Let's Move initiative, the First Lady acknowledged our industry stating, "This is exactly the kind of vital information parents need to make good choices for their kids."

We offer a wide range of products, some of which are displayed thoughtfully on the tables behind me, including zero-calorie and low-calorie sodas, to 100 percent juices and beverages with varying ranges of calories. The fact is that today more and more Americans are drinking our no- and lower-calorie beverages than they did 10 or 15 years ago, resulting in a 21 percent decrease in the calories per ounce produced from 1998 - 2008; that's across our entire beverage portfolio. Yet, obesity and overweight continue to be an epidemic in this country. That's why we as an industry are working to educate consumers about the importance of living an active, healthy, and balanced lifestyle. Our industry is committed to being part of the solution to the issues of obesity, particularly childhood obesity. We welcome the opportunity to continue this work with members of the committee and propose solutions to educate, inform, and benefit Californians of all ages and backgrounds.

In our view, a soft drink tax is not the solution to combat obesity and will only lead to higher prices for consumers, an additional tax burden for residents of the state, and potentially lost jobs.

We appreciate the opportunity to discuss these issues and I'm happy to answer any questions.

SENATOR FLOREZ: Thank you.

JULIAN CANETE: Good afternoon, Mr. Chairman. Thank you for the opportunity to present our comments. My name is Julian Canete. I'm the executive director of the California Hispanic Chambers of Commerce.

As you know, the State Hispanic Chamber represents the interests of over 700,000 Hispanic owned businesses here in California. Through our network of more than 65 Hispanic chambers and business associations, we are the largest regional Hispanic organization in the nation. Our members provide hundreds of thousands of jobs across the state and are acutely attuned to the critical issues of economic development, employment, education, and health care here in California. The issue of obesity and how we as a state and a nation approach the challenges it presents, has implications in all of these arenas.

Our members agree that obesity is a serious issue in California and throughout the nation. It is one that particularly affects the Latino community.

If we are truly to reduce obesity, however, a tax on soft drinks and other beverages in California isn't the answer as it simply won't work. A tax will not make Californians healthier. In fact, it could have an adverse effect on small businesses. If you consider two other states that have such excise taxes in place on soft drinks (West Virginia and Arkansas), you'll see that they have among the ten highest rates of obesity in the nation.

Our communities do not want our elected officials using the tax code to tell them what to eat or drink. An example: A couple of years ago in Maine, the government imposed a tax on beverages to pay for state health care programs. A few months later, the Maine voters rejected the tax by a 2 - 1 margin.

In today's economy, small business owners and retailers, just like our hard working families, are struggling. They work hard to provide food and beverages at an affordable price to their consumers, and as well, provide food for their families at a

price they can afford. Adding the additional burden of a tax to items in people's grocery carts hurts businesses and families alike.

Taxing certain products to solve our state's obesity problem will do nothing to teach us how to live a healthy lifestyle. A better approach to solving this problem is through widespread education about balancing our calories and getting regular exercise.

The State Hispanic Chambers support the work of the beverage industry in developing and implementing the national School Beverage Guidelines as part of a broader effort to teach our children about the importance of a balanced diet and exercise. These guidelines remove full-calorie sodas from all schools and provide more low-calorie, nutritious and smaller portion beverages. The results are reverberating through our communities nationwide. In the past five years, beverage calories available in schools nationwide have dropped by 88 percent, while shipments of full-calorie soft drinks are down 95 percent in all schools. These are the quantifiable results that have meaningful impact in our schools and with our kids and families across the country.

As California policymakers seek solutions to the obesity issue, the California Hispanic Chambers of Commerce encourage you to adopt policies that are based in both science and common sense. We simply cannot tax our way to better health.

I thank you for this opportunity today to discuss this serious issue about obesity.

SENATOR FLOREZ: Thank you.

SAMANTHA DABISH: Good afternoon, Chairman Florez and other Members of the Committee. My name is Samantha Dabish. I'm the vice president of Government Relations and Community Outreach for the Neighborhood Market Association. For those of you unfamiliar with our group, the NMA is a nonprofit trade association dedicated to empowering the independent retailers throughout the west coast and we represent over 2,000 retailers in the grocery food industry in the state of California.

The NMA serves all independent retailers and believes strongly that they are the cornerstones of the entrepreneurial spirit and the backbone of the economy. This innovative spirit and drive comes from many family operated independent businesses that employ over 20,000 people. The NMA proudly represents them and we do

everything that we can to help them succeed. That is why I'm here to speak to you today and to be the voice of the independent retailer.

Taxing soft drinks, juices, and other non-alcoholic beverages alone will not solve the very serious problem of obesity. However, in this very touch economic climate, it will only make it harder for consumers and retailers to make ends meet. Not to mention, we are concerned with any tax proposal that would require retailers and the clerks that they employ, to calculate a tax based on the amount of sugar in a beverage. This is a nearly impossible task that would both be burdensome and costly.

Many consumers rely heavily on our members for the basic food needs and for their refreshment beverages that they enjoy. A beverage tax will increase costs to retailers and consumers and cause severe economic hardship to independent retailers across the state, which can very realistically lead to loss of jobs. Furthermore, consumers can't afford another tax on beverages on top of CRV and the recently increased sales tax. It will hurt lower income families the most because percentage wise more of their income is spent on food.

The majority of our customers don't want a tax on their juice drinks, sodas and teas. They understand that we can't tax our way to better health and nobody wants government in their grocery cart or at their kitchen table. California families are already struggling in this difficult economy. There could not be a worse time to ask them to pay more for the products they consume by raising the grocery prices.

If we as Californians really want to have a significant effect on the state's obesity rates and financial challenges, we need to look at a comprehensive solution that will truly have an impact on our citizens, not simplistic approaches targeting one portion of the items in the grocery cart for additional taxation.

On behalf of California's independent retailers, thank you for giving me the opportunity to express our concern about addressing obesity and doing so without causing unnecessary harm to the industry.

Thank you.

SENATOR FLOREZ: Thank you. Okay. This is one of those break times where I need to go take a bill up. But let me just ask a couple of questions and then, if we could, we'll just rehuddle in about 15 minutes. Is that okay?

I'd like you to think about something on the break and that is when I come back I'd like you to tell me what your comprehensive solution is in detail and not a

simplistic reading of prose that in essence sometimes doesn't really get us to any sort of solution. Let me give an example for one: Were all of you in favor of taking sodas out of schools?

UNIDENTIFIED: Absolutely.

SENATOR FLOREZ: You were? So if I read the analysis, the beverage industry was in support of that, I doubt that.

MR. ACHERMANN: I feel like I want to respond to that.

SENATOR FLOREZ: Sure. You're touting it today and so I'm just wondering how supportive of that—I mean, in hindsight, I guess, it sounds really good, but were you, at that time, in support?

MR. ACHERMANN: We did oppose legislation dealing with high schools. We thought there were better ways to approach that. We thought local control was a better option. The world is a different place than it was five years ago.

SENATOR FLOREZ: It is because the Legislature moved.

MR. ACHERMANN: And the industry moved collectively without the threat of legislation throughout the country.

SENATOR FLOREZ: You were drug. You were drug through that. You didn't move ahead of us. We're looking for proactive solutions. And after we did remove, and let me use some of your statistics, an 88 percent decrease in calories available in schools and by 2020 reduction of sugar of 25 percent. I mean, this all sounds like lofty goals going forward, but I'm trying to understand—how many jobs did we lose due to the fact that we took sodas out of schools? This was the Chambers' argument, "we're going to lose a lot of jobs." So, how many jobs did we actually lose when we took sodas out of the schools?

MR. ACHERMANN: I don't have anything definitive to give to you but I can probably show you some numbers...

SENATOR FLOREZ: Well, that's a pretty important stat if you're coming into a committee arguing that this is going to cost jobs, lose jobs, make the job situation worse, make the economic climate worse, when indeed we moved to an 88 percent decrease or are moving towards this massive change in our schools. We took a lot of products out of schools. That means a lot of folks that should have lost jobs. What were the jobs? I mean, you're the Chamber; what are they?

MR. CANETE: On the break I'm going to call our offices and see where we were.

SENATOR FLOREZ: I'd like to know how many jobs were lost due to one of the most far reaching initiatives by a Republican governor to take Cokes and Pepsis and some of these items out of our school.

DR. GREENBERG: That was a voluntary effort. That was not in response to the governor. That was something that was done by the beverage industry.

SENATOR FLOREZ: Shall we bring in Senator Escutia to talk about her bill?

MR. ACHERMANN: Senator, you're correct. We did have concerns with that approach. She's talking more on a national basis.

DR. GREENBERG: I'm sorry. I'm not from California.

SENATOR FLOREZ: Just so you know; it was California led and everybody kind of read—it's kind of hard when you have a mega-celebrity saying that this is something that's really important for our kids. And my very good friend and colleague, one of my best friends, Senator Escutia, pushed this bill for years and years and years. So we're glad the governor adopted that, but it seemed to me that with such a far reaching move to take all of these beverages out of schools, that we should have lost just tons of jobs. I mean, this just should have, sort of, devastated the industry.

MR. CANETE: Well, I know one of the concerns on that bill was the removing of vending machines. And the loss wasn't necessarily a job related loss, but it was more of a loss of school income to the student activities funds, etc.

SENATOR FLOREZ: Since you're speaking about that, you've mentioned earlier, your words were "common sense" and "science." What should we do to, in essence, implement some of the things you've mentioned? You said, "exercise" and "more student education." How do we pay for that?

MR. ACHERMANN: Do you want that answered now or after the break?

SENATOR FLOREZ: Rather than me asking you a million questions, I've allowed you to make statements, so I'm just kind of circling key words that some of you have mentioned, like "simplistic approaches," "common sense," "science," a very laudable diatribe about how important it is that we have now moved with Ms. Obama, who I think is great, to move to this new standard, and we're talking about all the schools that are pulling this out. We've talked about a whole host of proactive measures. But it seems that hindsight was that the Legislature, at least here, really

had to drag you folks over the line to get you to participate. And here we have a bill that's trying to drag you over the line to participate again, and so, I don't know, in ten years we look back and we go, "No, in fact we think this tax will actually reduce consumption." I mean, at what point do you proactively give us a solution that isn't just based on some of the things that we're already doing, in essence? There are things that we're already doing, correct?

MR. ACHERMANN: Well, you've asked a series of questions. I think, first of all...

SENATOR FLOREZ: So think about them and I will come back in 15 minutes. We'll start the panel again when we reconvene with you giving us your comprehensive solution to this problem—comprehensively. And if you're going to talk about education and programs, I'd like you to tell us where we get the money to do that—that would be kind of key. And if you can tell us how many jobs we've lost when we pulled all these cokes out of schools, that would be a good statistic, as well, since we're talking about job losses. And then we'll come back and talk a little bit about your view in terms of what is healthy for a child in terms of their allowable sugar. I'd like to get your perspective. And what is the industry doing for those two to five-year-olds that are popping those Dr. Peppers? Where do you stand on that?

So we'll come back in 15 minutes and more discussion.

Okay, let's reconvene. I'm sorry, 15 minutes turned into almost an hour-and-a-half plus. Let's go ahead and pick up where we left off with the panel for industry.

I left with a couple of questions: How many jobs did we lose?

MR. ACHERMANN: Let me talk a little bit about that issue. We didn't do any hard and fast calculations; we just talked to a few people. It was probably a couple of hundred jobs in the two major brands. That's a very small percentage of the overall soft drink industry, so it's not that huge.

SENATOR FLOREZ: What was the impact of the jobs lost versus the amount of calories decreased?

MR. ACHERMANN: I wouldn't be able to make that estimate. Most of the products, I would assume almost all the products on the counter behind us, were not available at the time that transition was made except for maybe the diet...

SENATOR FLOREZ: But you guys pointed earlier to a reduction in—is it due to the fact that they're selling water now and so, therefore, you weren't selling it in your product mix earlier?

MR. ACHERMANN: I think you're selling less volume; you're not selling the same range of products. There were people that were in that channel of sales in terms of servicing the school customers, and so, those jobs were not necessary because the volume wasn't there.

SENATOR FLOREZ: But volume is up for the industry or not up for the industry? Coke—Coke's earnings came out today. How did they do on Wall Street?

MR. ACHERMANN: I was too busy preparing for the hearing, but I think that ...

SENATOR FLOREZ: Up. They were up. Even in a down economy, so I'm must wondering...

MR. ACHERMANN: Soft drink sales in general are down.

SENATOR FLOREZ: Are down?

MR. ACHERMANN: Are down.

SENATOR FLOREZ: How about your zero-calorie drinks and your water sales; are they up?

MR. ACHERMAN: Water is down. I mean, I can get you something more precise. I know in general that those sales numbers are down. They're impacted by the economy. Consumers are not making purchases they made before.

SENATOR FLOREZ: Okay. Has that accounted for job loss much more so than when we pulled drinks out of the schools?

MR. ACHERMANN: Well, in the context of what you're proposing in your legislation, that would be a substantial tax on our product and that would impact, we think, the sales. That's going to impact jobs. You're going to sell less product and you're going to have less people involved in producing and distributing it.

SENATOR FLOREZ: What would be the industry's position in terms of the amount of calories—the less calories coming out of your factories versus the amount of jobs lost? In other words, if you could actually produce product and you see a market moving towards zero-calorie products—you're moving in those directions, correct, anyway.

MR. ACHERMANN: I think we follow the consumer and as consumers demand different types of products, you see the proliferation of different types of alternative beverages. So, we are consumer driven. I mean, you could make the argument we drive consumers; we don't. We market our products. We make a variety available.

SENATOR FLOREZ: Price drives consumers, correct?

MR. ACHERMANN: Well, in terms of product substitution, speaking as a loyal regular soft drink consumer, I don't switch to diet based on price, and I think people are pretty loyal in terms of the products they like.

SENATOR FLOREZ: I'm very pleased with where the industry is heading. I'd really like to see more commercials. I don't see them in Spanish, unfortunately. There doesn't seem, at least if you're watching Univision, which a lot of my family is, you're not seeing the zero products being pushed as heavily as you are in major prime time types of events. Sports events seem to have more of the zero feel to them, starting to move in that direction. And even in some of the sports drinks, you know, the lite products, Gatorade Lite and some of these others. I mean, I think that's a great trend. And, clearly, it seems the trend is for a reason. And, of course we'll always argue whether it's the consumer driving the companies or whether it's the company's market driving the consumer.

Do you feel that the companies have—you can't speak for all the companies—a moral obligation try to move consumers a bit through your marketing to products that are more zero-based, less calorie driven? You mentioned some salt reductions as well. What's the industry's role in that marketing mix in terms of getting consumers to buy more? If I saw Kobe Bryant with a zero product in his hand versus Dr. Dre saying, "Drink more Dr. Pepper," I mean, they're speaking to certain types of consumers, certain types of—adolescents, for example, is our biggest issue. I do know that Dr. Dre isn't talking to my father because he doesn't know who Dr. Dre is. I do. And so, when you're saying, "Drink more Dr. Pepper," like the doctor, I get that. My kids get that. My teenage son gets that. My daughter probably wouldn't get it. It's probably out of her realm as a nine-year-old.

But I'm just wondering what role do you see the industry is moving in if it isn't moving in a tax direction (which, of course, you oppose)? We're going to have a big hearing on that in a week or so. We're going to have a lot of up or down vote on it. But what do you see the industry doing in order to—there's a reason all these states

are doing this. You've mentioned some of them being the catch-up on the General Fund type things. Our particular bill is not trying to catch up with the General Fund, but trying to put more money into programs that voters support. So if you saw the field poll and agree with it or not, 84 percent of the folks liked physical education; 84 plus percent of people like the types of programs that we'd all nod our heads and say we need more of. I think it was the Chamber who said, "We need more of these types of programs but we can't pay for them." And so, we're trying to figure out a way to pay for those. What does the industry weigh in on those types of efforts?

MR. ACHERMANN: Well, I think that we would respond in terms of adequate funding on a variety of programs that are the comprehensive solution, which include physical education and education of consumers, parents. Your question about two to five-year-olds: two to five-year-olds are not walking into stores purchasing products; it's their parents who are doing that for them. We don't market to children. We believe that people should be more aware of what they're purchasing and how it impacts their diet overall. I think that's what the kind of thing we've done in terms of the Clear on Calories initiative in labeling.

SENATOR FLOREZ: On labeling, for example, you would never put on your can, "Children five years and younger, not recommended," would you—on the front of your can? "Not recommended for children five and under"—a big warning.

MR. ACHERMANN: There's a lot of things consumer products could contain. I don't think we'd go there. We don't ...

SENATOR FLOREZ: Well, why not?

MR. ACHERMANN: We don't buy into the argument that there's anything inherently dangerous.

SENATOR FLOREZ: You don't believe that? You think it's okay for kids two to five to drink 17 spoonfuls of sugar?

DR. GREENBERG: On certain occasions; at a birthday party or something like that, I don't think it's something that I would say is alarming. But I certainly don't think that it's something that would be part of an everyday diet either. I think there's a real difference. But I think that's where educating the parents as to where discretionary calories fit in the diet; how they are; how much they are; and what's appropriate for a kid?

SENATOR FLOREZ: Got it. Let me ask a broader policy question. I mean, this is probably—do you believe, as the industry, from your perspective, that there should be some sort of limit to the amount of sugar a person, an individual, should have in their diet? Just as a threshold question; is there a limit?

MR. ACHERMAN: I don't think you can look at beverages in isolation. And when we're talking about caloric intake, that's one segment of what you put into your body everyday. Consumers, if they're dieting, consumers, if they're watching their weight, have to be conscious of food and beverages. So I mean, I think where we probably disagree with you is focusing on this product exclusively in the equation as a solution. And I think the industry has done a lot to educate consumers. We keep saying that word; I know it's been overused, but that's what we're doing. So you make better decisions about what you do. And if you are over-consuming it's a better solution too.

SENATOR FLOREZ: I understand. So you're saying, looking at the mix of things, is an approach that you would support. But, obviously, looking at these sugary drinks, which you heard in some of the studies were a good percentage of the new caloric intake—the 300 extra calories we're all taking in is a huge part of that. I mean, how would you refute some of the medical studies that have said this? I mean, you have medical studies as well, right? So, I mean, how do we get to some balance in terms of trying to get consumers to switch over to some of your other products; the ones right behind you; not to your left, but to your right?

DR. GREENBERG: I think we're doing that. I mean, you know, not just through marketing, but also through developing new products. That's how there's been a decrease in the number of calories produced. I mean, an 11 percent decrease in six years, that's really quite substantial. And we are, without legislation, moving further in that direction. Our commitment to reducing sugars by another 25 percent across the portfolio, that's a substantial commitment. This is something that we are looking to do and we are moving in that direction. And we're looking for broad-based solutions, that not just focusing on one product.

The weight loss industry is a \$30 billion industry. Pharmaceutical companies have invested enormous amounts of money trying to develop products that would help people with weight management. To date, it's very difficult. Obesity is a very complex

issue. Even Weight Watchers, which is the most successful weight loss program, the recidivism rate is something like 92 - 93 percent.

SENATOR FLOREZ: No, I understand. But in all of those programs and all of those diets and all of those various weight loss reduction programs, is there anyone you can point to that says it's okay to drink a can of Coke?

MS. GREENBERG: Yes, absolutely. Weight Watchers.

SENATOR FLOREZ: Really? Weight Watchers you can drink a can of Pepsi?

MS. GREENBERG: Absolutely. Yes, you can.

SENATOR FLOREZ: Interesting.

DR. GREENBERG: As your points, absolutely. You most definitely can. And furthermore ...

SENATOR FLOREZ: So what are you trading off for your can of Coke? A whole meal?

DR. GREENBERG: You're trading off exercise.

SENATOR FLOREZ: A whole meal?

DR. GREENBERG: No. You need to get your base nutrition first. But even in your base nutrition ...

SENATOR FLOREZ: And your base nutrition is based on solid food or liquid?

DR. GREENBERG: Both.

SENATOR FLOREZ: Really?

DR. GREENBERG: Yeah.

SENATOR FLOREZ: Sugary liquids?

DR. GREENBERG: No. No. No. That's not the base foods.

SENATOR FLOREZ: Okay. What's the base foods?

DR. GREENBERG: The base foods are things that fall into the fruits and vegetables, whole grains, other grains, and milk. Let's see, what did I leave out? I'm sorry. Protein is part of your—that's your legumes, your meats, fish, etc.

SENATOR FLOREZ: Alright.

DR. GREENBERG: So, you have those. If you have those, that comes out and you have the adequate amount that most people need, that comes out to about 1,200 calories a day; maybe 1,100. You have, even on a weight loss program, a few more hundred calories. On Weight Watchers, those are your points, you exchange them. If you exercise more you get more points.

There are studies. For example; there was a study done by Christine Williams, who is a professor of pediatrics at Columbia University, where she looked at inner city adolescents. Everyone got 1,500 calories a day. One group was permitted 150 calories as a treat type food. The other group had all nutritious food only. They all lost the same amount of weight. This was a weight loss program. In the weight loss program, the people drinking the Pepsi or the Coke were able to lose as much weight. That is where it's how many calories you're having. You definitely need to have the nutrients. No one would suggest otherwise. But there is room in the diet for treat foods, whether it's a soda, a cookie, a donut, a bagel, a glass of wine. Those are foods that are not meeting very many nutritional needs, but they are part of the pleasure of food.

SENATOR FLOREZ: Right. But getting back to the example that started this; the two-year-old, the five-year-old category, do they understand the Weight Watcher caloric tradeoffs?

DR. GREENBERG: Even the two-year-olds could have some. You wouldn't expect ...

SENATOR FLOREZ: Oh, don't go there. Don't say a two-year-old can have a Pepsi. I think you're going to lose ...

DR. GREENBERG: No. A two-year-old could go to a birthday party once a year and be given this much soda as a treat.

SENATOR FLOREZ: Right. But they don't give that much. Do you sell them that small?

DR. GREENBERG: A two-year-old? Who's going to give—I mean, what are you going to give ...

SENATOR FLOREZ: I've seen two to five-year-olds drinking one ...

DR. GREENBERG: Well, those are parents that need to be educated.

SENATOR FLOREZ: The statistics that were just mentioned earlier by the prior panel points to a group of two to fives ...

DR. GREENBERG: That's educating the parents.

SENATOR FLOREZ: Right. How do you do that in your marketing campaign? In every commercial I see, how are you educating those parents that may be drinking this whole can is probably not the best solution to future type 2 diabetes which is increasing substantially now? In California, particularly, more kids with

type 2 diabetes coming in at freshman year than at any other time probably in our history. How do you educate—what's your role there in terms of giving those parents that type of education?

DR. GREENBERG: Well, I think that's something that we could do more on. And I think that that's something where that's an opportunity for a public/private partnership where the not just—it's not just a matter of giving a kid a soda; it's also giving them french-fries; it's also giving them cookies or donuts or cake if having that everyday. I mean, there's ...

SENATOR FLOREZ: I agree with you.

DR. GREENBERG: There's education that's needed. And I do think the industry could play more of a role there.

SENATOR FLOREZ: Right. That's good to hear because I think, obviously, we're trying to figure out what role industry can play—bigger role. And, of course, the efforts that you mentioned earlier and the trend—I mean, we like the trend in the sense of where the industry is going, we just haven't kind of caught up with people in my district that drink one can a day and that's the—In the old days in my agricultural district everyone urged everyone to eat one can of almonds a day. Just one can a day is all we ask. And today in my district, which is, I think, one of the epicenters of type 2 diabetes, one of the largest statistics. And it's interesting because if you look at the poll that's been mentioned earlier, it's really a disconnect, if you think the education is so important, because most people in my district, in this poll, don't favor any sort of tax on sodas but yet it is a district that has—I think it's Coke or Pepsi or soft drink capital of California. It gets the highest rates of drinking these items. And a lot of it is the fact that it's so abundantly there and it seems to be kind of the drink of choice. And I'm just kind of wondering how does one turn that around from an industry perspective? I know Coke and Pepsi do marketing battle, but it just seems, from my perspective, that more could be done.

DR. GREENBERG: I think the one thing I can point to is the schools initiative. I don't know if they're playing these commercials here but on the east coast there is a commercial that the beverage industry put out that shows rivals and how we all got together to get full-calorie soft drinks out of schools. Is there a possibility for doing more? Absolutely. But I think that we as an industry have tried to step up to the plate here and I think we're continuing to do that. And I think that looking for

education opportunities and marketing opportunities where through the marketing we're educating, I think that's something that, certainly, my company would be very interested in and my guess is that the other companies would be as well.

SENATOR FLOREZ: Okay. And a lot of it is the marketing we see on television at the same time. You mentioned labeling and I asked you—the sizes seem to get bigger. I never see cans getting smaller.

MR. ACHERMANN: There is some of that going on now. Coke is coming out with a reduced size.

SENATOR FLOREZ: Are they?

MR. ACHERMANN: Yeah.

SENATOR FLOREZ: Is it for airlines?

DR. GREENBERG: No. No.

SENATOR FLOREZ: To save costs or what? It's going down in size.

MR. ACHERMANN: To consumer available package. To bring it down to 90 calories.

SENATOR FLOREZ: Because we went to a 20 ounce. I mean, that's an amazing amount of sugar if you think about—from Big Gulps to—and I know consciously people make choices to do that. And I guess the point of the bill is to say to some folks, my perspective, that if you're making that choice, you're not just making a choice for yourself, you're making a choice to, in many cases, increase the load for every taxpayer on the health care side—\$41 billion for it. So it isn't just you that's drinking that, so in my world you ought to be paying premium for that. People who are not doing that ...

DR. GREENBERG: Shouldn't they be paying the same premium for donuts, and shouldn't they be paying the same premium for the 1,500 calorie Big Mac, and the 1,700 calorie lunch at the Cheesecake Factory, and the other sources?

SENATOR FLOREZ: But are they doing that every day? Are you going to buy that every day?

DR. GREENBERG: Big Macs, etc.?

SENATOR FLOREZ: Every day? Is that an every day?

DR. GREENBERG: For some people.

SENATOR FLOREZ: Yeah. But when you have a Big Mac you normally don't have a Big Mac and a water. I mean, this is the discretionary calorie that's sold

together. It complements, right? There's a reason that—you guys support value meals, I suppose, right? In other words, you sell a Coke, french-fries and a Big Mac together; if not, we would sell them separately. It's kind of a lower cost ...

MR. ACHERMANN: Those are decisions made by people other than us in terms of how they market their products and how they package them. I mean, we're interested in the beverage business.

SENATOR FLOREZ: No. No. I get it. I mean, you do the bev. I'm just saying in general I think we are all moving towards, in many cases, those types of environments where it becomes very difficult to reduce. And in one sense, your companies that are getting to zero products, you're getting to smaller serving sizes, I mean, those are all good trends. I don't know if I've ever seen a Coke or Pepsi product I could pick up in a can that says, "7 teaspoons of sugar in it - no more." I think ...

MR. ACHERMANN: There is nutritional labeling in there. But to your point; you do have restaurant labeling now. You have fast food restaurants that will have menu labeling that will have both food and beverage calorie content there.

SENATOR FLOREZ: I get it. But you guys weren't for that bill either, right?

MR. ACHERMANN: We didn't oppose that.

SENATOR FLOREZ: Okay. I'm just saying; all of these efforts sometimes—we're trying to drag you across the line and there's nothing wrong with it. And I see you're moving somewhat in the right direction, but the bill is really about trying to challenge, in many cases, the industry to come up with—have you ever thought of pricing your own products differently in order to encourage some of the zero products?

MR. ACHERMANN: It's a very competitive marketplace, as you know, for beverages. And you see, especially with new products being introduced, very competitive pricing being used in order to encourage people to try the product, so I think that does go on. Do we look at distinguishing between full-calorie and diet products? I mean, consumers make that decision in terms of price points.

SENATOR FLOREZ: Right. That's why we're trying to put a tax on it—exactly our point.

MR. ACHERMANN: Yeah, but raising the tax on regular soft drinks that dramatically, is going to drive those sales down, not necessarily drive other sales up. And at the end of the day what's to stop—This is a very competitive marketplace for beverages. I think prices are pretty low compared to other consumer products. This

is going to drive them up. You may think that's a good idea; we don't think it is a good idea.

SENATOR FLOREZ: Well, yeah. Lower priced products on sugary drinks means more people buy them, so, no, I don't think it's a good idea. You do because you'll sell more volume. I get that. But I'm just trying to figure out why we are encouraging more consumption unless there are zero products. And I don't know how we do that.

Look, let me put it this way. We took Cokes out of schools, right. But if we were to go back and put Cokes and Pepsis and Sprites in schools that were zero calorie products, I mean, wouldn't you do better?

MR. ACHERMANN: Well, we actually have suggested that it's time for the Legislature to look at those list of permissible beverages. I mean, all those products back there for the most part, are not allowed on school campuses. And I think that's another good part of education, is letting students see that there are other things to drink.

SENATOR FLOREZ: Right. Or to get them used to a taste that's somewhat different than what their thought might be in terms of a diet Coke—taste and differential. I want you to know, we're not, like, against, I, personally, I'm not necessarily against the product as a brand; I'm more worried about the amount of teaspoons of sugar in a particular product.

So what would be the argument for the Legislature if you were to come back and say, "All of our products are all zero-based; none of them have any sugar in them; and we'd like to see them back in schools?"

MR. ACHERMANN: Well, we actually had the discussion with Senator Padilla (who has a bill on sports drinks), who wants to ban sports drinks and we've said there are now no calorie and very low-calorie sports drink options; don't you want to include that? And the comment back has been that, "No, we don't want to have high school students taste something artificially sweet because it's the equivalent of a gateway drug to sugar." I think that's kind of a silly argument to make, quite frankly, but that's ...

SENATOR FLOREZ: Well, you might have my support on that. I mean, if indeed we're being realistically talking about the amount of sugar in these things ...

MR. ACHERMANN: Right. It's about calories.

SENATOR FLOREZ: Right. But wouldn't it be an easier argument from the gateway type of argument to say that one costs more than the other so therefore you're incentivized to buy the product that actually costs less and it's a zero product? It seems to me that would solve it right there from a marketing mechanism perspective.

MR. ACHERMANN: Well, that's above my pay grade in terms of making decisions for the companies. But I assume they spend a lot of time and money researching the marketplace in terms of what works. It may be something they're thinking about.

SENATOR FLOREZ: And I get it. And the analyze everything we do here to figure out how we will impact their sales. So I mean, I'm sure every time there's another state or another legislator or another mayor that puts another opportunity to tax a product that one sees as non-beneficial, then the industry responds accordingly. I mean, in the marketplace government also and government regulation is part of the marketplace; it's what they react to. And a lot of what we're trying to do with this bill is to get the industry to continue to move in a direction (which you are, I believe) that says maybe we should move to products that are in essence zero-based and try to keep the taste, with those wonderful scientists that we have, exactly the same. I think it's a very interesting thing to watch. I think it's fascinating that the new marketing for Coke, for example, is "it tastes exactly the same." And I think it will be challenging to see if people will actually take that dive. And if they do, great. The question simply is, how do we get to the fact that right now we have kind of an epidemic going? I think, in my view, and I know you may blame other items, but I think this is the discretionary calorie that a lot of folks continue to partake in, and I'm just trying to figure out the best mechanism for that to occur.

Do you think, from what you heard earlier, to get your perspective, is there any sort of proof that there's a link between obesity and sugary drinks? Do you need anything more to tell us that there is a link or are you completely saying that there's not?

DR. GREENBERG: I think that to do the correct type of science you would need to do a different type of study than the ones that have typically been done. Because the ones that have typically been done have said, "Okay, I'm asking you how much soft drinks you eat and then I'm looking at how much you weigh." And if you ask the same question of how much Big Mac or how much (I'm sorry, I don't know the

name of the equivalent Burger King thing is) how many donuts you eat; how many cookies you eat; how many french-fries you eat? and did the same analysis, you would then be saying that french-fries are the major cause of obesity. The types of studies that would be needed to do this would involve some statistical kinds of looking at something that I would call "a factor analysis," where you would essentially look at all food products and see whether or not one pops out. Those types of studies have not been done, at least to date.

SENATOR FLOREZ: But it seems to me given that that's your approach, I mean, you're the industry, I can imagine you doing those studies every week. Why hasn't the industry put out these additional studies that show the link? I mean, gosh, you come in here with ten studies that say ...

DR. GREENBERG: Those kinds of studies are not easy to do. They would require a great deal of data and they're not simple. But, my statements would be—to me if it was so simple that soft drinks were specific—everyone wants to be thin. No one wants to be fat. People would stop drinking soft drinks and that would be it. You go to a diet doctor, "Okay, no more soft drinks. That's it." It's just too simplistic. The problem is extremely complex. It's not a matter of one type of food or beverage and that eliminating that one type of food or beverage is going to solve the obesity situation.

SENATOR FLOREZ: Right. And so, why do you think we started with schools and why didn't we pull out other items out of our schools when we have a captive audience? Why didn't we ban certain foods on certain days, or certain types of foods that our cafeterias are feeding our kids? Why did we choose soft drinks and then now you guys have embraced it and said, "Man, this is something we really believe in?" Why did we make a choice there that you support that they not be in schools?

MR. ACHERMANN: I think that was a more comprehensive approach. There were also food and beverage centers that were part of Senator Escutia's bills. So I think it did address the food side. Unfortunately, the funding for the School Lunch program was insufficient to provide a lot of the healthier alternatives and they're still struggling with that.

SENATOR FLOREZ: Maybe if we took some of this tax and actually made our school lunches even healthier so it would be a comprehensive approach, as you mentioned, both drinks and food in our schools and then we capture everything. We

provide a little more than the National School Lunch Program in terms of better foods and salad bars and things of that sort in our schools. That's comprehensive.

MR. ACHERMANN: Well, we don't think it's fair, again, to target one product to solve this larger problem. Why did we do the beverage restrictions in schools? Because schools were a special place; less parent involvement. There was a desire that we eventually did embrace that that is something where you should look at caloric content more specifically. And to our earlier discussion and conversation, we think that should be revisited in terms of what's available, again, focusing on caloric content, not specific types of beverages.

SENATOR FLOREZ: Okay. Just to close a little more discussion on the industry marketing, then; so you see yourself maybe moving in a different direction? How about in minority communities, non-English speaking communities, in terms of some of the marketing that's occurring from the industry, do you see—I don't know what the statistics are, but I mean, are there diet products being marketed more so on television and radio than the current standard products that may not be zero-based types of products?

MR. ACHERMANN: I don't know specifically. I'll try to certainly get you more information about it. I mean, certainly the diet beverages are being promoted. I don't know how that cuts across different ethnic lines in terms of marketing, but we'd be happy to share what data is available.

SENATOR FLOREZ: Yeah.

MR. CANETE: I know, Senator, we've had some discussions with Coke and Pepsi in regards to their use of advertising in our Hispanic communities through our network of publications. There's more educational type material being printed and distributed out of the Hispanic communities as well.

SENATOR FLOREZ: Let me just ask a few more questions if I could. Talk to me about the—somebody mentioned earlier being the regressiveness of this; that's it's regressive on poor folks. Are we still arguing that in this day and age? Is this one argument of the bill? Do you stand by that? Explain that to me.

MR. ACHERMANN: Well, the less money you have in terms of disposable income, the more those taxes impact you. Food and beverages are discretionary. You could certainly argue that soft drinks might be more discretionary, but there's a

variety of products that contain sweetener, and so, you do impact people's ability to pay for their groceries and I think it hits you in the pocketbook.

SENATOR FLOREZ: And again, if we were trying to reduce the amount of sugary drinks people would partake, this would probably be one of those discretionary products they would leave out of the basket in other words, correct? They're not going to pick a six-pack of Pepsi over bread and baloney, right? I mean, if they are poorer folks are they going to say, "Well, we're not doing bread, baloney, whatever; we're going to do these two six-packs of Pepsi."

MR. ACHERMANN: Well, I think our response again would be, "Why are you focusing on one beverage product in this effort? If you really want to encourage a healthier lifestyle it's got to be broader than that. And I know this is somewhat repetitive but that's our answer there. And you know, we're already subject to sales tax, unlike other food and beverage products; we're already subject to CRV, which a consumer, like me, who uses my curbside collection bin, donates five cents for every 12 ounce can I buy. I think there's a significant tax burden already in terms of other positive influence in terms of solid waste management and in terms of sales tax revenue that the industry actually embraced when the whole snack tax was repealed.

SENATOR FLOREZ: Okay. One of the other questions before I let you all go: I also mentioned to give me your overall comprehensive plan for funding, if you will, or finding better ways for obesity prevention programs. How would we do that without this tax?

MR. ACHERMAN: I think the industry's response is that we are doing our part. It's not just our part that's a solution here. There is no silver bullet to this. If you look at us and say, "Well, what else could you do and why can't we raise taxes on your product to do better things?" Our response is, those are more General Fund obligations. Having properly funded schools, having a school lunch program that works is a bigger societal burden than just the beverage industry. I think that's how we would respond.

SENATOR FLOREZ: So how do we fund not just your industry, but other folks participating in this? So in other words, more taxes on other things that are—no tax at all? Then we can't fund anything, so how does it ...

MR. ACHERMANN: Well, we're not arguing against taxation. I would think a better approach—our industry would say a better approach to this is a broad-based

sales tax is a better approach than demonizing one product in this process. So if you want to fund ...

SENATOR FLOREZ: Doesn't that hurt the poor more? It seems to me it would. That's the Chamber argument. So I'm just kind of wondering why every time we want to do a targeted tax on maybe some folks that might be making triple digit net profits the argument is, let's do a broad general sales tax so therefore everyone pays. And so, when we do a bill that targets something and people argue, well, that's unfair because you're targeting folks and everyone should pay. The general sales tax seems to hit poor folks harder, particularly if they're making good choices, like they're saying, "Hey, the sales tax went up and I'm really not going to buy your product, but now I've got enough money to buy staples."

MR. ACHERMANN: Well, you made that decision from a policy point of view by not taxing food. It's not subject to sales tax. Our product is and any fruit juice less than 100 percent is taxable, so you're already generating sales tax revenue for that. The whole concept of taxation is probably broader than what I could respond, but I mean, it's one of the arguments people make about value-added taxes. That you could do a lot to eliminate the tax code, discrepancies in income (what's reported/what's not reported), and look at value-added taxes in terms of purchase, which is equivalent to sales tax. It has some merits.

SENATOR FLOREZ: Okay. That's good to know. Anyone else who would like to add anything? Thank you. We'll go onto the next panel. Appreciate it. We'll see you in committee when our bill is up. I'm sure we'll hear a lot of the same arguments.

Let's have panel 3, if we could; Michael Goran and Lisa Katic.

A very easy threshold question as you're coming up, since this deals with the biological consequences of sugar sweetened beverages. It's the same question I've asked just about everyone and that is, what's the daily allowance of sugar people should be having, particularly kids? And I don't know if you can base your presentations, at some point, at the end of that to that question. I think it's something we're interested in.

Thank you for joining us.

We can start with Michael, if that's okay.

MICHAEL GORAN: Thank you for the opportunity to continue this dialogue. I'm going to talk today a little bit about the actual metabolic and healthy facts of sugar

in the body beyond the argument of whether sugar causes obesity or not. I'd like to kind of turn away from the emotional baggage of that issue and focus more on the actual biological and metabolic effects of sugar on the body.

Obesity by itself affects multiple organ systems in the body. I had a picture of this, but it ranges from effects on the liver and the pancreas, the cardiovascular effects, and some of those effects are the results obesity itself, so the very effect of carrying excess body fat in your body does cause, through a range of metabolic mediating factors does lead to those diseases. But in addition, there are some effects of the obesity and some of the effects of the dietary factors, the nutrients itself, that directly affect obesity, so the combination of obesity itself and dietary factors may exacerbate each other. There's also the issue at play, I believe, that the effect of sugar in the body is probably exacerbated in the obese state, so no longer looking at the effect of increased sugar intake on a healthy body, we're now looking at the effect of increased sugar on top of an unhealthy body, or an overweight or obese body, where the effects are really exacerbated. And there's a couple of examples of that that I can tell you about.

One is the fact that as you become more overweight or more obese, you become more insulin resistant. That means it's harder for insulin to do its job of clearing glucose from the circulation. And the harder it is for insulin to be cleared from the circulation, the harder the pancreas has to work, and the pancreas has to work exponentially harder with the increased obese state. And on top of that, and this all becomes additive, there are other factors affecting minority populations specifically independent of obesity minority populations for reasons we don't quite yet understand and are more insulin resistant to begin with. So, all these factors make it harder and harder for the pancreas to do its job of clearing glucose from the circulation. So the more glucose that's coming into the circulation makes it harder and harder and that's the path to diabetes, because eventually the pancreas can no longer do its job. So in the lean state it's not as difficult for the pancreas to its job, but that becomes harder.

There's been some talk whether or not high sugar consumption in and of itself is damaging and causes disease. There are several large—thousands of subjects studied over decades and longer. There is a health study, for example, and other population-based epidemiological studies, have shown clearly that there are independent effects of soda intake on long-term risk for both type 2 diabetes and

cardiovascular disease independent of other nutrients, and independent of the effects of obesity. So again, these things are kind of synergistic in a sense, or at least additive.

We do know there are mechanisms, biologically, that link soda over the long-term. This is a long-term phenomenon but there are short-term effects. We know that consuming high amounts of sugar under laboratory conditions leads to things like high blood pressure, high circulating lipid concentrations, insulin resistance, and so on. These are the mediating variables that eventually contribute to those disease states.

I also want to talk a little bit about fructose and glucose, and particularly high fructose corn syrup which has been mentioned, because there is a distinctive difference between fructose and glucose which are very similar structurally. Chemically, they're both six-carbon sugars that look very similar but yet are handled by the body very, very differently. For example, fructose is much sweeter. Fructose is absorbed through a very specific mechanism in the gut, whereas glucose is more generally absorbable. Fructose also is metabolized almost exclusively in the liver where it can be a substrate. And again, especially in the overweight state it can be a substrate for what's called "de novo lipogenesis," which means new fat synthesis in the liver. So if you have an excess of fructose in the system and your fat stores are already saturated and you have an excess substrate coming in, that fructose will be reconfigured and packaged as fat in the liver.

And fatty liver disease is another emerging problem. In fact, the numbers for fatty liver disease are more frightening than pediatric type 2 diabetes. That's an increased deposition of fat in the liver which eventually leads to liver disease and liver dysfunction. That is directly attributable to increased fructose consumption, in particular; not exclusively fructose, but other sugars as well.

Just so that everybody knows, high sucrose, which is table sugar, is a disaccharide of a glucose molecule connected to a fructose molecule; whereas high fructose corn syrup is still a mixture of fructose and glucose, but it's a synthetic mixture of glucose and fructose derived from glucose, which is blended, typically, in a mixture of 55 percent fructose and 45 percent glucose.

But another issue at play here, I think, in this story, is the fact that since fructose is really the more damaging sugar, we need to have better information on

what the actual fructose content is. At the moment, we don't really know what the blend or the mixture of sugars is. The label just says, "sugars." We have to make an assumption that the blend is 55/45, but we really don't know that.

A typical can of soda has about 50 grams of sugar. Some people may argue that apples contain just as much sugar. I had a graphic to show you that an apple does have 15 grams of fructose versus a can of soda which has about probably double the amount of fructose and probably no other nutritive value, whereas an apple, of course, comes with lots of other beneficial things.

There has also been some discussion today about whether the effects of sugar in the body are reversible. I'd like to just briefly address those. We know from various intervention studies and trials, and we've done some studies ourselves looking at the question of whether you can reverse these effects, and the answer is that education and behavioral intervention alone is not going to be sufficient. You can't just educate or tell people that sugar should be reduced and expect it to be reduced. There are many other factors at play in the environment that are overpowering such as motivation, peer pressure, marketing, cultural norms and other factors that we've talked about. However, we do know that if you do reduce sugar that you can get the metabolic benefits of reduced risk for some of these things that we've talked about. So we have to look beyond education and information to really make a change.

Just to summarize: Sugar has detrimental effects on the body at various organ levels. Primarily, the things we're talking about are the pancreas: eventually leading to type 2 diabetes in the liver; contributing to fatty liver and fatty liver disease, and these effects, I believe, are more damaging against the background of obesity. So these are things we might not have used to worry about 20 or 30 years ago when obesity wasn't prevalent in the population, but because it is now, we have double reason to be really concerned about the effect of these things on the body.

Fructose has more damaging effects than glucose probably, and levels of its intake are really going unchecked at the moment and very difficult to determine.

Third, the effects of high sugar intake are reversible but it takes more than simple education, it takes a multi-level integrated approach, so we need a combined approach to tackle this problem.

So those are my main key points. I want to thank you again for the opportunity to discuss this with you and for the leadership here on this issue.

SENATOR FLOREZ: I appreciate the testimony. Thank you.

LISA KATIC: Good afternoon. My name is Lisa Katic. I'm a registered dietician and principle of a practice based in D.C., K Consulting. I specialize in food policy, communications, and education. And I just want to start out by saying that I've been working with the food and agricultural industries for most of my career. I've thought about and have been involved in developing strategies to combat obesity for most of my career. And, actually, I was prepared to come today to talk more about some of the programs that I think we're seeing put in place right now as opposed to some of the metabolic effects of sugar, but we can certainly talk about that after the fact.

But I think what's important to focus on is what I've seen in the past five years with respect to the programs and strategies that have been implemented to address this problem is really remarkable. What I saw ten years ago when I embarked upon trying to help companies and consumers with respect to obesity, we really were nowhere close to where we are today. We're seeing government, industries, schools, communities, health professionals; all of these groups are collaborating on these programs to try to reduce obesity rates in this country.

And I want to talk a lot about the most recent program that's already been mentioned, which is the First Lady's program. I think a couple of the things that are important to mention about that is it was, of course, launched in February. This is really the first time that I have seen a program as comprehensive. This program involves every one of the president's cabinet members. This is unprecedented for a comprehensive government program. It involves everyone from USDA, Health and Human Services, Housing and Urban Development, the Labor Department, the Education Department, and what that says to me is first of all, it shows how complicated this issue is and it shows how every single of these entities have to be involved to help solve it. I think it really shows that the President and First Lady certainly understand that and get that, and they're trying to put a program in place that's getting to the real problem.

I think the other thing that I saw in her program that was really encouraging, that sounds very obvious but I don't know if we've seen a lot yet, is targeting parents. She really understands as a parent, that parents are certainly busy. They don't have a lot of time, but they certainly don't have a lot of the tools that they need to teach

themselves and their children about healthy eating, so I think that's another really important cornerstone to this program.

I was going to talk about how the industry has jumped onboard with that, but I think we've already kind of covered that, so in the interest of time I'll not really talk about that, other than to say this: I think when you see programs, like the School Beverage Guidelines and this Clear on Calories and announcements being made, you know, in the circles that I run in with dieticians in the health professional community, they're really encouraged when they see stuff like this. And they, obviously, were congratulating the industry about making a commitment and showing change and progress, I think, moving in the right direction.

I think the next thing I want to talk about that we haven't heard about today that's another pretty landmark program that I personally think, and we can talk about it, will really show some results. This is the Alliance for Healthier Generations health care initiative. And we've already talked about their school beverage part of their program, but they launched this health care initiative in February of '09. And what is really critical about this program is for the first time ever, we're going to see consumers get access to primary care. They're going to get to see their doctors; have visits to the doctor focused on obesity; and they're also going to get access to the dietician's services and they're going to get reimbursed by insurance companies. This is something that members, like, myself, of the American Dietetic Association, have really been advocating about for years. I use the example that I can go to the gym; I can get a great workout, but when I go to the gym and workout with my trainer who's a professional, I get a much better workout and I see much better results. So in that same vein, when consumers can really sit down with their professionals, a doctor, a nurse, or a dietician, and get one-on-one care, I think is when we're really going to start to see some results.

And so, the Alliance made a commitment that—well, actually in the year that this has already been launched, we've already seen a million children have access to some of these visits. And their goal is over the next three years, 25 percent of all overweight children, which is approximately 6.2 million children, will have access to this kind of care. I think that's pretty significant. They also are committed to continuing to recruit insurance companies and hospitals and employers to join onto this program, which they've already had some significant support already.

So I wanted to highlight a couple of those programs because, again, I think when we talk about comprehensiveness, I think when we talk about getting away from simple solutions, I picked these examples because I think these are examples that show exactly that. They're short-term, but they're also very long-term, and so, I think we can't lose sight of both of those things.

I think I mentioned about the School Beverage Guidelines.

So in closing, I just want to say that I'm really encouraged when I look at some of the things that we're seeing implemented right now. I know that this is an epidemic and we talk about all the statistics, but until, again, we really start getting some programs in place that are giving consumers access to meaningful care, and that we focus on families, and that we are not taxing or banning foods for the sake of just trying to do something, I think that's when we're really going to see something that's going to work. When families work together to improve health and that they have access to the right tools that they need for themselves and their families, I think everyone is going to benefit.

I just want to thank you again for listening and the invitation to be here. I appreciate it.

SENATOR FLOREZ: Well, thank you both for putting it on the record. We very much appreciate it.

Let's go onto the marketing section. Katie Woodruff is here with us. Thank you. We'll be brief. We just have a few questions. You've heard most of the testimony.

KATIE WOODRUFF: Well, thanks very much for inviting me here today. I'm Katie Woodruff. I'm deputy director of the Berkeley Media Studies Group, and we are a project of the Public Health Institute.

For the last 16 years, we have been studying how public health issues are portrayed in the media and covered in the news. And recently we have been studying how soda and other sweetened beverages are marketed, particularly to young people.

And as your one woman marketing panel here today I was asked to do several things. I'm going to try to be as brief as I can but still address, I think, some of the key points that have come up.

First of all, in addition to what you've already heard about soda and sweetened beverages, their unique contribution to diabetes and obesity, I wanted to be sure to

leave everyone here today with the understanding of how beverage companies marketing practices also are unique in terms of other food and beverage categories. Their extensive marketing expenditures and practices really put them far beyond any other food or beverage category in terms of their pervasive and aggressive promotion of products to young people. Even though there are some steps in the right direction, there is still much that is still problematic. And a lot of this marketing happens outside of parental control or even awareness. And I, as a parent of two kids, absolutely agree that parents are the gatekeepers and it's very important to educate parents and get them involved in better decision making for their children.

I'm hopeful that the tax that you proposed will raise funds for more education campaigns, because right now the food and beverage industry really is by far the leading nutrition educator in this country. They spend so much money putting out messages about food products. And the messages that they give, especially on the soda issue, is these products are fun; they're exciting; they're a good source of energy; and there's very little balance in terms of other kinds of nutrition messages in them.

I think in order to understand how the impact of marketing on youth which what I was asked to address, I need to just briefly talk about how broad the marketing function really is. It includes not just promotions, like TV ads and digital campaigns, but the development and packaging of new products and the pricing of those products and the ubiquitous access to places where those products can be changed. And in all of these areas, the landscape on beverages has really changed significantly in recent years.

First, as we've heard, the range of product offerings has broadened dramatically. It used to be just a few flagship sodas, and now there is, as Pepsi says, "There's something for everyone." Really between energy drinks, sports drinks, sugared teas, flavored waters, all of these caloric, as well as non-caloric beverages, as Coca Cola calls it, "A whole world of choices." Just to be clear from our understanding Coke and Pepsi are still really the giants in terms of beverages. They dominate and account for 70 percent of the U.S. non-alcoholic beverage market.

Of course, as you know, portion sizes have increased dramatically. It used to be 8 ounces was the single soda serving, and now 20 ounces is the default. People do tend to consume whatever is in the package, so as the package volume has gone up, the sugar intake has gone up significantly as well.

At 7-11, in the soda fountain, the smallest cup size now available is the 16 ounce Gulp, which, of course, looks sort of quaintly modest next to all these other options, including the gigantic Double Gulp which gives you 48 teaspoons of sugar.

While we're on the product and packaging side, though, I did want to say I absolutely was encouraged and happy to hear about the Clear on Calories initiative that we heard about earlier today. I think that putting the calorie counts right on the package front and on the point of decision making at the soda fountain, is very important for people to have true information about the choices that they're making and I applaud the industry for adopting that. I look forward to having that logo so I can put that slide into my next presentation.

On the pricing front, research has shown that over the last couple of decades the pricing of soft drinks really hasn't changed very much while many other food categories, including fruits and vegetables, have become relatively more expensive. And this was from my local Safeway recently, Coke products on sale for \$3.33 for a 12-pack; that's 28 cents per can, which is ounce for ounce, cheaper than the cheapest milk that I could find at the store.

Does this low pricing encourage soda consumption? Well, many studies seem to think so, and the industry itself seems to think so. An industry trade publication reported that when prices of Coca Cola increased by 12 percent, sales dropped by 14.6 percent (that's from *Beverage Digest*), and this and other non-industry research on the price elasticity of sweetened beverages, indicates that the demand for these beverages is really quite price sensitive. And I really believe given this research, that your proposed excise tax on sweetened beverages may be one of the most effective population wide obesity prevention strategies we could enact in terms of seeing a real difference in consumption.

And then finally on the marketing level there are the promotions which are aimed at developing and reinforcing positive associations with a brand. It's important to realize that the purpose of corporate marketing is not only to sell products now, but to develop customers for life. And marketing may influence children to develop positive feelings about a branded beverage before they even get a chance to taste it and this can lead to the industry's dream achievement which is called from an industry document, "Cradle-to-grave brand loyalty." To make this happen, beverage marketers reach out to children constantly, starting when they're very young.

And we do have a brief that's been made available to the committee that goes into significant detail. I just want to highlight a few key facts.

Beverage companies lead the food and beverage industry in marketing to youth. They invest more than any other food or beverage subgroup in their marketing directed at children and adolescents. They spent almost \$500 million marketing just to children and adolescents in 2006 alone. That's half a billion dollars in one year, well over one million dollars every day, just targeting youth in one year. These companies spend far more on new media, which is digital marketing, than any other food or beverage category does and these are their fastest growing marketing techniques and I'll give some examples of those.

Where do all these marketing dollars go? Well, TV ads are still the single biggest marketing expense for the industry. However, beverage companies are buying less air time. These are three main categories, and Cadbury Schwepps has since changed its name, but this was the data for 2006 and 2007. As you can see, their TV purchases are declining and we would expect to see that trend continue.

As you've heard earlier from the industry, in recent years the beverage companies have pledged that they won't advertise their sweetened products on children's TV, by which they mean programs where kids under age twelve make up half or more of the audience. But realistically, this doesn't mean that they've given up much ground. SpongeBob SquarePants is the number one children's TV program and Coke and Pepsi won't advertise during it, but almost twice as many children watch American Idol as watch a typical episode of SpongeBob. And Coke pays \$35 million a year just to co-sponsor American Idol and get their Coke cups on the Idol judge's table. That kind of product placement is outside the traditional TV advertising budget and because it's on a show with a mixed aged audience, it doesn't violate their own narrow self-regulatory guidelines on marketing to kids. But clearly, they are reaching millions of American children this way.

At the same time as they reduce their TV ad buys, sugar sweetened beverage companies are dramatically increasing their efforts in the digital marketing realm. These are interactive promotions on websites and via cell phones and text messages, which are far cheaper than buying TV time. Again, given the unique contribution of sweetened beverages to diabetes and obesity, the industry's efforts to reach young

people through this ubiquitous digital marketing is alarming to me. And I just wanted to give a few examples.

Coca Cola's Twist/Txt/Get program, which places a reward code inside every bottle cap of Coke and Sprite and people can text the code to the company and then return the get rewards, like ring tones and screensavers and video clips, sent to their cell phone. And, of course, the company gets a database of cell phone numbers of their customers who they can and do contact several times a month. The company has said this is a critical part of their effort to, quote, "establish an omnipresent, on-the-go, everywhere relationship with teens." This is particularly problematic for youth of color as they are earlier adopters of mobile technology. They tend to use texting at twice the rates of non-youth of color, and they are definitely a particular target market campaigns.

Mountain Dew's DEWMOCRACY campaign is also online. It's encouraging its youthful fan base to become co-creators of the brand. You can log on. This is touted as a participatory form of consumer empowerment and the young fans can vote for the next new product flavor. You can see—I think, "White Out" is in the lead as of yesterday; we'll see which one ends up winning.

Taking the voting theme in a different direction is Pepsi's "Refresh Everything" campaign, which awards grants of between \$5,000 and \$250,000 to projects that will have a positive impact in their community. Winning projects are chosen by votes garnered on the website and users are encouraged to come back online and vote for their favorites up to ten times a day. Pepsi has gotten a lot of attention for their philanthropy on this campaign and I do applaud them for their donations on a lot of creative projects that might really make a difference in local communities.

I also, without taking anything away from that, want to point out what a savvy investment this campaign is for the company. This kind of "cause" marketing can help companies increase their social networking engagement with consumers. Pepsi says it doubled its Facebook fans in a single month this year as a result of this campaign. And since you have to register an email address in order to vote, it's a great way to build databases of interested consumers.

There was an article in *Advertising Age* recently pointing out that this type of social engagement effort is effectively free market research that results in more effective advertising campaigns. Pepsi gets to track the votes on different kinds of

projects and they get an idea of what is meaningful to their consumers so that they can reach out to them in more meaningful and targeted ways.

Again, Pepsi has claimed to reduce its advertising to youth, but certainly this campaign is engaging millions of young people, both in generating ideas and in voting for them, as this ad shows.

“Johny Cohen had an idea to put Plexiglas shields on old school buses to make them more aerodynamic and fuel efficient. He called the idea “Green Shields.” When he submitted it to the Pepsi Refresh project with his friends, he bagged a busload of votes and a \$25,000 Pepsi Refresh grant. Johnny cares about turning yellow gas guzzlers into big green machines. What do you care about? Find ideas near you and vote to refresheverything.com.”

So in addition to what I just said, this is also an example of the way that increasingly digital online campaigns and TV campaigns are interwoven.

And here’s another example of the “Happiness Factory” campaign from Coca Cola. I’ll show you a quick ad here. (plays ad)

Alright. So that’s like a lot of soda ads. It’s fun. It’s cute. It may be engaging to people of all ages. It doesn’t actually tell you very much about the product itself; it’s more about creating nice feelings, although, this one does seem to imply that Coke gives you some energy when you’re dragging. But what people watching this ad might not realize is that it’s really more than just a TV ad, it ties into an entire immersive online environment where you can play interactive animated games, download music from popular artists, get free stuff, and invite your friends to join in the fun. Indeed, this is the whole issue with digital marketing, unlike traditional TV ads; kids are actively engaged for many minutes, even potentially hours at a time with the brand. These experiences are intensive, interactive, and socially stimulating for youth. In the public health field we are only just beginning to explore the impact of this kind of immersive marketing, but the implications are troubling.

And of course from the industry’s perspective; every click of the mouse, every text message they get from a teen gives the companies more valuable data about their target market. I find it ironic and disturbing that these corporations are collecting vast amounts of data on our children while parents may have no awareness that this is even going on and certainly have limited ability to monitor or to control the marketing messages that their children receive. And again, remember, sweetened

beverage companies are devoting far more resources to these forms of digital marketing than any other food or beverage category does.

I had some examples of ethnic target marketing as well. I did want to address this because beverage companies do intensively drive their promotions to the populations that suffer the most from the health problems that are associated with sweetened beverage consumption. We find this a trend, just like tobacco and alcohol companies did before them, beverage companies reach out to the African American and Latino communities fashioning products to fit their tastes, creating price points favorable to these groups and saturating these communities with targeted ads for their products.

As I mentioned before, youth of color are a particularly attractive market segment for soda companies not just because they're earlier adopters of the new media technologies, but because of the way in which they're influential over the broader youth culture and where ideas and trends and products then start in a youthful urban hip-hop kind of culture and then spread out.

The companies know this, and when Coca Cola unveiled its 2007 Sprite Yard program for mobile phones, which was aimed at its mostly African American youth target audience. This program used the slang conception of a "yard," a place where everyone hangs out, to characterize a virtual space. And the Sprite brand director said, "We know that when it comes to reaching teens, mobile is the medium. This program will enable us to connect with teens by putting Sprite both in their hand and in their phone," so, again, that pervasive "we're with you everywhere" kind of campaign.

Just a couple examples of other ethnic target marketing by these companies:

This last ad that I'll show features P. Diddy and it both pokes fun of, but also reinforces, the way that hip-hop culture is used to sell products to the broader American Public. You can see how the trend here starts with Diddy and trickles down to a less hip demographic, as you'll see:

P. DIDDY: "Hey, I'm late for this award show. Can I get a ride?"

PEPSI TRUCK DRIVER: "Come on. Hop in."

UNKNOWN: "I'll tell you, Carson, the excitement here is tremendous."

CARSON DALY: "Look at that. I didn't know P. Diddy drove a Pepsi truck."

EVA LONGORIA: "Don't scratch it."

UNKNOWN: "Alright, this is how I roll." (Hip-hop dancers in the truck blaring hip-hop music)

UNKNOWN: "Hey, yo, P. Check out my new ride."

I have to say, I showed this at home last night to my husband and he was, like, "I love that ad." It's really cute, right? But again, it's not about the product, it's about increasing identification with the product; increasing engagement and those good feelings and who that calls out to in terms of a demographic.

And just lastly, I wanted to show, last fall Pepsi launched this PepsiWeInspire.com, which is an online blogging community specifically targeting African American moms. As a mom myself, this one was particularly problematic to me because they claim to be concerned about improving children's health and then they do something like this that's really aimed directly at influencing the nutritional gatekeepers of the kids who have the highest rates of diabetes and obesity in our country. And I just feel like it's really unfair. These beverage companies make so much money off of these communities and then they effectively leave a public health disaster in their wake. It's unfair to target communities in this way.

There are many other problematic beverage marketing practices and the brief that I showed before goes into them. You can also find more details on digitalads.org.

I just wanted to say this one other pervasive form of communication that the beverage industry is engaged in. As you can see, this millions of dollars spent lobbying by Coca Cola, Pepsi, and the American Beverage Association (Investigative report by the Los Angeles Times a couple of months ago.) The dollars have gone along nicely, under \$5 million a year for the last several years, and then all of a sudden just bolted up dramatically last year because soda taxes were floated as a possible part of the federal health reform legislation; they were proposed in several states. And this is an indication to me that the industry is highly concerned about these kinds of attacks. They are worried that it will have an impact on their products.

Also, I just want to take a moment while I'm here to say in the face of this kind of opposition and this kind of spending, I really want to applaud the leadership that you're showing, Senator, and appreciate the Committee considering this legislation.

Happy to answer any questions on marketing that I can. Thank you very much.

SENATOR FLOREZ: Thank you. That was very informative and very much appreciated. I wonder if we could have a copy of that for the Committee. Maybe you could just email it to us. That would be great.

Just the question that I mentioned earlier in terms of you've heard from the industry in terms of them marketing more towards less consumption versus—I don't think they're going to be marketing no consumption—do you see this as something the companies would be moving towards?

MS. WOODRUFF: I can't imagine the industry ever advocating consuming less of their products overall. I can see them putting more money into marketing for the zero-calorie alternatives, and I would support that and applaud it, for sure.

SENATOR FLOREZ: Okay. And the zero-calorie products that they could be marketing to, do you see more of these coming online; more of these types of products? Or are we just at the very beginning stage of this?

MS. WOODRUFF: It seems to me that the product lines have blossomed amazingly over the last couple of years; really, just in a couple of years. A lot of these products are brand new. And I haven't done a content analysis to look at what types of products are getting advertised where and how much. And as you asked earlier on the Spanish language media, I don't have that information but I'd be very interested to see. I'd be curious to see what happens.

And in terms of context, the largest public education campaign that we've ever had nutrition on this country is the Five-a-Day campaign, which at its height was funded by the federal government at \$10 million a year, and Coke spends \$35 million just on the American Idol sponsorship. So some funding to try to balance that, to even begin the increase health nutrition messages would be very welcome.

SENATOR FLOREZ: Thank you. Appreciate it. Great presentation.

Okay, let's go to our last panel, which is on local perspectives. We have Genoveva Islas-Hooker and Dana Richardson. Thank you for joining us. I'd love to get your testimony. And we'll, then, wrap up. Appreciate you guys sticking with us this whole time.

GENOVEVA ISLAS-HOOKER: Thank you, Chairman Florez and Committee Members. My name is Genoveva Islas-Hooker. I'm a board member of the Latino Coalition for Healthy California, as well as a board member for the California Food Policy Advocates. I live in Tulare, California. I'm involved in creating healthier food

and physical activity environments through the Central California Regional Obesity Prevention Program. We are a partnership between public health departments, community-based organizations, and grassroots community members, including youth in Fresno, Kern, Kings, Madera, Merced, San Joaquin, Stanislaus, and Tulare Counties, so essentially all eight of the Central Valley communities.

Through CCROPP, we've been able to create change and address obesity by working with community members, elected officials, and various stakeholders. Our efforts have been successful at bringing healthy foods into communities that previously did not have them through farmers' markets, community gardens, school farm stands. We've also been successful in improving physical activity environments by supporting improvements to playgrounds, parks, and advocating for better community design through general plans. More about our project can be found at our website, CCROPP.org

But essentially, what I want you to know about our program is that our approach to obesity prevention is really by changing environments and policies so that the healthy choice is the easy choice in our communities and unfortunately that isn't true at this point. To suggest that personal responsibility is the only solution to curbing the obesity epidemic is erroneous when we aren't really supporting environments that allow healthy choices to be made.

I've been asked to testify on the over consumption of sugar and sweetened beverage drinks, such as soda and its link to obesity. To that end, I want to provide you some context about the communities where I live and work.

Central California is the fruit and vegetable bowl of our state, our nation, and perhaps the world. It's everything that you can imagine a rich agricultural valley to be: it's picturesque; it's green; and it's rural; and it's many things that probably you don't necessarily imagine it to be. In this place of bounty we have hunger and we have poverty. The Central Valley is the poorest congressional districts and much of that poverty stems from the super exploitation of farm laborers.

This context is important in understanding why obesity is such a prevalent health issue in our area. Central California, as you noted, has some of the highest rates of obesity and obesity related diseases, like hypertension, heart disease, diabetes, and cancer.

So in a nutshell: Poverty; the over-availability of cheap, unhealthy foods, like sodas; the under-availability of healthy affordable foods and beverages, like water; the omnipresent targeting, promotion, and marketing of unhealthy choices, as Katie so eloquently discussed earlier; and the really limited resources and infrastructure that we have to support physical activity are predisposing us to an increased incidence of obesity.

In my region, community members are making choices about their food and beverages that are driven by their economic means and by what is available.

Sodas have become a default beverage choice because they are cheap and they are readily available. And this has been documented in the Bubbling Over report in which Central California counties have some of the highest rates of soda consumption in our state.

I'd like to share with you a photo that was taken by a Sunnyside High School youth. Stephanie Chavez is a youth from Pixlie, who has joined us. But in this photo that was taken by Jasmine from Sunnyside High School, she says that, "I see a food store and near the door there are seven soda machines and only one water machine. People are more likely to buy soda, especially because some sodas cost 25 cents, while the water is more than a dollar. This affects the community because people are more likely to consume soda. This challenge exists because by selling soda at such a low price it attracts more people than water which is more expensive."

So our youth are very conscious about the environments that we've created for them and they're asking for support in making healthier choices. And making the healthy beverage more affordable is one of the solutions, I think, that we can work towards.

There's many other opportunities to change the food and beverage and physical activity environments in order to support better health. So for example; in many Central Valley communities water is undrinkable. Community members are having to pay a bill for water that they can't drink; then they are having to pay for bottled water in order to be able to quench their thirst. So if you're struggling and you're trying to make ends meet, you're trying to stretch your dollars, you look for the most affordable options that are out there. And at this point, those most affordable options are the unhealthy options, like soda and sugar sweetened beverages. So free, safe drinking water oftentimes cannot be found, especially in many rural and incorporated

communities. And as you know, these unincorporated communities are predominantly Latino communities who are severely burdened by chronic diseases that relate to the obesity epidemic.

Another quote I'd like to highlight for you from a youth is that she's stating—this is Jessica Sanchez, a student from Orosi High—she says, "It's not fair that I don't have safe drinking water at home and that when I go to school I have to deal with the same thing."

So we need resources to fund change. We need resources to create equity in our communities. Revenues generated from the tax are desperately needed to turn the tide on the obesity epidemic. These resources could help to change the food and beverage and physical activity environments in many under-resourced communities.

- We need things like increased access to safe drinking water, the healthiest beverage choice next to breast milk, of course.
- We need support for increased access to healthy foods. It's such an irony that we live in this area and that our families do not have access to the foods that they're picking.
- We also need more physical activity environment resources in parks and recreation and improved community design.

So in closing, I think that this is a very important tax. I think that you do have a great support in the Central Valley for resources that you're helping to bring in. And I really just want to thank you for legislating for the health of our communities.

SENATOR FLOREZ: Thank you. A good presentation.

Yes. Thank you for joining us.

DANA RICHARDSON: My name is Dana Richardson. I am a resident of southern California—in the city of National City. I also represent a project called the Healthy Eating Active Communities in the city of Chula Vista, which is located just ten minutes north of the U.S./Mexico border at Tijuana. Since March 2005, our project has worked to reduce childhood obesity and promote healthy lifestyles by improving food and physical activity environments, particularly in western Chula Vista.

I'd like to offer you some local perspective. I support the legislation. I'd like to just offer you some additional solutions leading us forward in policies that address the complexity of these issues which are part of some of the earlier discussions.

Clearly, there are connections between health and place, which have become increasingly prominent as communities struggle with alarming levels of asthma, obesity, heart disease, and diabetes particularly, in low-income communities of color. Neighborhood environments are critical factors which can either support or undermine any community's ability to engage in physical activity and adopt healthy lifestyles.

A white paper by one of my colleagues was written. Mary Lee from the Policy Link organization states, "Environmental conditions, along with social and economic factors, play a much larger role in determining the health outcomes of a community. To this end, it is becoming increasingly clear that where you live affects your health and that the health of individuals depends on the neighborhoods in which he or she lives. Economics and the zip code of residence tell us much more about the key factors that shape negative or positive health behaviors and health outcomes than does their physiology.

So how do we set our youth up and our families up to be successful and to adopt healthy lifestyles? Well, the answer is: We need to create neighborhood environments that facilitate achieving these goals. And we use policies with a broad focus on health to address the underlying issues that fuel unhealthy habits and lifestyles. And provide populations with the resources and tools to avoid what's ailing them in the present day.

Many poor lifestyle decisions are made in the context of the community environments. As I stated earlier, where you live affects your health. A physical environment that supports health obviously does not contain what I currently experienced in my own community which includes a prevalence of alcohol outlets, literally on every corner; and numerous billboards and alcohol advertisements targeting Latino and black populations; a prevalence of fast food outlets; and a limited number of healthy food and beverage options (for example, the only major chain grocery store in my community is in the process of closing as we speak); and areas of our community with no sidewalks and little to no access to parks and open space. This is just my short list. There is actually a much longer list of community conditions I can share with you which contribute to childhood obesity and community-based violence.

Conversely, neighborhood environments that do support health, provide a balance of healthy and accessible food and beverage options for the community and

also match these strategies with long-range plans to incorporate ample public transportation, affordable, well-maintained housing, schools, parks, complete streets for all users, thriving businesses, new employment opportunities, and accessible, safe public play and recreation facilities, just to name a few.

Senator Florez' legislation, it's great. It's definitely a step in the right direction to strategically address poor health outcomes as it relates to the consumption of sugar sweetened beverages, particularly in low-income, vulnerable populations. Yet, from an infrastructure standpoint, the legislation is also visionary because it proposes to contribute resources toward creating healthier community environments by improving nutrition in schools and ensuring access to safe, quality parks and open spaces. The legislation literally provides a short-term immediate strategy, yet also recognizes the long-range sustainable elements that communities need to achieve health.

Therefore, I encourage the committee to continue to support this policy today. Together, we can begin to turn the tide on poor health outcomes that persist in vulnerable populations, and strive to create renewed communities of opportunity.

Thank you for the opportunity to speak.

SENATOR FLOREZ: Thank you very much. Very good testimony. Just before you both go, I'd just like to get your take on the impact, from your perspective, of the tax in low-income communities. I mean, you've heard both sides; some discussion on that. Maybe you can give me your perspective on it.

MS. ISLAS-HOOKER: So one of the points that I highlighted was the fact that there are many things that are regressive within our communities. Water in the Central Valley is an example of families having to pay both a water bill and then having to purchase bottled water because the water that comes out of their tap is unhealthy. So if this tax, which would disincentivize the soda consumption, could be used to ameliorate that. I think that it would gain, or it would actually be received with much approval from the community members that it's meant to serve the most.

MR. RICHARDSON: My perspective is that I don't think we understand a lot about what's going to happen on the end. But if we know that this tax is going to find some way to bring resources back into the community, then I think we'd be for it.

MS. ISLAS-HOOKER: I think just to stress the point again; I come from a region that traditionally has not supported increased taxation and I think a large reason for that is that the taxes haven't traditionally benefited them. They haven't

come back to those rural and unincorporated communities. They're paying taxes on their homes and they're not seeing streets improved. They're paying taxes on their cars and not seeing improvements in air quality. So I think, again, if we keep our promise and the commitment that you're showing that these resources would be directly driven back to them to improve their environments, that again, it would be important.

SENATOR FLOREZ: Right. Exactly. Thank you both. Appreciate that.

Okay, is there any public comment? Would anyone like to say anything? Okay, if not, I want to thank everyone who's traveled, particularly if you're coming from Chula Vista. I appreciate it. I know how far it is. It's in Senator Ducheny's area. And I would like to thank everyone for coming.

We would like to have a transcript of this, possibly, if staff could get on that, prior to the bill being heard. It's kind of a shorter timeframe, but we'll try. I know we had some starts. The hearing is actually not as long as the hour because we were gone—I was gone for about two hours, so it's probably not our longest hearing we've had to do. So if we could have that that would be great.

Obviously, this is a pre-hearing of the real hearing which will occur May 12th when the bill will be heard in its entirety.

I would particularly like to thank staff and all the work that went into this. I'm very intrigued by the new poll and I think, obviously, the Members will be as well.

We will adjourn the Senate Food and Ag and the Senate Health Committees. We'll see you the next time.

###

1473-S

Additional copies of this publication may be purchased for \$14 per copy (includes shipping and handling) plus current California sales tax.

Senate Publications & Flags
1020 N Street, Room B-53
Sacramento, CA 95814
(916) 651-1538

Make checks payable to SENATE RULES COMMITTEE.
Credit cards not accepted. Please include stock #1473-S when ordering.