Does Food Processing Contribute to Childhood Obesity Disparities?

Bo MacInnis and Gordon Rausser

Childhood obesity in the United States has become epidemic: the prevalence of obesity among children aged six to eleven years more than doubled between the late 1970s and 2000, rising from 6.5% to 15.3% (National Center for Health Statistics). Serious health consequences of childhood obesity include asthma (Belamarich et al.), type 2 diabetes (Pinhas-Hamiel et al.), and high blood pressure (Must and Strauss). Half of overweight children become obese adults, and adult obesity is a strong predictor for numerous major health conditions. Annually, the cost of obesity comes to $117 billion plus 300,000 deaths (U.S. Department of Health and Human Services).

Disturbing evidence has also emerged on the distributional effect of childhood obesity: there is a disproportional increase in the number and severity of overweight children. Figure 1 compares the distribution of z-score (body mass index [BMI] for age) of children aged two to ten years between 1989–91 and 1998 using the Continuing Surveys of Food Intakes by Individuals (CSFII). A child is considered at-risk overweight or overweight, respectively, if his/her BMI exceeds the 85th or 95th percentile of the age- and gender-specific growth chart. This figure reveals that within a decade the right tail is expanding and there is an increase in severely obese children.

Health care costs increase by 2.3% for every added unit of BMI (Raebel et al.). Moderately (BMI 30–35) and severely (BMI $\geq$ 35) obese individuals, respectively, incur 25% and 44% more health care costs than normal weight individuals; these additional costs are largely explained by the increased risk of coronary heart disease, hypertension, and diabetes (Quesenberry, Caan, and Jacobson).

There is a growing consensus that the rapid increase in childhood obesity is a direct result of an environment that discourages physical activities and promotes overconsumption of energy, including readily available inexpensive, energy-dense tasty foods (Hill et al.). Positive correlation is difficult to establish empirically (Drewnowski and Specter) with the exception of (1) the consumption of sugar-sweetened drinks and obesity in children (Ludwig, Peterson, and Gortmaker) and (2) higher energy density and food additives with an increased BMI in children (MacInnis).

Children's diet is increasingly shifting to energy-dense, additive-rich processed foods. Fast foods, a large component of children's diets, are nearly twice as energy-dense as foods recommended for a healthy diet (Prentice and Jebb). Foods that accounted for the greatest increase in energy intake by Americans from 1982 to 1998 were salty snacks, desserts, soft drinks, fruit drinks, hamburgers and cheeseburgers, Mexican food, and pizza. In 1977–78, these foods accounted for 18.1% of Americans' dietary energy consumption; in 1994–96, the percentage increased to 27.7%. These foods are largely composed of refined grains, added sugars, and fats (Nielsen and Popkin).

Many processed foods contain chemical compounds (e.g., flavorings, colorings, preservatives, and trans fatty acids) designed to enhance flavor, color, texture, and taste. Currently, over 3,000 food additives are registered (U.S. Food and Drug Administration). In the United States, about 10,000 new processed food products are introduced every year; almost all of them include flavor additives (U.S. Department of Agriculture 1995).

Biomedical studies show that processed foods in the form of refined starches and concentrated sugars are energy-dense (Foster-Powell, Holt, and Brand-Miller) and found to be associated with an increased risk of obesity (Pawlak, Ebbeling, and Ludwig; Liu et al.).

Bo MacInnis and Gordon Rausser are postdoctoral scholar and Robert Gordon Sproul Distinguished Professor, respectively, at the Department of Agricultural and Resource Economics, University of California at Berkeley, 207 Giannini Hall no. 3310, Berkeley, California 94720-3310.

Authors thank Wade Brorsen for his editorial comments.

This article was presented in a principal paper session at the AAEA annual meeting (Providence, Rhode Island, July 2005). The articles in these sessions are not subjected to the journal's standard refereeing process.

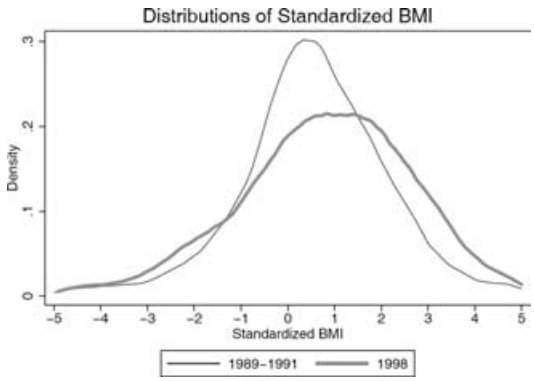

Figure 1. The distributions of age- and gender-adjusted *z*-scores of body mass index (BMI) between 1989–91 and 1994–96, 1998

Heavyweight children may be particularly vulnerable to the impact of processed foods on their weight (Liu and Manson; Hu, van Dam, and Liu).

The scale of preparation and processing brings cost savings to consumers. Food has become less expensive, and processed foods have become increasingly less expensive than nonprocessed foods (U.S. Department of Agriculture 2002). Additional studies show that high-energy density means low-energy cost (price to consumers). For example, a dollar's worth of potato chips provides 1,200 calories of energy, compared to 250 calories from a dollar's worth of fresh carrots. Fats and oils, sugar, refined grains, and processed potatoes are among the least costly sources of energy. The differential in costs between sugar and strawberries is in the order of several thousand percent (Drewnowski and Specter).

Empirical Specification

Given this background, the logical question is, "what do dietary concentrations of processed foods, in the form of high-energy density and food additives, contribute to the incidence and the distributional effect of childhood obesity?" Accordingly, the purpose of our analysis is to isolate the impact of processed foods on obesity risk by controlling for confounders arising from individual heterogeneity and dietary influence.

Heterogeneity in the preferences for processed foods may arise from genetic differences and family background. We represent individual heterogeneity in four elements: (i) observed child characteristics, C, such as age and gender, (ii) observed family characteristics, F, such as income and parental education, (iii) unobservable family-specific characteristics, a^F, such as discount factor, parents' food preferences, and common genetic predisposition to weight gaining among siblings, and (iv) unobservable child-specific characteristics, a^C, such as variance among siblings regarding genetic predisposition to processed foods.

Recent advances in the biomedical literature on obesity suggest that weight status may depend on characteristics of food consumed in addition to total energy and dietary fat intakes. We also control for energy expenditure using hours of watching television as a negative proxy for exercise, and child's health behavior using vitamin supplement intake as a proxy. We denote all these child-specific dietary variables as D.

We investigate whether processed foods impact a child's chance of being at or exceeding a particular percentile of the BMI distribution. The 85th percentile is particularly important, since it is the cutoff point where a child is considered at-risk overweight if his/her BMI exceeds the 85th percentile of his/her age- and gender-adjusted growth chart.

Let W^θ denote the cutoff point for the θth percentile of the age- and gender-specific BMI distribution and W denote a child's BMI; the probability of a child's BMI exceeding the θth percentile is given by the following logistic specification:

$$(1)\qquad \Pr(W > W^\theta \mid a^F, R, ED, X) = \Lambda\left(a^F + \beta_{1\theta} R + \beta_{2\vartheta} ED + X'\pi_\theta\right)$$

where $X'\pi_\theta = C'\pi_{C\theta} + F'\pi_{F\theta} + \pi_{E\theta}E + D'\pi_{D\theta}$, Λ is the cumulative density of the logistic distribution. It will be presumed that the child-specific factor is uncorrelated with all other covariates.

The identification of parameters $\beta_{1\theta}$ and $\beta_{2\theta}$ is based on within-sibling variations in dietary characteristics, namely, energy density and amount of food additives. Sufficient variation exists in energy density and food additives within siblings, and variations are exogenous (MacInnis).

We estimate (1) using a fixed-effect estimator to eliminate all observable or unobservable cofounders common to children in the same family. Comparing siblings controls for genetic components common to siblings, as well as effects of income, price, and parental time constraints. The child-specific heterogeneity is accounted for to the extent that the sources of the heterogeneity originate from family

characteristics. Please note that within-family heterogeneity and potential interaction between family-specific cofounders with child-specific characteristics are not controlled for, which may be a source of some bias.

Data

We use the CSFII 1994–96, 1998 to investigate the incidence and distribution effect of childhood obesity. CSFII are national representative cross-sectional surveys conducted by the U.S. Department of Agriculture. CSFII has a large set of family and child characteristics that are important determinants of weight, plus twenty-four-hour dietary recalls of every food item consumed for two nonconsecutive days.

To characterize the increasing concentration of processed foods in children's diet, we use residuals and energy density. We calculate food residuals (in grams) as unlabeled and uncounted substances in food that are not proteins, fats, carbohydrates, or water in all food items in a child's twenty-four-hour diet. We calculate each food's residual by subtracting the grams of fat, protein, carbohydrates, and water from the total grams of food; the total residuals are then the sum of all the food items in the child's twenty-four-hour consumption.

We construct two forms of dietary energy density: overall and weighted energy density, both measured in kilojoules per gram (kJ/g). Overall energy density is calculated as the ratio of total energy intake in calories to total weight of food in grams; weighted energy density is calculated as the weighted average of energy density of four foods (hereafter, four principal foods) from which a child obtains the most calories, using the caloric contribution of food items as the weights. The weighted energy density represents the degree of the concentration of calories from energy-dense foods.

Figure 2 compares the distributions of amount of food residuals and weighted energy density of children aged two to ten years between 1989–91 and 1998. Within less than a decade, the average amount of residuals in children's food more than doubled from 6.42 to 14.35 g, an increase of nearly 8 g. The weighted energy density in the same period increased 60%, from 0.83 to 1.33, an increase of half a unit, while the overall energy density remained the same. The higher weighted energy density indicates that children increasingly rely on energy-dense foods.

Table 1 contains summary statistics for 4,087 usable observations of children under age of ten years. The sampled children have an average BMI of 18.08 kg/m^2 and are on average at the 78th percentile of the control population, which shows that American children are becoming heavier. On average, children's

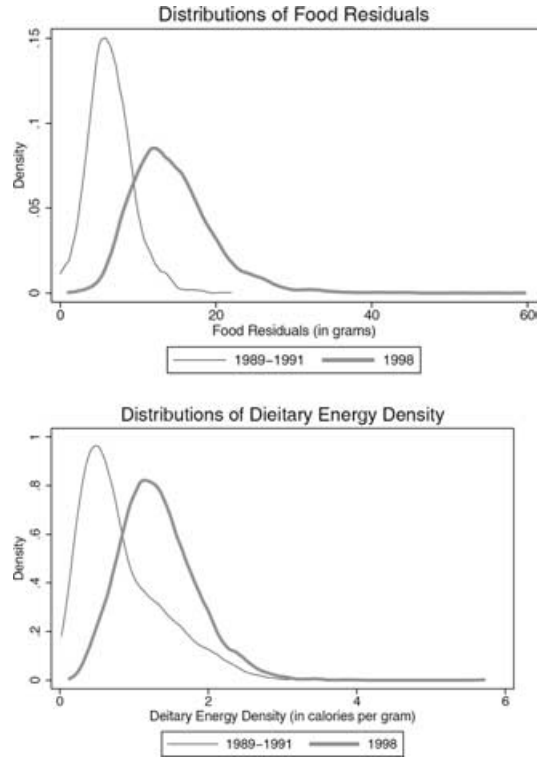

Figure 2. The distributions of children's dietary energy density and the amount of residuals in their food intake between 1989–91 and 1998

Table 1. Sample Means of Selected Dietary and Health-Related Behavioral Variables

Variables	Sample Mean	Standard Error
BMI (kg/m^2)	18.08	(0.07)
z-Score BMI	0.79	(0.03)
Total energy intake ('000 kJ)	1.68	(9.15)
Total fat intake (g)	60.81	(0.43)
Total food weight (kg)	1.49	(8.29)
Overall energy density (kJ/g)	1.16	(0.0045)
Weighted energy density (kJ/g)	1.36	(0.008)
Total residuals (g)	14.23	(0.08)
Residual concentration 10^{-3}	9.79	(0.04)
TV hours	2.54	(0.03)
Infrequency of taking vitamins	2.07	(0.01)
Age	4.20	(0.03)

Notes: These are unweighted sample averages. The variable *infrequency taking vitamins* is a categorical variable where 1 = daily, 2 = often but not daily, and 3 = never. Sample size is 4,087.

overall and weighted dietary energy densities are 1.16 and 1.36 kJ/g, and their intake of residuals is approximately 14 g with average residual concentration 9.79 (grams of residuals in 1,000 g of food). On average, children consume 1,682 calories and 1,490 g of food by weight, including about 60 g of dietary fat; they watched television 2.54 hours/day.

Results and Discussion

Our estimation focuses on overweight or at-risk overweight children aged two to ten years with one or more normal-weight siblings in the same age group. Table 2 presents fixed-effect logistic estimates of (1). These results demonstrate that both residuals and energy density significantly increase a child's probability of being overweight or at-risk overweight. An additional 10 g of residuals increases the probability of being overweight by 29%. A one-unit increase in energy density increases the probability of being overweight by 13.8%.

We examine the distributional impacts of processed foods on children's weight using several other indicators to evaluate the impact of processed foods on the chances of children's BMI exceeding a particular high percentile of the control population. For example, a dependent variable *Is95* is an indicator that is set to 1 if the child's BMI is equal to or greater than the 95th percentile of the growth chart, and 0 otherwise. The impact of energy density is most pronounced in at-risk overweight children (i.e., at the 85th and 87th percentiles); the marginal effect is 16.9% and 13.8%, respectively. Energy density also has a significant impact for the severely obese; at the 97th percentile, the marginal effect is 5.9%. The impact of residuals is also concentrated in the at-risk overweight children; the marginal effect is 3.4% for the 87th and 90th percentiles, 2.9% for the 85th percentile, and 2.3% for the 80th and 77th percentiles.

There are several limitations to the interpretation of our findings. Our study is observational and does not prove causality. The intake of processed foods may reflect unobserved child-specific factors that affect the obesity incidence. Variables used in the analysis originate from parents' recall and may contain measurement or reporting errors. Finally, although we use siblings as controls and account for several important factors, the possibility of confounding is especially strong if the child-specific genetic makeup and a child's physical activities were the primary determinants of obesity incidence.

Table 2. Estimates of Fixed-Effect Specification (1) and Distributional Effect Analysis

Variables	*Is99*	*Is97*	*Is95*	*Is90*	*Is87*	*Is85*	*Is80*	*Is77*
Energy density	0.003	0.059**	0.098	0.121	0.169**	0.138*	0.087	0.077
	(0.532)	(0.389)	(0.365)	(0.324)	(0.307)	(0.310)	(0.291)	(0.285)
Residuals	0.0004	0.003	0.008	0.034***	0.034***	0.029**	0.023*	0.023*
	(0.081)	(0.060)	(0.060)	(0.055)	(0.053)	(0.054)	(0.052)	(0.053)
Total fat	0.000	0.001	0.004	0.003	0.002	0.002	−0.0001	0.005
	(0.020)	(0.014)	(0.014)	(0.012)	(0.011)	(0.012)	(0.011)	(0.011)
Total energy	−0.007	−0.016	0.244	−0.427**	−0.381**	−0.279	−0.122	−0.206
	(1.318)	(0.780)	(0.859)	(0.795)	(0.735)	(0.745)	(0.694)	(0.681)
TV hours	−0.001	−0.0005	0.011	0.023	0.043**	0.028	0.043**	0.021
	(0.136)	(0.101)	(0.095)	(0.082)	(0.078)	(0.080)	(0.076)	(0.076)
Diet discipline	0.020***	−0.041	0.287	0.310	0.418**	0.401**	0.450**	0.471*
	(1.322)	(1.424)	(1.255)	(1.166)	(1.226)	(1.197)	(1.194)	(1.194)
Vitamins infrequency	−0.005**	−0.026	0.050	0.111	0.090	0.168**	0.090	0.072
	(0.456)	(0.371)	(0.333)	(0.318)	(0.289)	(0.321)	(0.290)	(0.288)
Age	−0.005***	−0.078***	−0.170***	−0.218***	−0.220***	−0.271***	−0.205***	−0.183***
	(0.299)	(0.232)	(0.215)	(0.195)	(0.191)	(0.194)	(0.184)	(0.180)
Age × Age	0.0002	0.004***	0.009***	0.012***	0.012***	0.016***	0.012***	−0.011***
	(0.027)	(0.020)	(0.018)	(0.016)	(0.016)	(0.016)	(0.015)	(0.015)
Female	−0.001	−0.027	−0.095**	−0.056	−0.051	−0.003	−0.023	−0.028
	(0.284)	(0.229)	(0.223)	(0.207)	(0.200)	(0.202)	(0.189)	(0.188)
Sample size	398	491	528	605	650	650	663	646

Notes: ***significant at 1%; **significant at 5%; *significant at 10%. Presented are derivative estimates calculated at the sample averages. Standard errors are given in parentheses. Dependent variable, for example, *Is90*, is set to 1 if a child's BMI exceeds the 90th percentile of the control populations. Additional regressor is total food weight in grams.

Conclusion

Many have hypothesized that the increased prevalence of childhood obesity is a result of environmental factors that promote the overconsumption of low-cost and good-tasting energy-dense foods. We have empirically investigated this hypothesis and quantified the contribution to the childhood obesity epidemic of such foods. Our results strongly suggest that dietary concentration of energy density and food additives can be an important contributory factor to the incidence of being at-risk overweight—above and beyond the effects of total energy and dietary fats intakes themselves; furthermore, it can partly explain the disparity in childhood obesity.

References

Belamarich, P.F., E. Luder, M. Kattan, H. Mitchell, S. Islam, H. Lynn, and E.F. Crain. "Do Obese Inner-City Children with Asthma Have More Symptoms Than Nonobese Children with Asthma?" *Pediatrics* 106(2000):1436–41.

Drewnowski, A., and S. Specter. "Poverty and Obesity: The Role of Energy Density and Energy Costs." *American Journal of Clinical Nutrition* 79(2004):6–16.

Foster-Powell, K., S. Holt, and J. Brand-Miller. "International Tables of Glycemic Index and Gylcemic Load Values: 2002." *American Journal of Clinical Nutrition* 62(2002):5–56.

Hill, J.O., H.R. Wyatt, G.W. Reed, and J.C. Peters. "Obesity and the Environment: Where Do We Go from Here?" *Science* 299(2003):853–55.

Hu, B., M. van Dam, and S. Liu. "Diet and Risk of Type II Diabetes: The Role of Types of Fat and Carbohydrate." *Diabetologia* 44(2001):805–17.

Liu, S., and J. Manson. "Dietary Carbohydrates, Physical Inactivity, Obesity, and the 'Metabolic Syndrome' as Predictors of Coronary Heart Disease." *Current Opinion in Lipidology* 12(2001):395–404.

Liu, S., W.C. Willett, J.E. Manson, F.B. Hu, B. Rosner, and G. Colditz. "Relation between Changes in Intakes of Dietary Fiber and Grain Products and Changes in Weight and Development of Obesity among Middle-Aged Women." *American Journal of Clinical Nutrition* 78(2003):920–27.

Ludwig, D.S., K.E. Peterson, and S.L. Gortmaker. "Relation between Consumption of Sugar-Sweetened Drinks and Childhood Obesity: A Prospective, Observational Analysis." *Lancet* 357(2001):505–8.

MacInnis, B.Y. "The Costs and Health Consequences of Food." PhD dissertation, University of California, Berkeley, 2004.

Must, A., and R.S. Strauss. "Risk and Consequences of Childhood and Adolescent Obesity." *International Journal of Obesity* 23(1999):S2–S11.

National Center for Health Statistics (NCHS) and Centers for Disease Control and Prevention (CDC). *Health, United States, Overweight Children and Adolescents 6–19 Years of Age, According to Sex, Age, Race and Hispanic Origin: United States, Selected Years 1963–65 through 1999–2000*. Hyattsville, MD, 2002, Table 71.

Nielsen, S., and B. Popkin. "Patterns and Trends in Food Portion Sizes, 1977–1998." *Journal of the American Medical Association* 289(2003):450–53.

Pawlak, D., C. Ebbeling, and D. Ludwig. "Should Obese Patients Be Counseled to Follow a Low-Glycemic Index Diet? Yes." *Obesity Review* 3(2002):235–43.

Pinhas-Hamiel, O., L.M. Dolan, D.S. Standiford, P. Khoury, S.R. Daniels, and P. Zeitler. "Increasing Incidence on Noninsulin-Dependent Diabetes Mellitus in Children and Adolescents." *Journal of Pediatrics* 128(1996):608–15.

Prentice, M., and A. Jebb. "Fast Foods, Energy Density and Obesity: A Possible Mechanistic Link." *Obesity Review* 4(2003):187–94.

Quesenberry, C.P., B. Caan, and A. Jacobson. "Obesity, Health Services Use, and Health Care Costs Among Members of a Health Maintenance Organization." *Archives of Internal Medicine* 158(1998):466–72.

Raebel, M.A., D.C. Malone, D.A. Conner, S. Xu, J.A. Porter, and F.A. Lanty. "Health Services Use and Health Care Costs of Obese and Nonobese Individuals." *Archives of Internal Medicine* 164(2004):2135–40.

U.S. Department of Agriculture. *USDA Food Composition Data, Selected Foods Containing Trans Fatty Acids*. Available at http://www.nal.usda.gov/fnic/foodcomp/Data/index.html, 1995.

——. *The U.S. Food Marketing System 2002: Competition, Coordination, and Technological Innovations*. Available at http://www.ers.usda.gov/publications/aer811/, 2002.