Childhood Obesity:
Is There Something Amiss with Food Processing?

Bo MacInnis* and Gordon Rausser
Dept. of Agricultural and Resource Economics
University of California
Berkeley, CA 94720-3310

Abstract

In this paper, we estimate the impact of processed foods—characterized by energy density and the amount of unidentified residuals in the food including food additives —on children's weight. We find that there is a direct correlation between the increase of energy density and residuals in children's dietary intake and their weight and body mass index (BMI) as well as their chance of becoming overweight. We also find that the impact of processed foods is asymmetric across the distribution of BMI; overweight children are more susceptible to the impact of processed foods than normal-weight children. Our estimates are robust to unobservable family characteristics, potential omitted variables, simultaneity, and measurement errors. Our findings suggest that the changed characteristics of processed foods are partly responsible for the increase in childhood obesity. Policies that reduce the amount of additives and energy density may be considered.

* Corresponding author: macinnis@are.berkeley.edu. I would like to thank Jeff Perloff and Guido Imbens for their suggestions.

Introduction

Childhood obesity in the United States has become an epidemic in recent decades. It has been suggested that environmental causes rather than genetic factors may be largely responsible. The commonly held beliefs as to the causes of obesity—overeating and inactivity—may or may not fully explain the current epidemic. There is evidence of immaterial change in children's total energy intakes and an overall decrease in their dietary fat intakes. There is also evidence that children are increasingly shifting to energy sources with refined carbohydrates and added sugars and fats. Furthermore, evidence indicates that the body's natural weight-control mechanisms are not functioning properly in the obese. What has been largely overlooked is that food itself has changed significantly during the last few decades: food is now intensely processed. Processed foods are physically refined and often made with synthetic and chemical additives such as colorings, flavorings, preservatives, and fat substitutes. Concomitantly, Americans spend 90 percent of their food expenditures on processed foods, creating a dramatic rise in children's consumption of processed foods.

What is it about processed foods that might contribute to the obesity epidemic? The human body produces energy from food, and the amount and the type of food eaten determines the efficiency of bodily energy production. Processed foods such as refined sugar and white flour raise blood sugar levels quickly and produce high levels of glucose in the blood, which in turn stimulates the pancreas to release high levels of insulin to transport glucose to the cells for immediate energy use. When these immediate energy needs are met, the liver converts excess glucose into glycogen, which is stored in the liver and muscles for later energy use. Once this need is met, the liver converts any remaining glucose into fat.

When insulin levels are frequently or chronically elevated, there are two separate compounding effects. The ability of the insulin to transport glucose to cells is compromised and the insulin receptors on the cells' surfaces are blocked, preventing glucose from reaching the cells effectively. The medical term for this condition is insulin resistance. Studies show that insulin resistance is more prevalent in obese people than normal-weight people. As previously noted, this lack of glucose at the cellular level in turn makes the individual crave sweets and

carbohydrates, which compounds the problem into a vicious cycle: insulin resistance leads to obesity and obesity increases insulin resistance.

Concomitant with the biomedical vicious cycle is the economics of food processing. The response of the food industry to the increased demand for processed foods has been to escalate the processing, producing food with higher energy density and more added chemicals, which further shift children's preferences towards processed foods. Energy-dense foods provide great instantaneous gratification and are thus more satisfying to eat than less dense foods. Colorings, flavorings, and fat substitutes are added to enhance visual, olfactory, and taste appeal. Production economics is another motivation: chemicals are massively produced with near zero marginal cost, and energy-dense foods are inexpensive to package and transport. The per-calorie price of processed foods declines, casting a positive effect on demand for calories from processed foods.

It has been widely suggested in the literature (Hill and Peters 1998) and there is mounting evidence from experimental studies that there is a positive association between dietary energy density and overweight. But this relation has been difficult to establish empirically, and no studies with community-based data have shown such a connection, possibly due to cofounders (Drewnowski and Specter 2004). We obtain strong empirical results on the relationship between energy density and overweight, and we attribute our findings to two contributions to the literature: we fully control for all observable and unobservable family effects, and our measure of energy density takes behavioral choice into account.

Using the Continuing Surveys of Food Intakes by Individuals Intakes (CSFII)[1], we test the following hypothesis: altering the characteristics of food by increasing energy density and adding chemicals generates a negative externality on childhood obesity. In particular, we ask three questions. Does processed foods increase a child's weight? Does processed foods increase a child's chance of being overweight? Are overweight children more affected by processed foods than normal-weight children?

[1] The description of the surveys is available at: http://www.pop.psu.edu/data-archive/daman/csfii.htm.

We present the distributions of children's body mass index using the two most recent waves of CSFII (1989–91 and 1994–96, 98) in Figure 1. Like Cutler et al. (2003), we find the overall distribution of U.S. children's BMI, adjusted with age and gender, shifts to the right; what is more pronounced is that the right tail becomes thicker. Just within one decade, the right tail from the point of 1.5 has expanded uniformly. The increases in mean and the expansions of the right tails are more pronounced in children of lower permanent incomes.

We characterize processed foods in two metrics: residuals and energy density. We construct a variable to capture the residuals in the food, defined as unlabeled and uncounted substances in the food that are not proteins, fats, carbohydrates, or water. The residuals in all food items in a child's daily intake are summarized. We characterize a child's dietary energy density by averaging calories per gram of food items weighted by their caloric contribution to the total energy intakes.

Figure 2 compares the distributions of amount of food residuals (top panel) and dietary energy density (bottom panel) of children under age 10 between 1989–91 and 1998. Within less than a decade, the average amount of residuals in children's food more than doubled, with an increase of nearly 8 grams: from 6.42 grams in 1989–91 to 14.35 grams in 1998. The dietary energy density in the same period increased 60 percent, with an increase of half a unit: from 0.83 kJ/g[2] in 1989–91 to 1.33 kJ/g in 1998.

Our fixed-effect estimation with the Box-Cox transformation indicates that higher energy density and residuals in food increase a child's BMI. The impacts are heterogeneous, affecting younger children more than older children and overweight children and more than normal-weight children. The social significance of our estimates can be illustrated with a four-year-old boy with 75th percentile BMI of approximately 16.5 kg/m^2. An additional 10 grams of residuals would increase his BMI by 0.8 kg/m^2, and a one-unit increase in dietary energy density would increase his BMI by 0.7 kg/m^2. The difference between the 75th and 85th percentiles when he is considered at-risk overweight is 0.5 kg/m^2, and the difference between the 75th and 95th percentiles when he is considered overweight is 1.5 kg/m^2.

[2] It is calories per gram of food in weight.

Our fixed-effect logistic estimation indicates that higher energy density and residuals in food increase a child's chance of becoming overweight. We find that an additional 10 grams of residuals would increase a child's chance of becoming overweight[3] by 29 percent, and that a one-unit increase in dietary energy density would increase a child's chance of becoming overweight by 14 percent. Applying these estimates to the increases in food residuals and dietary energy density between 1989–91 and 1998, our model would explain the 34.4 percent increase in the prevalence of childhood obesity.

Our quantile regression results provide additional evidence that overweight children are more susceptible to the impacts of processed foods than normal-weight children. For example, an additional gram of residual would increase a child's BMI by 0.157, 0.105, or 0.079 if his/her BMI is at the 95th, 85th, or 75th percentiles, respectively; a one-unit increase in energy density would increase a child's BMI by 1.52, 0.725, or 0.405 if his/her BMI is at the 95th, 85th, or 75th percentiles, respectively.

Our estimates are robust to unobservable family characteristics, potential omitted variables, simultaneity, and measurement errors in the characteristics of processed foods (energy density and residuals). Our estimates account for the child's dietary behavior, including total energy intake and fat intake; hours of watching television; vitamin supplement intake (if any); any diet discipline; age; and gender.

The rest of the paper follows. Section 1 presents a conceptual framework that integrates the economics and biomedical evidence. We develop an economic model in Section 2 and econometric specifications in Section 3. Data description and estimation results are in Sections 4 and 5. Section 6 presents robustness analysis and policy implications. Section 7 concludes.

[3] According to Center for Disease Control and Prevention, a child is considered overweight if his/her body mass index (BMI, defined as the ratio of weight in kilograms to the square of height in meters) is between the 85 and 95 percentiles of the age-sex-adjusted growth chart, and obese if in the 95 percentile or higher. In this paper, we use overweight and obese interchangeably to refer to children whose BMI exceeds the 85 percentile of the distribution.

1. A Conceptual Framework

In this section, we develop a conceptual framework, depicted in Figure 2, that integrates the economic and biomedical factors through which processed foods may contribute to childhood obesity.

1.1. Economics Studies on Obesity

Recent economic studies on obesity provide a context within which we develop our conceptual framework. Studies on adult obesity suggest that technological progress, and agricultural innovation in particular, is the primary explanation of the increase in adult obesity. Lakdawalla and Philipson (2002) hypothesize that technological change has raised the cost of physical activities and lowered the cost of calories. They find approximately 40 percent of obesity may be due to expansion of the food supply, while 60 percent is due to demand factors such as a fall in physical activities. Cutler, Glaeser, and Shapiro (2003) propose that the switch from individual to mass preparation of food drives adults to over-consume because food is less expensive to purchase and take less time to consume. Drewnowski and Specter (2004) suggest that the concentration of adult obesity among the poor and less educated is a result of their excessive consumption of energy-dense foods, which are less expensive and considered more palatable than low-energy dilute foods.

Studies on childhood obesity are few, the most notable being Anderson, Butcher, and Levine (2002), who test for a causal relationship between maternal employment and childhood obesity. They find that a child is more likely to be overweight if the mother worked most of the time. The effect is more evident for educated Caucasian mothers with high incomes. The effect is relatively small: specifically, increased work hours among mothers led to a 0.4 to 0.7 percent increase in overweight children, which represents just a small share of the overall increase.

1.2. The Characteristics of Processed Foods

In contrast to non-processed foods, processed foods can best be characterized by energy density, as expressed in calories per gram of food weight (kJ/g); amount of chemicals added, such as

flavorings, colorings, and trans fatty acids; and glycemic index or load, that measures the effect on blood sugar and insulin production.

Processed foods tend to be starchy products with little water content. For example, potato chips (23 kJ/g), chocolate (22 kJ/g), and doughnuts (18 kJ/g) are all energy-dense foods, while non-processed foods such as raw vegetables and fruits (0.4–2.0 kJ/g) are low in energy density (Drewnowski and Specter 2004). Energy-dense foods tend to be high in both sugar and fat.

Processed foods contain chemical compounds that are designed and manufactured to enhance the flavor, color, texture, and taste of the food; they are widely used in prepared foods, beverages, dairy food, and confectionery products (USDA 1995). The American flavor industry has annual revenue of about $1.4 billion with estimated global sales of $11 billion (International Flavorings and Fragrances 2003). There are more than 3,000 food additives registered by the FDA[4]. About 10,000 new processed foods products are introduced every year in the United States, and almost all of them use flavor additives.

Finally, processed foods with their refined starches and concentrated sugars are high in glycemic index (Foster-Powell et al. 2002). The starch structure, fiber content, food processing, and physical structure of a food determine its glycemic index[5] (Morris and Zemel 1999). Processed foods are additionally often high in glycemic load, which is the glycemic index multiplied by the amount of food consumed (Mensoda 2003).

1.3. The Economics of Food Processing

We hypothesize that food is increasingly processed to reduce production and distribution costs. We use artificial flavorings as an illustrative example. Flavorings present an attractive alternative input to food manufacturing, because flavor is a small percentage of the volume and cost of the end product sold to the consumer, but a major factor in consumer selection and acceptance of the product (International Flavors and Fragrances 2003). Soft drinks contain a larger proportion of

[4] FDA maintains a database of these additives in an inventory often referred to as "*Everything" Added to Food in the United States* (EAFUS). http://www.cfsan.fda.gov/~dms/eafus.html.

[5] For example, apples, a lower glycemic index food, have a glycemic index of 38, while higher glycemic index foods include popcorn, at 72, and cornflakes, at 81.

flavor additives than most products[6]; the flavor in a 12-ounce can of Coke costs only half a cent (Schlosser 2001). International Flavors and Fragrances, the largest flavoring company in the world, produces 21,000 compounds for food flavorings annually, and reported that "with their Cocoa Extender system, their customers were able to replace up to 50 percent of the natural cocoa in food, confectionery, and beverage formulations, and reduce their costs by about 20 percent without altering the products' mouthfeel, color or taste" (IFF 2003).

The scale of preparation and processing brings the cost-saving benefits to consumers. Figure 3 shows that prices of food, relative to prices of non-food, have steadily declined over the past few decades. That would partially explain an overall increase in food consumption. Furthermore, processed foods have become increasingly less expensive than non-processed foods; and the price differentials would be more pronounced if we adjust for energy content because processed foods are becoming denser in energy content. Published studies show that high energy density means low energy cost[7] (Drewnowski and Popkin 1997, Drewnowski 2003, and Drewnowski and Specter 2004). This may explain why processed foods have increasingly become the major energy sources of American children.

1.3. Increased Consumption of Processed Foods

Consumption of processed foods increased in direct response to their lowered prices. Food identified as accounting for the greatest increase in energy intake by Americans comprised salty snacks, desserts, soft drinks, fruit drinks, hamburgers and cheeseburgers, Mexican food, and pizza (Nielsen and Popkin 2003). These foods combined accounted for 18.1 percent of the dietary energy consumed by Americans in 1977 and 27.7 percent in 1996, and many such foods are composed of refined grains, added sugars and fats.

During this same period, children's consumption of grain products increased 50 percent for girls and 74 percent for boys; consumption of fruit and soft drinks increased 120 percent for girls and

[6] Food flavorings are present in parts per billion, because our noses can detect aromas present in quantities of a few parts per trillion.

[7] This is the price that consumers pay, and represents the cost of energy to consumers. The energy cost of cookies or potato chips was 20 cents/MJ (or 1200 kcal/$), compared to fresh carrots at 95 cents/MJ (or 250 kcal/$). Fats and oils, sugar, refined grains, and processed potatoes were among the least costly sources of energy. The differential in costs between sugar and strawberries was in the order of several thousand percent (Drewnowski and Specter 2004).

149 percent for boys. The increases are higher for adolescents (defined as ages 12 to 18): 91 percent for girls and 109 percent for boys in grain consumption, and 228 percent for girls and 527 percent for boys in fruit and soft drinks consumption (USDA 2002, 2003).

1.4. Biomedical Evidence on the Link between Obesity and Processed Foods

Processed foods in the form of refined starches and concentrated sugars are high-glycemic-index foods. High-glycemic-index foods play an important role in the metabolic rate of carbohydrates and, consequently, may significantly affect the risk of cardiovascular disease, diabetes, and obesity. Liu et al. (2003) find a positive association between weight gain and the intake of refined-grain foods in a prospective cohort study with 74,091 female U.S. nurses. It has been suggested that a diet that is high in saturated fat, sugar, and refined carbohydrates contributes to increased plasma lipids and is partly responsible for the growing obesity epidemic (Keller and Lemberg 2003 and Roberts et al. 2002).

High-glycemic-index foods contribute to insulin resistance and promote overeating, completing the vicious cycle of insulin resistance and obesity. Rapidly digested and transformed into glucose, high-glycemic-index foods cause a large increase in blood glucose and insulin (Pawlak, Ebbeling, and Ludwig 2002). They also induce a sequence of hormonal changes that limit the availability of metabolic fuels, increase hunger, and promote overeating (Ludwig 2003 and Roberts 2000). Animal studies show that the risk for excessive food consumption and obesity in mice is more likely with a diet containing refined sugars than a diet with non-refined sugars (Potezny et al. 1986). A high intake of refined carbohydrates may increase the risk of coronary heart disease by aggravating glucose intolerance and dyslipidemia, and that obese and insulin-resistant individuals are particularly prone to the adverse effects of a high dietary glycemic load (Liu and Manson 2001). Major dietary patterns are found to be predictors of plasma biomarkers of obesity risk (Fung et al. 2001). Added sugars, which are a common ingredient in processed or prepared foods and a major additive in soft drinks, are associated with obesity (Johnson and Frary 2000, Bray et al. 2004).

Food additives are regulated with the FDA[8], and neurobehavioral and/or chronic health effects are not required. Processed foods often contain added fats, much of which is in the form of trans fatty acids[9]. The Institute of Medicine reported, "Americans are eating food contaminated with dangerous levels of manufactured trans fatty acids, an ingredient that has no safe level for human consumption, and the upper intake level is zero." Chemically reconfigured, artificial trans fatty acids harden cell walls, take cell space, and impede flexibility and mobility. Trans fatty acids can also increase insulin resistance (Bhathena 2000, and Bray et al 2002), and that the effect is more pronounced in overweight individuals than normal-weight individuals (Hu van Dam and Liu 2000). Risk of cardiovascular diseases is strongly and clearly correlated to the consumption of trans fatty acids (Liu and Willett, 2003). The FDA estimates that by eliminating just margarine and three percent from baked items intake, more than 17,000 heart attacks and more than 5,000 deaths could be prevented in the U.S. every year.

It is well established in rats that neonatal treatment of monosodium glutamate (MSG) induces obesity and changes the intestinal functions and other natural weight regulatory mechanisms, including insulin resistance (Morris et al. 1998; Mozes, Lenhardt, and Martinkova 2000; Dolnikoff et al. 2001; and Racek, Lenhardt, and Mozes 2001). This kind of chemically induced obesity may persist across the life span (Macho et al. 2000).

2. An Economic Model

A household production model, where parents maximize an intertemporal utility from consuming time-intensive and goods-intensive commodities given their budget and time constraints, represents the core economic framework. Parents optimally choose the share of income on goods-intensive commodities, and food expenditure in particular. Parents allocate between processed foods and non-processed foods, taking into account their differing characteristics. The conceptual framework suggests that, compared to non-processed foods, processed foods are (a) less expensive in purchase prices on a per energy basis, (b) less expensive in time costs of

[8] The FDA regulates food additives through a system generally recognized as safe (see Burdock and Carabin 2004 for a review). For food additive toxicity testing, the FDA requires the manufactures to fulfill an extensive set of protocols including teratological evaluations in rodents.

[9] Trans fatty acids are present in 95 percent of cookies, 75 percent of snacks and chips, 70 percent of cake mixes, and almost half of cereals.

consumption since it requires less time to prepare, eat, and clean up, and (c) more palatable and satisfying to consume.

2.1. The model

Our theoretical model is extended from Lakdawalla and Philips (2003). A child's current period utility depends on processed foods consumption, X, non-processed foods consumption, Z, non-food consumption, K, and her current weight, W. Let $U(X, Z, K, W)$ be the instantaneous utility, where U increases with consumption of all kinds, but is not monotonic in weight. We assume that an individual has an ideal weight, W_0, and that deviations from the ideal weight are undesirable. Suppose processed and non-processed foods are substitutes such that $U_{XZ} < 0$; food (processed or non-processed) and other consumption are not substitutes, $U_{XK} \geq 0$ and $U_{ZK} \geq 0$.

We suppose that weight gain is a dynamic process where a child becomes overweight when she takes in more calories than her body can metabolize. Weight is a state variable that increases by consuming food and decreases by exercising. Weight responds to the characteristics of food, in addition to the quantities of the food. Changes in weight also depends on child-specific characteristics, denoted as a. Let γ denote the vector of characteristics of processed foods relative to non-processed foods, and let E denote energy expenditure such as the strenuousness and frequency of exercises. The motion equation for weight can be written as follows:

$$W' = W + g(X, Z, E; \gamma, a) \quad (1)$$

Where g is a continuous and concave function that increases with X and Z and decreases with E; W' denotes weight in the next period. If processed foods, X, is more energy-dense than non-processed foods, Z, one would expect the partial effects $g_X > g_Z$; which states that X is more weight-promoting than Z. Let V denote the value function for the individual and the problem becomes:

$$V(W) = \max_{X, Z, K, W'} \{U(X, Z, K, W) + \beta V(W')\} \quad (2)$$

Subject to the income constraint $p_X X + p_Z Z + K \leq Y$ and the weight motion equation (1), where Y is income, β is the discount factor; p_X and p_Z are prices of processed and non-processed foods with the price of non-food consumption normalized to unity.

2.2. The results

Let x^* and z^* denote the steady-state choices of processed and non-processed foods, and W^* denote the steady-state weight. This model formulation generates two predictions of interest. The first result shows that the steady-state weight increases with the effect of processed foods on weight gain, that is, $\frac{\partial W^*}{\partial g_X} > 0$. Differentiating (2) gives:

$$V'(W + g(X, Z, E; \gamma, a)) = \frac{p_X U_K - U_X}{\beta g_X} \tag{3}$$

The left side of the equation is the marginal benefit of weight tomorrow, and the right side is the marginal cost of spending resources on weight gain. This result states that when processed foods become denser and more weight promoting, the steady-state weight is greater, as a result of concavity. This would explain the increase in the mean of weight (or BMI); it would also explain why the distribution of weight (or BMI) shifts to the right.

The second result pertains to the relative price effect: when the relative prices of processed to non-processed foods decline, the relative consumption of processed to non-processed foods rises. The optimal allocation between processed and non-processed foods at the steady state follows:

$$\frac{p_X U_K - U_X}{p_Z U_K - U_Z} = \frac{g_X}{g_Z} \tag{4}$$

Holding $\frac{g_X}{g_Z} = l$ constant, we have $U_X - U_Z = (P_X - l\, P_Z)\, U_K$. When p_X declines more than p_Z, U_X decreases more than U_Z, and consequently, x^* increases more than z^*. This might help explain the drastic increase in the consumption of processed foods during the past few decades.

Equation (3) explicitly defines weight as a function of physical activities, quantities and characteristics of food consumed, individual child-specific characteristics, and family characteristics such as income and discount rate, as in:

$$W = W\ (a,\ Y,\ \beta,\ E,\ X,\ Z,\ \gamma) \tag{5}$$

This provides the theoretical basis for our empirical specifications and consequently, hypothesis formulations.

Equation (5) can also be conceptualized as a weight production function, where inputs are physical activities and quantities and characteristics of food consumed, and technology is influenced by family income, discount rate, and individual characteristics. The technology may also be influenced by the quantities and characteristics of food consumed.

3. Empirical Specifications

3.1. Choice of the covariates

Our primary purpose is to isolate from other influences the effects of processed foods on children's weight by adequately controlling for confounders. Our model suggests that individual heterogeneity, complexity of dietary influence, and the interaction between the two are important determinants of weight.

First, there can be heterogeneity in the preferences for processed foods, or in the responses to processed foods on weight transformation. The heterogeneity may arise from genetic differences, where some children could be born with greater preferences for, or greater responses to, refined starches with added sugars and fats. A more likely source of the heterogeneity is family background. It is suggested that familiarity with repeated exposure is a key influence in children's food preferences, such as parents' consumption patterns, characteristics of school meals, and prior consumption patterns. Much of this source of heterogeneity is unobservable and likely to be correlated with socioeconomic status.

We represent individual heterogeneity in four elements: (i) observed child characteristics C, such as age and gender, (ii) observed family characteristics F, such as transitory income and permanent income, (iii) unobservable family-specific characteristics, a^F, such as discount factor, parents' food preferences, and common genetic predisposition to weight transformation among siblings, and (iv) unobservable child-specific characteristics, a^C, such as variance among siblings with respect to genetic predisposition to processed foods.

Second, weight transformation involves complex biological interactions, and its functional form is difficult to specify. Part of the difficulty in specification is capturing the complexity and multiplicity of dietary pattern, which is found to be predictors of biomarkers of obesity risk. The

recent advance in the biomedical literature[10] on obesity suggests that the weight transformation association, $g(X, Z, E; \gamma, \alpha)$, may depend on characteristics and quantities of food consumed, as well as the common representations of the diet[11], such as a child's total energy intake, total fat intake, and overall dietary discipline. We denote these child-specific dietary variables as D.

Third, we control for energy expenditure using hours of watching television as a negative proxy for exercise E, and we control for child's health behavior using supplement intakes as a proxy, which is included in the variables D.

Substituting these weight determinants into (7) yields that weight is a function of characteristics of processed foods denoted as energy density, ED, residuals in food, R, child characteristics, C, family characteristics, F, dietary and health-related behavior, D, as well as child unobservable, a^C, and unobservable family characteristics, a^F, as follows:

$$W = W(ED, R, C, F, D, E, a^C, a^F) \quad (6)$$

3.2. Choice of the functional form

To make (6) estimable, we employ Box-Cox transformation to allow flexible functional form of (6) parsimoniously. The use of Box-Cox transformation is motivated by both biomedical and statistical considerations.

Our specification reflects the weight transformation association, $g(X, Z, E; \gamma, \alpha)$, but empirically, the functional form of $g(X, Z, E; \gamma, \alpha)$ is not well understood. This requires our specifications to be flexible, such that we would allow that (a) weight determinants may influence weight in a complex way with higher-orders terms beyond the linear term, (b) there may be complex interactions between weight determinants, and (c) there may be heterogeneous effects of weight determinants on children with differing weights. There is evidence in the biomedical literature

[10] The biomedical literature on obesity has shifted from a single aggregate measure, such as total energy intake or total fat intake, to a more specific measure, such as total carbohydrate intake. It has also shifted to multiple disaggregated measures; recent studies find differing effects of components once thought of as a single measure with a uniform metric. For example, emphasis has been placed on the importance of distinguishing whole grains from refined grains to estimate the effect of carbohydrates. Similarly, distinguishing polyunsaturated and monounsaturated fats from saturated fats or total fats is critical in estimating the effect of fats.

[11] It is to note that the exact specification is subject to the availability of data. A detailed description of data and the characteristics of processed foods are in the following section.

that some natural weight-control mechanisms are malfunctioning in obese individuals, which make them more susceptible to external shocks such as the impact of processed foods.

Statistically, the dependent variable, BMI as a measure of body weight, shows that BMI is highly skewed[12]. Hypothesis testing would require the normality of the conditional distribution of the dependent variable. Heteroskedasticity is likely among individual children, which reduces the efficiency of least square estimates.

The idea of Box and Cox transformation was to find a parameter λ such that if the dependent variable, y, is transformed, as in: $y^{(\lambda)} = (y^{\lambda}-1)/\lambda$ if $\lambda \neq 0$ and $y^{(\lambda)} = \log y$ if $\lambda = 0$, then (a) $y^{(\lambda)}$ is normally distributed, (b) $y^{(\lambda)}$ has a homoskedastic conditional variance, i.e., $var\,(y^{(\lambda)} \mid x) = \sigma^2$, and (c) the mean of $y^{(\lambda)}$ is linear in covariates x, i.e., $E\,[y^{(\lambda)} \mid x] = x'\beta$. Let B denote the BMI measurement of a child's weight, and let λ denote the parameter that transforms B. We have the following estimable specification:

$$B^{(\lambda)} = a^F + \beta_R R + \beta_{ED} ED + \pi X + u \qquad (7)$$

Where $X'\pi = C'\pi_C + F'\pi_F + \pi_E E + D'\pi_D$, u is a composite random error component defined as $u = a^C + v$, and v is a random error; we assume that child-specific unobservable characteristics are uncorrelated with the remaining explanatory variables. Our null hypothesis is that the combined impacts of processed foods, β_E and β_{ED}, are jointly zero.

Given that BMI is defined as the ratio of body weight and square of height, a more flexible form of (7) is that we use a child's body weight directly as the dependent variable and include the square of the child's height as an additional regressor. Like BMI, body weight is highly skewed and Box-Cox transformation is applied to it. Let W denote a child's body weight in the unit of pound, and let θ denote the parameter that transforms W. We have the following specification:

$$W^{(\theta)} = a^F + \beta_R R + \beta_{ED} ED + \pi_{hh} H \times H + \pi X + u \qquad (8)$$

Where $H \times H$ denotes square of height in inches and π_{hh} its parameter.

[12] Both Shapiro-Wilk and Shaprio-Francia tests reject that BMI is normally distributed. The test statistics of Shapiro-Wilk and Shaprio-Francia are both approximately 0.8 with p-values of 0.0000. In addition, the index for departure from normality in Shapiro-Wilk and Shaprio-Francia tests are approximately 607 and 227; the median values of these indexes are 1 for samples from normal populations, and large values indicate non-normality.

3.3. Choice of the estimators

We estimate our parameters of interest β's (7)[13] in two steps. In the first step, we use the maximum likelihood estimator to estimate the transformation parameter λ, denoted as $\hat{\lambda}$. In the second step, we substitute $\hat{\lambda}$ into (9) and estimate the rest of the parameters, as in:

$$B^{(\hat{\lambda})} = a^F + \beta_R R + \beta_{ED} ED + \pi X + u \qquad (7')$$

To eliminate all observable or unobservable cofounders common to all children within the same family, we estimate β's in (7') using a fixed-effect estimator, which allows the unobservable family characteristics, a^F, to be arbitrarily correlated with all covariates.

By differencing among siblings, our fixed-effect estimates of (7') allow the genetic components to be partly controlled, to the extent that siblings share the same set of genes. The effects of income, price, and parental time constraints are controlled because siblings share the same set of prices and constraints. The heterogeneity of preferences is accounted for to the extent that the sources of the heterogeneity originate from the family characteristics only. However, the fixed family effect does not eliminate the within-family heterogeneity, and potential interaction between family-specific cofounders with child-specific characteristics is not controlled for.

3.4. Do processed foods increase the incidence of obesity?

We are interested in estimating the impacts of processed foods on children's chance of becoming overweight or at-risk overweight. A child is considered overweight if his/her BMI exceeds the 85th percentile of his/her age- and gender-adjusted growth chart, denoted as W^0. Let O denote whether the child is overweight, and set to one if $W > W^0$. We have the following logistic specifications in terms of the probability of obesity with Λ denoting the cumulative density of logistic distribution:

$$\Pr(W > W^0 \mid a^F, R, ED, X) = \Lambda\left(a^F + \beta_{RO} R + \beta_{EDO} ED + X'\pi_O\right) \qquad (9)$$

We estimate (9) using a fixed-effect estimator that eliminates the unobservable family characteristics, a^F.

[13] The estimation procedure for specification (8) is identical to that of (7).

3.5. Are heavyweight children more affected? A quantile regression model

Specification (7) estimates the impact of processed foods on body mass index at the mean. By using one single parameter, the Box-Cox transformation of the dependent variable allows the impact of processed foods on BMI to vary with the child's BMI, but the coefficients β_E and β_{ED} are the same across all children. We wish to employ a more flexible specification than (7) to allow the coefficients to vary with a child's BMI.

Biomedical and statistical considerations motivate us to apply the quantile regression model. First, the biomedical literature suggests that the effect of processed foods may vary across groups. It is found that insulin resistance is more pronounced or more prevalent in obese subjects than non-obese subjects. The quantile regression allows us to estimate the marginal effect of processed foods on arbitrary points of the conditional distribution of weight, and we can test whether the marginal effect is heterogeneous across the conditional distribution of weight. The benefits are twofold: statistically, we would reduce the potential bias, and policy-wise, effective intervention instruments can be formed to target the most susceptible children.

A second motivation of employing quantile regression is that the dependent variable, BMI, shows that it is far from normal distribution, and it exhibits a thick right tail. Quantile regression is superior to least square regression when the data is thick-tailed, and benefit can be substantial in improving the precision of the estimates (Koenker and Bassett 1978). The consistency of the quantile regression estimates does not require normality of the dependent variable.

The quantile regression analogy of the linear specification case of (7) is as follows:

$$W_{(q)} = a^F + \beta_{R(q)}R + \beta_{ED(q)}ED + X'\pi_{(q)} + u \qquad (10)$$

Where $X'\pi_{(q)} = C'\pi_{C(q)} + F'\pi_{F(q)} + \beta_{E(q)}E + D'\pi_{D(q)}$, q is a percentile between 0.01 and 0.99, and $q = 0.5$ corresponds to the median regression.

4. The Data

We use the Continuing Surveys of Food Intakes by Individuals (CSFII) 1994–1996, 1998 to test our hypothesis. CSFII are nationally representative cross-sectional surveys conducted by the U.S. Department of Agriculture. The surveys contain a large set of family and child characteristics

that are important determinants of weight. They also include 24-hour dietary recalls for two non-consecutive days, and each contains details of every food item a child consumed, which allows us to calculate various characteristics of food.

4.1. Characterizing the Processed Foods

Central to our analysis are the characteristics of processed foods. We use two metrics to capture the characteristics of processed foods that differentiate them from non-processed foods: food residuals and energy density. First, we construct variable residuals to represent the chemicals added to the food as what is in the food that is not recognized as common macronutrients; 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 24-hour consumption.

Second, we construct dietary energy density (kJ/g). Overall energy density is calculated as the ratio of total energy intake in calories and total weight of food in grams. Weighted energy density is calculated as the weighted average of energy density of four foods from which a child obtains the most calories, using the caloric contribution of food items as the weights. We use the weighted energy density in the regression analysis because it allows for the potential interactive effect between energy density and the amount of individual food consumption. A biomedical analogy is that glycemic load may be a better measure than glycemic index[14].

4.2. Descriptive Analysis

Of the 4,160 observations of children under age ten, 2,394 are normal weight and 1,766 are overweight or obese[15]. Table 1 contains the summary statistics for all observations and for both overweight and normal-weight children. The sampled children have an average BMI of 18.08 kg/m^2, and are on average at the 78^{th} percentile of the control population, which shows that American children are getting heavier on average. Overweight children are, on average, 6.5 kg/m^2 heavier than normal-weight children and at the 99^{th} percentile of the control population.

[14] Glycemic index describes the effect of an individual food per unit of weight on insulin production; glycemic load describes the composite effect of a specific food item for a given amount of consumption.

[15] The categorization of overweight or obesity follows the CDC growth chart definitions. For the remainder of the paper, we use the terms overweight and obese interchangeably; furthermore, we define "overweight" as those who are either at-risk overweight or already overweight.

On average, children's overall and weighted dietary energy densities are 1.16 kJ/g and 1.36 kJ/g, and their intake of residuals is approximately 14 grams with average residual concentration 9.798 (grams of residuals in 1000 grams of food). We find insignificant differences in these measures of processed foods between the overweight and normal weight children.

Children consume an average 1,682 calories and 1,490 grams of food by weight, including about 60 grams of dietary fat. Overweight children have a significantly lower caloric intake and significantly fewer grams of food than normal-weight children, and the difference in fat intake is insignificant. On average one percent of children on a disciplined diet, and there is no difference between overweight and normal-weight children. Children watched television for an average of 2.54 hours; overweight children spent significantly more time watching television than normal-weight children.

There is evidence of intergenerational transmission of obesity. American parents on average are at-risk overweight[16]. Mothers of overweight children are statistically heavier than those of normal-weight children. Compared to normal-weight children, overweight children have less family income with less educated parents, and their families are less likely to own a home or have cash savings; they are about three percent more likely to participate in the Special Supplemental Nutrition Program for Women, Infants, and Children (the WIC Program) and six percent more likely for food stamps programs.

5. Estimation Results

5.1. Do processed foods contribute to childhood obesity?

We examine whether processed foods, when represented by energy density and residuals, contribute children obesity using (7)-(9) with three forms of the dependent variables: weight, BMI and indicator of obesity. Results are presented in Table 2.

[16] The Centers for Disease Control and Prevention consider adults underweight if their BMI is less than 18.5, normal weight if it is between 18.5 and 25, overweight if it is between 25 and 30, and obese if it exceeds 30.

The fixed-effect estimates of coefficients and derivatives of (7) are presented in columns [1]-[2] in Table 2, after we apply the Box-Cox transformation to the dependent variable[17]. The effects of energy density and residuals are positive, large, and significant at one percent and ten percent levels. Calculated at the sample mean $B = 18.41$, the marginal effects are 0.86 for energy density (*ED*) and nearly 0.1 for residuals (*R*). The marginal effects of *R* and *ED* are given by: $\partial B / \partial ED = 0.0041 \times B^{1.8341}$ and $\partial B / \partial R = 0.00047 \times B^{1.8341}$. It is clear that the weight-promoting effects of processed foods increase monotonically with a child's BMI; that is, the impacts of energy density and residuals are stronger for overweight than normal-weight children.

The fixed-effect estimates of coefficients and derivatives of (8) are shown in columns [3]-[4] of Table 2 after we apply the Box-Cox transformation to the dependent variable[18]. The effects of energy density and residuals are positive, large, and significant at one percent levels. Calculated at the sample mean $W = 41.22$, the marginal effect is 3.358 for energy density and 0.374 for residuals. The marginal effects of *R* and *ED* are given by: $\partial W / \partial ED = 0.099 \times W^{0.9471}$ and $\partial W / \partial R = 0.011 \times W^{0.9471}$. It is clear that the impacts of energy density and residuals are more pronounced for overweight than normal-weight children.

The fixed-effect estimates of coefficients and derivatives of (9) are shown in columns [5]-[6] of Table 2.We find that processed foods, when presented in residuals and energy density, increase a child's probability of being overweight or at-risk overweight. The estimates of the impacts are positive and significant. Additional ten grams of residuals would increase the probability of being overweight by 29 percent. A one-unit increase in energy density would increase the probability of being overweight by 13.8 percent.

Are these estimates of social significance? Consider a four-year-old boy with 75th percentile BMI of approximately 16.5. An additional ten grams of residuals would increase his BMI by nearly

[17] The maximum likelihood estimate of the parameter, λ, in the Box-Cox transformation of the dependent variable *B* is –0.8341 with a standard error of 0.037 and *t*-stat of –22.52. We strongly reject the hypotheses of linear ($\lambda = 1$), *log* ($\lambda = 0$), and inverse ($\lambda = -1$) specifications.

[18] The maximum likelihood estimate of the parameter, θ, in the Box-Cox transformation of the dependent variable *W* is 0.0528642 with a standard error of 0.018 and *t*-stat of 2.87. We strongly reject the hypotheses of linear ($\theta = 1$), *log* ($\theta = 0$), and inverse ($\theta = -1$) specifications at 1% significance levels.

0.8 and one unit increase in dietary energy density would increase his BMI by 0.7. The BMI difference between the 75th to the 85th percentile where he is considered at-risk overweight is 0.5; the difference between the 75th to the 95th percentile where he is considered overweight is 1.5.

5.2. Are overweight children more affected by processed foods than normal-weight children?

Table 3 shows the results of quantile regression specification (10). We find that processed foods, represented in energy density and residuals, are positively correlated with children's BMI for all selected percentiles. For example, the marginal effect of residuals and energy density is 0.105 and 0.725 for the 85th percentile, and 0.079 and 0.405 for the 75th percentile; both estimates are significant at five percent levels.

The heterogeneity of the impact of processed foods among children with different weight can be summarized as follows. First, there is an overall pattern that the impact of processed foods on BMI increases along the BMI distribution. This is consistent with findings that overweight individuals are less efficient or effective in producing energy from food intake than normal-weight individuals.

Second, there is an asymmetry that the impact of processed foods is concentrated in children whose BMI is above the median of the population. The estimates are greater in magnitude and more statistically significant for the above-median children than for the below-median children. We find the impact of processed foods is strong and highly significant at the 85th percentile, which is the cutoff point for at-risk overweight.

Third, we perform several formal tests on equality of coefficients among different percentiles. Interested in the symmetry of coefficients, we test the equality of coefficients of the 95th versus 5th percentiles, 85th versus 15th percentiles, and 75th versus 25th percentiles; we reject the null that coefficients of energy density in each pair are equal with five percent, ten percent, and five percent significance levels[19]. Interested in the joint equality of coefficients at the 95th, 85th, 75th, 50th, 25th, 15th and 5th percentiles, we reject the null that the effect of energy density is uniform

[19] We also test the equality of coefficients of the 95th, 85th, and 75th percentiles against median; we reject the null that coefficients of energy density at these higher percentiles are equal to that of the median with 5 percent, 10 percent, and 5 percent significance levels, respectively.

among these percentiles at five percent level[20]; we also reject the null that the effects of energy density and residuals identical among these percentiles at five percent level.

5.3. The importance of socioeconomic characteristics and unobservable family characteristics

The quantile regression results indicate that socioeconomic characteristics are important determinants of children's weight. Income, transitory and permanent, has a strong and highly significant "protective" effect across the entire range of the distribution; the effect is asymmetric across the median and increases with weight. For example, a one percent increase of family income (at the sample mean) has a protective effect of reducing a child's BMI by 2.5, 2 and 1 for the 95th, 85th and 75th percentiles, respectively.

Consistent with existing findings that maternal employment increases a child's chance of being overweight (Anderson et al. 2004[21]), we find that parental employment has a large and significant effect on children whose weight is above the median. Having both parents who are employed full time increases a child's BMI by 3.4, 2.4 and 1 at the 95th, 85th and 75th percentiles, respectively. We find that the WIC program has a strong and negative effect on BMI, and the effect is most pronounced in overweight children: –2.67, –1.78, and 1.39 at the 95th, 90th and 85th percentiles, respectively. Participation in the food stamps program is overall positively but insignificant correlated with BMI.

6. Robustness Analysis

Several econometrics issues are of interest; the endogeneity of energy density and residuals being the most notable. Sources of endogeneity include omitted variables, simultaneity, and measurement errors. Stated differently from the causative factors, our estimates of the link between processed foods and childhood obesity could reflect the following three hypotheses: (I) *A* causes *B*, (II) *B* causes *A*, and (III) *C* causes *A* and *C* causes *B*, where *A* refers to processed foods in terms of energy density and residuals, *B* refers to obesity, and *C* refers to a variable other than *A* and *B*. It would be ideal if we had statistical tests that would enable us to reject the

[20] We perform the same set of tests for equality for coefficients for the variable residuals. The null that coefficients are equal in each hypothesis cannot be rejected at the conventional significance levels.

[21] Anderson et al. explain that working parents have less time to care for their children themselves and rely more on the market supply of childcare than non-working parents; the positive relationship between parental employment and body weight is a results of market failures of the childcare markets.

last two hypotheses directly and definitively. Our data limits us to indirect tests, which provide suggestive evidence for the rejection of hypotheses (II) and (III). We focus on the impacts of processed foods on children's body weight as in specification (8)[22] in the robustness analysis, and results are presented in Table 4[23].

6.1. Omitted variables: can we reject hypothesis (III)?

Hypothesis (III) states that there is another factor that increases childhood obesity and children's intake of processed foods. This is a special case of omitted variables bias. An example of such a factor is genetics particular to the individual child, since genetics common to non-identical siblings is already controlled for. How plausible and how important does this factor impact our estimates? We argue from several perspectives using biomedical evidence and statistical tests. First, results from genome scans suggest the existence of a few genes with substantial effects on obesity. However, the large number of genetic loci likely to be involved means that many of these genes on their own may account for only a small portion of the total phenotypic variance (Comuzzie and Allison 1998). Second, the gene pool has not evolved rapidly in the last three decades, while the increases in childhood obesity and children's intake of processed foods are pronounced with no signs of slowing.

Third, if the hypothesis of genetics as the cause of the increases in obesity and desire for processed foods holds, then one would expect that overweight children have higher intakes of processed foods than normal-weight children, because their genes increase their preference for processed foods. We can test this by examining whether the distribution of energy density and residuals differ significantly between overweight and normal-weight children. The Kolmogorov-Smirnov test for the equality of distributions shows that the distribution of processed foods does not differ significantly between the overweight and normal-weight children with *p*-value of 0.651 for residuals and 0.818 for energy density[24].

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[22] We perform the same set of robustness analysis on specification (9) when the dependent variable is children's BMI. We obtain qualitatively similar results. Details of the analysis are available from the authors upon request.

[23] There is an exception when we use (10) to assess the effect of unobservable family-specific characteristics with results in Table 5.

[24] We obtain the same conclusion when we truncate the sample by certain percentiles of body mass index. The p-values for the Kolmogorov-Smirkov test for the distribution of residuals are 0.304, 0.950, 0.578, and 0.954 for the 95, 85, 75 and 50 percentile cutoffs. P-values for the distribution of energy density are 0.503, 0.308, 0.098 and 0.519 for the 95th, 85th, 75th and 50th percentile cutoffs.

Another example of such a factor is bad parenting, in the sense that parents offer more processed foods to their children because of convenience and affordability, even though they may be aware of the adverse effects on their children's weight. This bad parenting can be extended into not encouraging participation in active physical activities. However, unless one supposes that the degree of bad parenting varies among their children, bad parenting that is common to siblings has already been controlled for through the use of unobservable family characteristics.

6.2. Are the unobservable characteristics a source of bias?

It is plausible that omitted variables influence body weight but do not influence energy density and residuals in food consumed. We first consider the potential bias from unobservable child-specific characteristics. The estimates of (7) indicate a strong and positive link between diet discipline and a child's body weight. Diet discipline may also reflect child-specific unobservable characteristics that are important to a child's body weight. We perform an artificial regression by regressing (7) without diet discipline and results are in column (3) of Table 4. The Hausman test rejects the null that all coefficients in (7) with and without diet discipline are the same.

We consider the potential bias from unobservable family-specific characteristics. We perform a series of artificial regressions by regressing (10) by taking out one family characteristic at a time, and present the results in Table 5, using the 85th percentile BMI as the dependent variable. We find that the estimates of residuals are mostly insensitive to omitted variables with the largest upward bias of 11 percent that is correlated with permanent income. The largest upward bias for the estimates of energy density is 27 percent that is correlated with WIC participation.

6.3. Simultaneity: can we reject hypothesis (II)?

Hypothesis (II) states that obesity causes children to consume energy dense processed foods. We test for the simultaneity bias using instrument variables (IV) and results are in column (4) of Table 4. Suppose there is simultaneity between body weight and dietary energy density (and residuals), where energy density increases body weight and overweight children crave energy-dense foods. We instrument the endogenous variables *ED* and *R* with total weight of food and total protein intake, and the results of the first stage regressions are strong. The Hausman test

fails to reject the null that all coefficients in columns (4) and (2) do not differ systematically with *p*-value of 0.61, suggesting that simultaneity bias is insignificant.

6.4. Measurement errors

The consequence of measurement errors of the dependent variable is that the ordinary least squares estimates would be unbiased with reduced precision – a lower *t*-stat and a reduced R^2 when the explanatory variables are measured without errors. When the explanatory variables are measured with errors that are uncorrelated with the true variables, the ordinary least squares estimates would be biased downward and the extent of attenuation is proportional to the ratio of the variance of the "signal" (true variable) and the sum of the variance of signal and the variance of "noise" (error in measurement). As the variance of noise becomes larger relative to the variance of signal, the bias increases; the fixed-effect estimates of (7) increase the variance of the noise and also increase the downward bias.

We use "repeated measurement" as instrument variables to attain consistent estimates when energy density and residuals are measured with errors and present the results in column (5) of Table 4. We instrument energy density and residuals with the repeated construction of energy density and residuals from the second dietary recall, as well as total weight of food and total protein intake. The Hausman test fails to reject the null that all coefficients in the IV estimation with "repeated measurement" and in the baseline estimation do not differ systematically with *p*-value of 0.43, suggesting that measurement error bias is insignificant.

6.5. When other covariates may be endogenous

We examine whether our estimates of the impacts of processed foods on children's body weight are sensitive to the potential endogeneity of other covariates, such as diet discipline. It is plausible that overweight children may be more likely to adopt a disciplined diet than normal weight children in an effort to lose weight because they are overweight. Column (6) of Table 4 shows the IV estimates when diet discipline is instrumented with a child's health status. The first stage result is strong and significant. The Hausman test fails to reject the null that all coefficients in columns (2) and (6) are identical with *p*-value of 0.98.

6.6. Are we capturing something other than processed foods?

We access whether our measures of processed foods primarily capture the effects of processed foods and do not capture something else that is correlated with processed foods. We find that the correlations between our measures of processed foods and other determinants are weak[25]. We apply the regression analysis with specification (7) to other health outcome that is considered irrelevant to processed foods. If we obtain strong and statistically significant effect of processed foods on the irrelevant outcome, it would cast doubt on our estimates of the impact of processed foods on body weight. Column (7) of Table 4 shows the estimates of a child's health status[26]. We find that energy density and residuals have small and insignificant effects on children's health, suggesting that it is unlikely that we are primarily capturing something other than the true effects of processed foods.

6.7. Limitations and extensions

Constrained by data, our analysis has several limitations. First, an ideal research design would ensure that the choice of processed foods, conditional upon covariates, is randomly assigned. A large and long-term experimental or quasi-experimental test would offer higher statistical power. A prospective or even retrospective longitudinal observational study would be a better research design than the cross-sectional data used in this analysis. Second, our data lacks information on energy expenditure, such as direct and accurate measures of the frequency and strenuousness of children's physical activities. Third, as discussed earlier, there may be a positive feedback between obesity and processed foods. Directly accounting for this positive feedback in the specification would lead to better estimates of the true effects of processed foods on obesity.

6.8. Policy Implications

We have estimated the contribution of processed foods in terms of residuals and energy density to childhood obesity, above and beyond the effects of total energy and fats intakes, the child's own health behavior, and family socioeconomic characteristics. Our results suggest that reducing the characteristics of processed foods in terms of residuals and energy density may mitigate the current childhood obesity epidemic.

[25] Energy density and residuals are both positively correlated with age and fat intake with correlation coefficients of approximately 0.25. Energy density and residuals are weakly correlated with a correlation coefficient of –0.004.
[26] Health status is a five-integer-valued categorical variable with 1 = Excellent and 5 = Poor.

The transformation of processed foods in recent decades has benefited food producers through lowered production costs, and consumers through declining prices. We calculate producer surplus and consumer surplus using the data compiled by USDA 2002. The profit differential between 1992 and 1997, adjusted for inflation, is $18.231 billion (in 1997 dollars) for the entire processed foods industry. The consumer food expenditure differential, which is defined as food purchased by all families and individuals, is nearly zero after adjusting for inflation. The sum of profit and consumer food expenditure differentials is $18 billion (in 1997 dollars) or $19 billion (in 2001 dollars), and this is the total cost of "reversing" processed foods of 1997 to processed foods of 1992.

The U.S. Surgeon General estimates that the national cost of an overweight and obese population is $117 billion (HHS 2001). Finkelstein, Fiebelkorn, and Wang (2003) report that obesity-related medical expense was $92.6 billion (in 2002 dollars) and accounted for 9.1 percent of total U.S. medical expenditures in 1998, and that Medicaid and Medicare paid approximately half of these costs. An average obese adult spends $395 more per year on medical care than an adult of normal weight (Sturm 2002). Wang and Dietz (2002) estimate that obesity-associated hospital costs for children aged 6 to 17 rose to more than $127 million annually, a threefold increase from the late 1970s. Obese children and their parents also bear the economic burden of obesity: obesity is negatively correlated with educational attainment and associated earnings (Cawley 2004). Overweight children are more likely than normal children to become overweight adults, who have a 50–100 percent increased risk for premature death, compared to those of normal weight (HHS 2001).

We calculate the economic benefits of "reversing" processed foods on reducing childhood obesity as follows. The intensification of processed foods from 1992 to 1997 can be characterized by an increase of eight grams in residuals and 0.8 in energy density. From estimates in Table 2, this intensification would increase the incidence of obesity by 34.4 percent. With the total cost of obesity being $117 billion, the benefit would be $40 billion (both are in 2001 dollars). Taken together, the net benefit would be $21 billion.

7. Conclusion

In this paper, we estimate the impact on children's weight of processed foods, which are characterized by energy density and the amount of unidentified residuals in the food. We find strong empirical evidence that increasing energy density and residuals would increase children's weight (in pounds), body mass index, and their chance of becoming overweight. Our estimates are robust to unobservable family-specific characteristics, simultaneity, omitted variables, and measurement errors. We also find that overweight children are more susceptible to the impacts of processed foods than normal-weight children.

The social significance of our estimates can be illustrated with a four-year-old boy with a 75th percentile BMI of approximately 16.5. An additional ten grams of residuals would increase his BMI by 0.8, and a one-unit increase in dietary energy density would increase his BMI by 0.7; he would be considered at-risk overweight resulting from each of these changes, and overweight resulting from both changes.

Our findings suggest that the changed characteristics of processed foods are partly responsible for the increase in childhood obesity. Policies that reduce the amount of additives and energy density may be considered as a contributing solution to the obesity epidemic. The pervasiveness and "inevitability" of additives and high energy density in processed foods bear an analogy from the history of air pollution. What we have learned in controlling air pollution may be applicable to processed foods.

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Table 1: Summary Statistics

Variables	**All Children**	**Overweight**	**Normal weight**	**t-stat**
BMI (kg/m^2)	18.08 (0.07)	21.84 (0.12)	15.28 (0.03)	53.03
Z-score BMI	0.79 (0.03)	2.45 (0.02)	–0.42 (0.02)	138.89
Total energy intake (kJ)	1682 (9.15)	1657 (14.45)	1700 (11.79)	–2.30
Total fat intake (g)	60.81 (0.43)	60.47 (0.68)	61.07 (0.56)	–0.68
Total food weight (g)	1490 (8.29)	1470 (12.65)	1505 (10.96)	–2.09
Overall energy density (kJ/g)	1.16 (0.0045)	1.16 (0.007)	1.17 (0.006)	–1.08
Weighted energy density (kJ/g)	1.36 (0.008)	1.36 (0.012)	1.36 (0.011)	0.00
Total residuals (g)	14.23 (0.08)	14.11 (0.13)	14.32 (0.11)	–1.23
Residual concentration 10^{-3}	9.79 (0.04)	9.82 (0.06)	9.77 (0.056)	0.06
TV hours	2.54 (0.03)	2.63 (0.05)	2.47 (0.04)	2.50
Diet discipline	0.01 (0.002)	0.01 (0.003)	0.01 (0.002)	0.00
Freq. taking vitamins	2.07 (0.01)	2.18 (0.02)	1.99 (0.02)	6.71
Age	4.20 (0.03)	4.05 (0.04)	4.32 (0.04)	–4.77
Mother BMI (kg/m^2)	25.60 (0.27)	26.76 (0.46)	24.77 (0.28)	3.70
Father BMI (kg/m^2)	26.90 (0.209)	27.07 (0.35)	26.77 (0.28)	0.67
Mother education	13.08 (0.12)	12.53 (0.19)	13.46 (0.15)	–3.70
Father education	13.44 (0.13)	13.21 (0.19)	13.60 (0.17)	–1.53
Family income ($1,000)	43.59 (0.45)	39.19 (0.66)	46.85 (0.60)	–8.59
Family size	4.51 (0.02)	4.51 (0.03)	4.50 (0.03)	0.23
Home owners	0.62 (0.008)	0.57 (0.01)	0.66 (0.01)	–6.36
Savings over $5,000	0.39 (0.008)	0.32 (0.01)	0.44 (0.01)	–8.48
Mother employed FT	0.04 (0.003)	0.04 (0.005)	0.05 (0.004)	–1.56
Father employed FT	0.09 (0.005)	0.09 (0.007)	0.10 (0.006)	–1.08
Anyone on WIC	0.16 (0.006)	0.18 (0.009)	0.15 (0.007)	2.63
Food stamps	0.16 (0.006)	0.19 (.009)	0.13 (0.007)	5.26
Sample size	*4,087*	*1,742*	*2,345*	

Notes: These are un-weighted sample averages. Standard errors are in parentheses. Variable *Diet discipline* is a binary indicator. Variable *Freq. taking vitamins* is a categorical variable with value 1 = daily, 2 = often but not daily, 3 = never. Overweight or obese is categorized by the standardized z-score BMI developed by the Centers for Disease Control and Prevention. The last column presents the t-stat under the null that the sample average of overweight or obese children is the same as the sample averages of normal-weight children.

Table 2: Sibling-Differencing (fixed effect) Estimates of the Impact of Processed Foods

Dependent variable (specification)	BMI $^{\lambda}$ (7)		Weight $^{\theta}$ (8)		IsObese (10)	
	Coefficients [1]	Derivatives [2]	Coefficients [3]	Derivatives [4]	Coefficients [5]	Derivatives [6]
Energy density (kJ/g)	0.0041 *** (0.0014)	0.859 *** (0.287)	0.099 *** (0.016)	3.358 *** (0.097)	0.119 ** (0.054)	0.138 ** (0.070)
Residuals (g)	0.00047 * (0.00025)	0.099 * (0.052)	0.011 *** (0.003)	0.374 *** (0.098)	0.560 * (0.310)	0.029 ** (0.013)
Total fat (g)	-0.00004 (0.000055)	–0.009 (0.012)	-0.002 (0.0006)	-0.054 (0.020)	0.007 (0.012)	0.002 (0.003)
Total energy (1,000 kJ)	0.0007 (.0032)	0.155 (0.663)	0.076 ** (0.037)	2.588 ** (1.246)	–1.130 (0.745)	–0.279 (0.181)
TV hours	0.00009 (.0004)	0.019 (0.080)	0.012 *** (0.004)	0.390 *** (0.150)	0.113 (0.080)	0.028 (0.065)
Diet discipline	0.0155 *** (0.005)	3.239 *** (1.026)	0.153 *** (0.057)	5.198 *** (1.927)	2.699 ** (1.197)	0.401 ** (0.217)
Freq. vitamins	0.001 (.001)	0.252 (0.239)	-0.018 (0.013)	-0.010 (0.447)	0.679 ** (0.321)	0.167 ** (0.067)
Age	–0.0092 *** (0.0008)	–1.935 *** (0.173)	0.110 *** (0.011)	3.725 *** (0.372)	–1.098 *** (0.194)	–0.271 *** (0.065)
Age X Age	0.0007 *** (0.00007)	0.150 *** (0.016)	-0.003 *** (0.0008)	0.102 *** (0.029)	0.066 *** (0.016)	0.016 *** (0.005)
Female	–0.0007 (0.001)	–0.139 (0.213)	–0.015 (0.012)	–0.520 (0.400)	–0.011 (0.202)	–0.003 (0.050)
Height X Height	---	---	0.00026 *** (0.00002)	0.009 *** (0.00007)	---	---
R^2	0.14	0.14	0.80	0.80		

Notes: ***: Significant at 1%; **: significant at 5%; *: significant at 10%. All estimates account for fixed family effect. Standard errors are in parentheses. Number of observations is 5,464 with 4,332 families for columns [1]-[4]. Sample size is 627 with 275 families in [5]-[6]. The Box-Cox transformation parameters, $\lambda = -0.8401554$ and $\theta = 0.0528642$. The derivatives are calculated at the sample mean of the dependent variable B = 18.41 and W = 41.22. Reported R-square is within R-square.

Table 3: Quantile Regression Estimates of the Impact of Processed Foods on Selected Percentiles of BMI

Percentiles	**0.95**	**0.85**	**0.75**	**0.50**	**0.25**	**0.05**
Energy density (kJ/g)	1.520 *** (0.601)	0.725 ** (0.243)	0.405 ** (0.212)	0.025 (0.136)	–0.046 (0.010)	0.023 (0.186)
Residuals (g)	0.157 (0.147)	0.105 ** (0.055)	0.079 ** (0.034)	0.034 (0.021)	0.029 (0.018)	0.012 (0.018)
Transitory income (ln)	–2.517 *** (0.566)	–1.406 *** (0.261)	–1.061 *** (0.186)	–0.447 *** (0.133)	–0.256 *** (0.089)	0.162 (0.152)
Permanent income	–0.203 (0.469)	–0.376 * (0.209)	–0.348 ** (0.151)	–0.276 *** (0.124)	–0.116 (0.075)	–0.059 (0.135)
Parents employment	1.704 *** (0.557)	0.528 * (0.284)	0.532 *** (0.191)	0.072 (0.124)	–0.041 (0.114)	–0.173 (0.158)
Program: WIC	–2.670 *** (0.870)	–1.393 *** (0.488)	–0.792 *** (0.308)	–0.489 *** (0.190)	–0.468 *** (0.134)	–0.381 * (0.215)
Program: food stamps	0.544 (1.138)	0.224 (0.546)	0.246 (0.370)	0.013 (0.224)	–0.126 (0.175)	–0.388 (0.246)
Fat intake (g)	–0.003 (0.025)	0.006 (0.012)	–0.006 (0.008)	0.0002 (0.005)	–0.003 (0.004)	–0.009 (0.006)
Energy intake (1,000 kJ)	–0.803 (1.500)	–1.304 * (0.763)	–0.107 (0.500)	–0.149 (0.314)	–0.267 (0.297)	0.151 (0.402)
Total weight (g)	–0.0003 (0.001)	0.0001 (0.0006)	–0.0005 (0.0004)	–0.0001 (0.0002)	0.0002 (0.0002)	–0.0001 (0.0003)
TV hours	0.333 ** (0.164)	0.162 ** (0.083)	0.061 (0.042)	0.064 ** (0.033)	0.018 (0.029)	0.014 (0.047)
Is on diet	4.927 (4.099)	1.042 (1.624)	1.659 *** (0.575)	0.903 (0.664)	0.324 (0.515)	0.018 (0.411)
Freq. taking vitamins	0.917 *** (0.347)	0.568 *** (0.153)	0.444 *** (0.097)	0.330 *** (0.056)	0.117 ** (0.050)	–0.081 (0.068)
Age	–3.138 *** (0.524)	–1.859 *** (0.292)	–1.613 *** (0.173)	–1.040 *** (0.119)	–0.642 *** (0.071)	–0.667 *** (0.125)
Age X Age	0.233 *** (0.047)	0.151 *** (0.026)	0.140 *** (0.016)	0.089 *** (0.011)	0.051 *** (0.007)	0.055 *** (0.011)
Female	0.012 (0.581)	–0.009 (0.283)	–0.012 (0.185)	–0.277 ** (0.118)	–0.329 *** (0.094)	–0.416 *** (0.149)
Constant	38.823 *** (2.598)	29.293 *** (1.282)	26.178 *** (0.857)	20.736 *** (0.555)	17.982 *** (0.434)	14.938 *** (0.605)
R^2	0.08	0.05	0.05	0.03	0.02	0.02

Notes: ***: Significant at 1%; **: significant at 5%; *: significant at 10%. Estimation equation is (10) with a quantile regression estimator – sqreg in STATA with 100 replications in calculating the bootstrapping standard errors. The dependent variable is a particular percentile of children's body mass index. Standard errors are in parentheses. Each column is estimated for a specific quantile and all quantiles are estimated independently. Sample size is 5,227.

Table 4: Robustness analysis with specification (8)

	Baseline		Omitted Variables	Simultaneity	Repeated Measurement	Endo. Covariate	"Irrelevant" Health
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Energy density	0.099 *** (0.016)	3.358 *** (0.097)	0.097 *** (0.016)	0.187 *** (0.040)	0.282 *** (0.095)	0.095 *** (0.017)	–0.0001 (.007)
Residuals	0.011 *** (0.003)	0.374 *** (0.098)	0.011 *** (0.003)	0.029 *** (0.007)	0.036 ** (0.018)	0.011 ** (0.003)	0.048 (.041)
Total fat intake	–0.002 (0.0006)	–0.054 (0.020)	–0.002 (0.0006)	–0.002 *** (0.0007)	–0.003 *** (0.0009)	–0.002 *** (0.0009)	–0.002 (0.002)
Total energy intake	0.076 ** (0.037)	2.588 ** (1.246)	0.078 ** (0.037)	–0.042 (0.057)	–0.065 (0.149)	0.083 ** (0.038)	0.023 (0.093)
TV hours	0.012 *** (0.004)	0.390 *** (0.150)	0.011 *** (0.004)	0.008 * (0.0047)	0.004 (0.006)	0.011 ** (0.0045)	–0.010 (0.011)
Diet discipline	0.153 *** (0.057)	5.198 *** (1.927)	—	0.173 *** (0.059)	0.191 *** (0.063)	–0.214 (0.249)	1.123 *** (0.143)
Freq. vitamins	–0.018 (0.013)	–0.010 (0.447)	–0.018 (0.013)	–0.013 (0.014)	–0.009 (0.015)	–0.020 (0.014)	–0.005 (0.034)
Height ^ 2	0.00026 *** (0.00002)	0.009 *** (0.00007)	0.00026 *** (0.00002)	0.00026 *** (0.00002)	0.00026 *** (0.00002)	0.00025 *** (0.00002)	–0.00008 (0.000055)
Hausman test stat, p-value	R^2 = 0.80	R^2 = 0.80	11.21 (10) p = 0.33	8.17 (10) p = 0.61	10.25 (10) p = 0.43	2.94 (10) p = 0.98	R^2 = 0.06

Notes: ***: Significant at 1%; **: significant at 5%; *: significant at 10%.

a. All columns use fixed effect with Box-Cox transformed body weight in pound as the dependent variable based upon specification (8). Standard errors are in parentheses. Column (1) and (2) are estimates of derivatives and coefficients as in Table 2, and the rest are coefficients estimates.
b. Column (3) is estimated without " Diet discipline".
c. Column (4) is estimated using two-stage least square where energy density and residuals are instrumented with total food weight and total protein intakes. The first stage R^2 and F-stat for energy density are 0.40 and 67, and for residuals, 0.83 and 515.
d. Column (5) is estimated using two-stage least square where energy density and residuals are instrumented with second day measures of energy density and residuals, and total food weight and total protein intakes. The first stage R^2 and F-stat for energy density are 0.19 and 20, and 0.77 and 277 for residuals,
e. Column (6) is estimated using two-stage least square where "Diet discipline" may be endogenous and is instrumented with the child's health status. The first stage R^2 and F-stat are 0.07 and 7.55.

Table 5: Robustness Analysis of specification (10)

	(1)	(2)	(3)	(4)	(5)	(6)
Energy density (kJ/g)	0.725 *** (0.279)	0.719 *** (0.274)	0.919 *** (0.315)	0.834 *** (0.298)	0.838 *** (0.294)	0.729 ** (0.327)
Residuals (g)	0.105 *** (0.038)	0.101 *** (0.037)	0.097 ** (0.043)	0.105 *** (0.040)	0.117 *** (0.040)	0.067 (0.044)
Transitory income (ln)	–1.406 *** (0.210)	–1.451 *** (0.188)	–1.354 *** (0.231)	–1.379 *** (0.224)	–1.683 *** (0.193)	—
Permanent income	–0.376 ** (.184)	–0.367 ** (.180)	–0.308 (0.210)	–0.389 ** (0.197)	—	–1.210 *** (0.188)
Parents employment	0.528 *** (0.246)	0.526 ** (0.241)	0.602 ** (0.278)	—	0.621 *** (0.261)	0.577 ** (0.290)
Program: WIC	–1.393 *** (0.341)	–1.307 *** (0.330)	—	–1.384 *** (0.362)	–1.266 *** (0.362)	–0.947 ** (0.393)
Program: FS	0.224 (0.388)	—	–0.384 (0.430)	0.182 (0.413)	0.150 (0.410)	1.376 *** (0.416)
R^2	0.05	0.05	0.05	0.05	0.05	0.05
sample size	5227	5239	5266	5227	5385	5227

Notes: ***: Significant at 1%; **: significant at 5%; *: significant at 10%. Estimation equation is (10) with a quantile regression estimator, where the dependent variable is the 85th percentile of children's body mass index. Standard errors are in parentheses. See notes in Table 3 for definitions of variables. Column (1) is a replication of column (.85) of Table 3. All columns use the same set of regressors as in column (1) except those indicated otherwise. For brevity, results of other regressors are not shown.

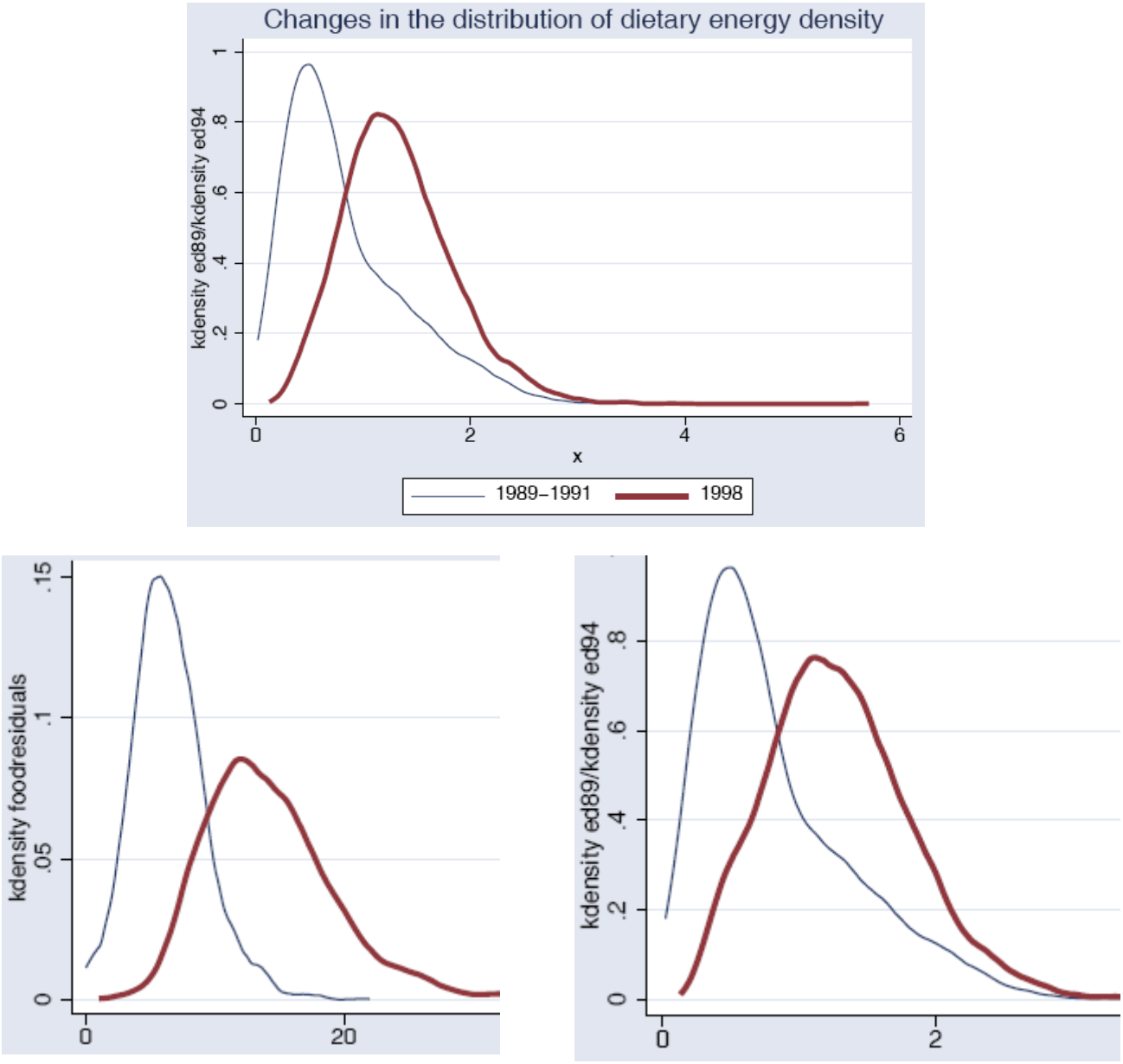


Figure 1: Changes in the distributions of age-gender-adjusted z scores of body mass index (top), food residuals (bottom left) and dietary energy density (bottom right) in children's diet between 1989–91 and 1998.

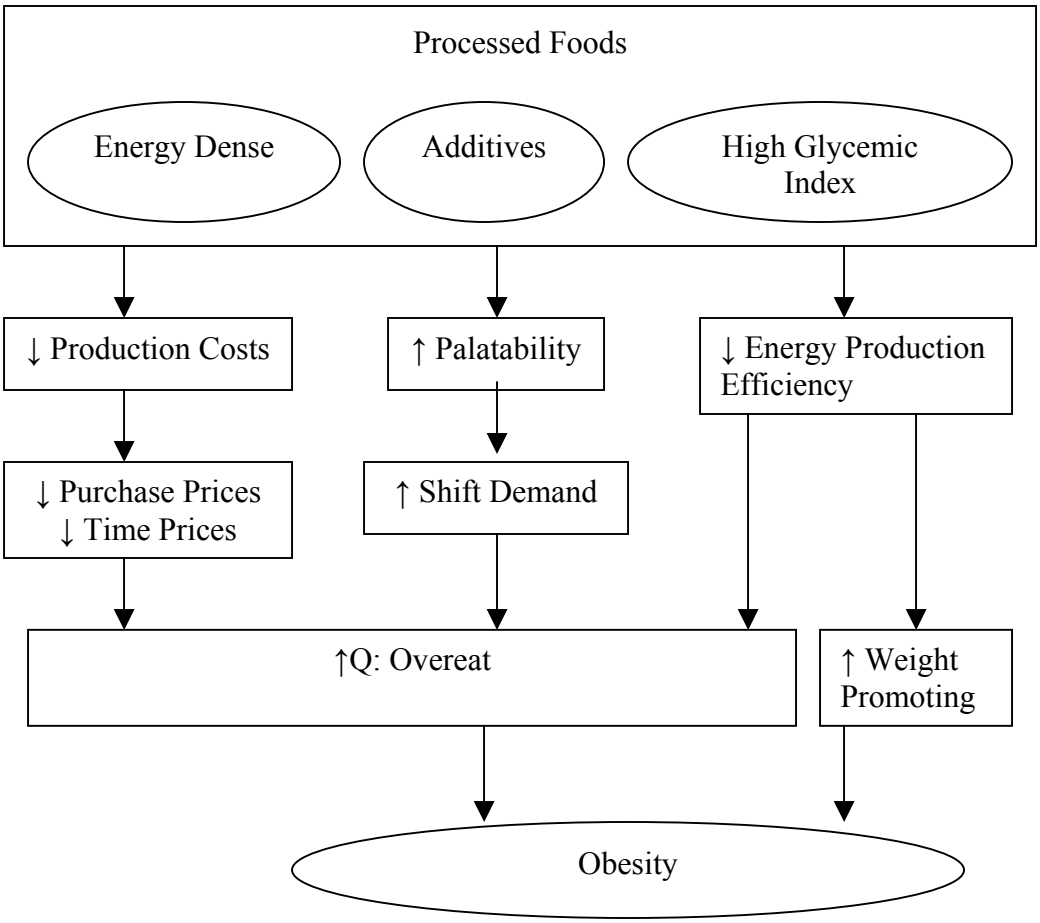
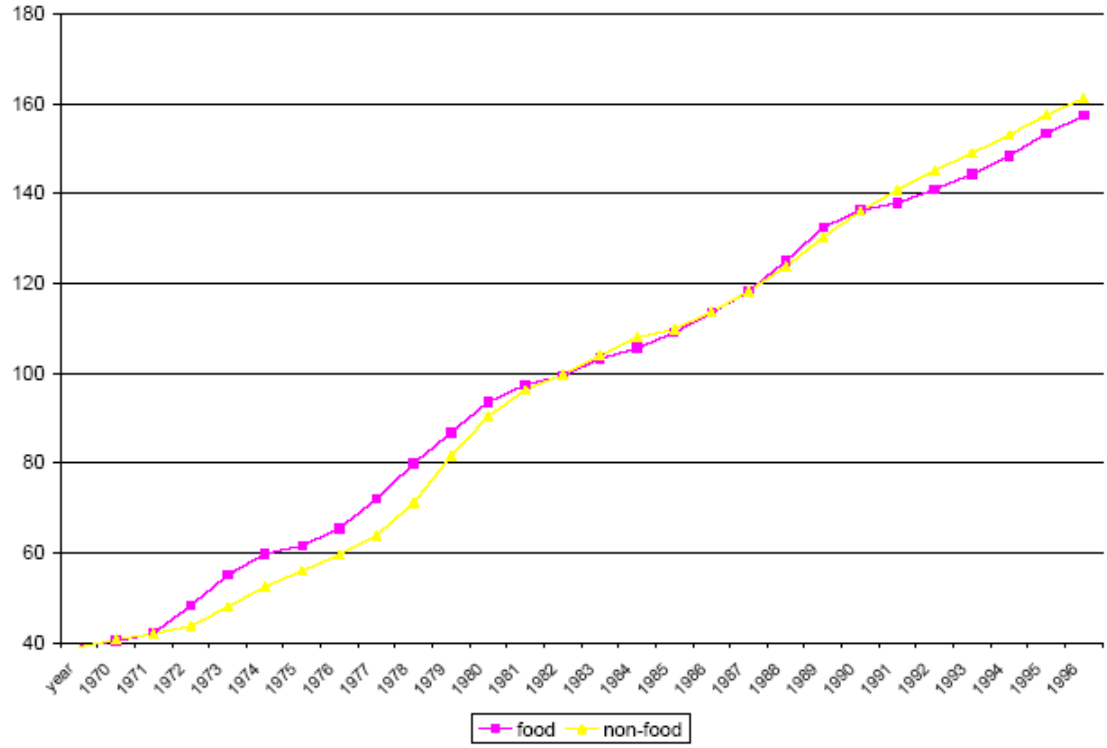


Figure 2: A conceptual framework on how processed foods may contribute to obesity



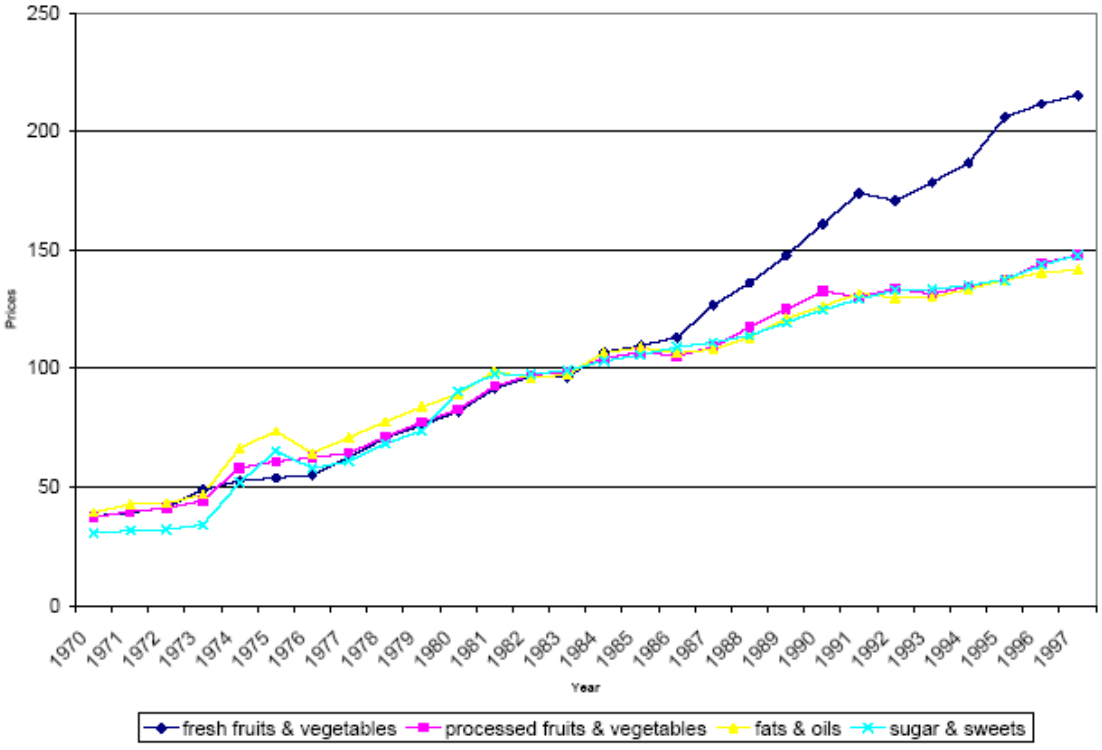


Figure 3: Prices of foodstuffs versus non-foodstuffs (top); prices of processed versus non-processed foods (bottom). Data source: USDA June 2002 with 100 = 1982–1984 prices.