

Tipping the Scales: Why Are American Kids Getting Fatter?

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Abstract

The so-called epidemic of childhood obesity is not particular to American culture but has been shown to be a worldwide phenomenon typified by a substantial increase in the prevalence of obesity occurring within a short time span. Despite a prodigious body of literature on obesity within the medical community, little work on this subject has been done by economists. This paper attempts to bring an economic perspective on the child obesity explosion that has been well documented but generally ill explained. Analysis of diet diary data rejects putting the blame of child obesity trends on diet. Similarly, changes in labor market allocations, parent education, family composition, and medical innovations were not found to be important. Empirical evidence on time allocations by children is also explored in the paper.

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1 Introduction

Hardly a day passes when the "epidemic" of obesity in America is not mentioned in the media. Surprisingly, though, despite this, there has been a paucity of economic research on obesity, not to mention child obesity. Being a serious and pervasive health issue, considerable research has already been done by the medical community, but, while their research documents the current trend in child obesity, the quality of the research in explaining the trend is usually lacking. Without doubt, though, numerous medical studies have painted a consistent picture of child obesity worldwide: the childhood obesity epidemic is a recent phenomenon, developing mainly in the 1980s, and most of the increase in obesity has occurred among kids who were already relatively obese to begin with. For example, Chinn and Rona(2001) found after looking at British children that, although there was little change in obesity from 1974 to 1984, the subsequent ten year period saw the fraction of children considered overweight increase from 5.4% to 9% for boys and from 9.3% to 13.5% for girls. In Germany, Kromeyer-Hauschild, et al.(1999) documented a similar pattern. From 1974 to 1985, the incidence of being overweight rose from 10% to 11.8% for boys and from 11.7% to 13% for girls. For the period 1985 to 1995, these figures jumped to 16.3% and 20.7%, respectively. Kalies, et al.(2002), also looking at German children, demonstrated that most of the rise in obesity in the last two decades occurred above the 75th percentile in the BMI distribution. In Japan, Kotani, et al.(1997), looking at over 200,000 Japanese schoolchildren, found that, although the incidence of obesity actually fell slightly over the years 1974 to 1979, the obesity rate nearly quadrupled from 3% to 11% from 1979 to 1995. In Taiwan, Chu(2001) documents an alarming jump in the prevalence of obesity among schoolchildren of one-third from 1980 to 1994. In China, Luo and Hu(2002) showed

that the average BMI for urban children rose over ten percent and prevalence of being overweight nearly doubled from 15 to 29 percent in just nine years since 1989. These time patterns have also been reflected among adults in the United States, also. The average BMI for people aged 20 to 74 years was relatively flat up to the 1980s after which it exploded from a BMI of approximately 25 to an average BMI of over 33.¹

Furthermore, it does not need to be said that obesity is not merely a superficial problem (see Figure 1). Several large medical studies have demonstrated positive, convex relationships between various types of killer disease and obesity. One large, longitudinal sample of Harvard alumni showed that those with BMIs of 26 or larger died two-thirds more often than others with BMIs of 22.5 or less. In 1993, more than half of mortalities of adults with BMIs larger than 30 was attributed to their being overweight. Obese adults are several times more likely to experience hypertension, type II diabetes, endocrinal problems, and certain cancers.² In regard to the diseases of diabetes, cardiovascular disease, hypertension, cancer, and gall bladder disease, Colditz(1992) estimated that obesity³ was responsible for over 5% of the total economic costs of having these diseases in 1986, or \$66.4 billion in today's dollars. Of course, it can be argued that overweight kids are not doomed to be overweight as adults, or even that these kids will necessarily suffer from any serious morbidity. In fact, medical research fails to show much evidence at all that obese children suffer from any particular morbidity over normal weight kids.⁴ This being the case, whether or not we should lose sleep over the current, worldwide explosion in childhood obesity depends on to what degree child obesity leads into obesity as an adult. The medical literature suggests that obese schoolchildren are from twice as likely to nearly seven times as likely to continue to be obese as an adult.⁵ My own estimates using NLSY data conform to this range. People whose BMI was over one standard

deviation above the mean when younger than 18 in 1981 were seven times more likely to continue to be overweight relative to their peers twenty years later in 2000, and twice as likely to be overweight if we compare their BMIs in the year 2000 with the mean BMI in 1981.⁶ So, even though medical opinion is still out in regard to the direct health affects of child obesity, this certainly is not the case with adult obesity, and, because a large number of obese children remain obese as adults, the increase in child obesity worldwide cannot be treated as simply media hype.

However, even if we all agree that we should be worried about our kids getting larger, the other question is what is the underlying reason? The primary behavioral explanations put forth by the medical community have usually focused on media consumption, e.g., video games and television, or food consumption, e.g., calorie-laden fast food. Economic research has focused on whether the growth in adult obesity is primarily due to technological change in food production, thereby driving the real cost of food down substantially, or mainly due to technological change in the work environment, which has driven up the value of leisure and, at the same time, made labor much more sedentary than in the past. As a result, burning off calories have become expensive, because people have to sacrifice valuable leisure time to do it. Now, technological progress may certainly be at work behind child obesity as well. Inexpensive food combined with heart pounding video games or movies are bounties of the technological progress from the last few decades, and they may indeed be the culprits behind child obesity. But, there are other plausible technology-based hypotheses for child obesity other than changes in the prices of calories and exercise. For example, improving real wages of parents would increase the opportunity cost of taking time to either play with their children or transport their children to areas or friends for exercise-intensive play. In addition, better medical technology in treating either obesity directly or the diseases associated

with obesity would reduce the long term cost of being obese.

The trouble (and the reason behind the present paper) is that there has been hardly any economic research into child obesity as opposed to adult obesity, and, although there has been a considerable amount of medical research, these papers are invariably stricken by fundamental methodological flaws. One of the more common defects, for example, has been simply treating obesity as a natural experiment as if fatness was some kind of unexpected, exogenous shock. This paper is intended to be as comprehensive an examination as possible, given the data employed, into plausible hypotheses that might underlie the trend in child obesity for the last 30 years within the United States. Considering the striking similarities of child obesity trends across so many different regions and cultures on both sides of the world, restricting ourselves to U.S. data should not significantly negate the generalizability of the results presented in this paper. We utilize five different datasets containing data together on over 40,000 children, and including time diary and diet diary information. One of these datasets, the PSID Child Supplement, contains detailed data on time allocations across various activities, which has, to my knowledge, not been utilized by any other paper on the subject of child obesity. Toward our goal of explaining trends, we will primarily examine household characteristics, time allocations, caloric intake, and food prices.

First, though, let us look at what has been happening to U.S. child obesity according to our own data. Consider Table 4 which presents summary statistics for child BMI from three datasets: NHES Cycle II data conducted in the years 1963-65, 1997 PSID Child Supplement data, and NLSY child data for the years 2000 and 1986.⁷ Comparing the NHES from 1963-65 with NLSY data from the year 2000, we can see that child obesity has risen by about 14% over this time period. Further, almost all of this increase has taken place within the last 20 years. Whereas the growth rate of child

BMI was virtually zero between NHES Cycle II and the 1986 NLSY data, the yearly rate of growth rose to 0.8% between 1986 and the 1997 PSID sample, and accelerated to 1.4% per annum from 1997 to 2000. The upper tail of the distribution grew at a much faster rate than that of the lower tail. This can be seen in Figure 2, which charts the deciles of child BMI for the above datasets. Compared to negligible increases in the proportion of children at the bottom 25th percentile of the BMI distribution, the proportion at the 75th percentile rose 15% and those at the 90th percentile increased by a full 20%. Thus, we too confirm the findings from international medical studies. Both the recentness and rapidity of the growth in childhood obesity must be borne in mind when evaluating any hypothesis that tries to establish causality.

To measure obesity, we will use the usual body mass index (BMI) defined as weight in kilograms divided by height in meters squared. The benefits of using BMI is that it is fairly well correlated with body fat, and less prone to measurement error than other techniques like skinfold thickness measurements.¹² We look at all kids aged seventeen and younger.

The following section presents a review of the existing literature on child obesity. We present a model of child obesity in Section 3. Sections 4 and 5 describe the data used and present empirical results. Finally, concluding remarks are given in Section 6.

2 Literature Review

It should be first noted that the following four works are the principal papers from the economic literature. As for the medical literature, some of the major works are commented upon below, but this review is not meant to be exhaustive of medical research papers. Interested readers may peruse

the bibliography for a more complete listing.

Philipson and Posner(1999) and Lakdawalla and Philipson(2002) attribute trends of adult obesity to technological change, which has reduced real food prices at the same time that the work environment has shifted from manual labor to sedentary labor, thus raising the price of burning calories. Lakdawalla and Philipson constructed a measure of job strenuousness and presented empirical evidence that showed that the shifting away of people from strenuous work did contribute to increasing adult BMI. We will use the model presented in both papers as a building block for our own work on child obesity.

Cutler, Glaeser, and Shapiro(2002) attribute adult obesity within the U.S. to the falling shadow price of food. Food is much cheaper to produce, both in terms of market inputs and in time. They found that people are consuming more meals, i.e., snacks, though, interestingly, people are not eating bigger meals. Changes in the time allocation across activities varying in energy expenditure were not found to be significant in explaining the trend in obesity. Thus, the authors attribute all of the increase in obesity to increases in calorie intake. The PSID data we will use contain time diary information for children, while the NHES has nutrition data, so we should be able to see if their results extend to children.

Anderson, et al.(2002) use NLSY data to find that an additional hour worked per week by the mother increased child obesity by about 0.5%. However, this explained only a very small proportion of the trend in child obesity over the sample time period. Surprisingly, working more weeks per year had no effect on child obesity despite controlling for hours worked per week. When separated by race, education, and income quartile, this result only held for non-Hispanic Whites, High School graduates, and the fourth income quartile. Number of children in the household generally had a

small negative effect on a child's obesity, while average family income had no statistically significant effect. The authors, however, did not concern themselves with explaining child obesity beyond presenting empirical evidence that maternal hours of work effects obesity slightly and mentioning possible hypotheses on what may be causing the trend in child obesity. It is unclear, for example, by what channel a longer workday for the mother would lead to their child getting more obese. Is it through nutrition, and, if so, why? Or, is it via increased indulgence by the child? They do not try tackling any of this. This paper will revisit the importance of labor supply decisions by parents on child obesity.

Vuille and Mellbin(1979) use longitudinal data and stepwise regression analysis on Swedish children. They found that for male children, the number of children in their family, their degree of activeness, and the height of their mother were all factors that were negatively associated with their weight relative to peers. Mother's BMI, treatment for "nutritional problems", weight gain in their first year, and a healthy appetite all had positive relationships with relative weight. For female children, activity level was the only factor that had a positive relation to relative weight. The BMI of their mothers, their father's weight, being an only child, and treatment for nutritional problems were positively associated with the child's relative weight.

Wolfe, et al.(1994) find that among households that the authors classified as having low socioeconomic status⁸, children with single parents tend to be thinner than those with having both parents. Specifically, they found that these children were about 5% less likely to have a BMI above the 90th percentile. Among households of high socioeconomic status, children of single parents were actually more likely to be obese compared to those with two parents. Within households with dual parenting, socioeconomic status had a negative effect but this was not statistically significant.

However, higher socioeconomic status did lead to a higher incidence of obesity among children having only one parent. The authors also found that a child with more siblings tended to be thinner, though the results were not statistically significant. There was also no statistically significant effect on obesity from sex or race. However, the authors failed to consider parental obesity, which, for example, would confound their results for socioeconomic status if labor market outcomes are not independent of a worker's obesity. Activity choices by the child are also not considered.

Concerning the relationship between child obesity and television viewing, Dietz and Gortmaker(1985) use NHES cycle II and III data to find that the probability of a child being obese rose 2% for each extra hour of television watched.⁹ The average number of hours spent in front of the TV by children aged 6 to 11 was reported to be 24 hours per week in 1981. Dietz and Gortmaker did not adjust for endogeneity bias between obesity and television watching, however, despite having longitudinal data, nor did they control for other activities engaged in by the child or for the child's diet, although all these factors are important if a child's obesity is subject to rational choice. Anderson, et al.(1998) used NHNES data and purported to find a positive relation between hours spent television viewing and child obesity. However, their methodology used nothing more complex than simply plotting graphs showing relationships between BMI, hours of TV watched, and bouts of exercise undertaken each week. Gortmaker, et al.(1996) used NLSY data and found that watching more than five hours of television daily increased the risk of a child being overweight by 5.3% even after controlling for various characteristics.¹⁰ Children watched an average of 4.8 hours of television per day. The authors say that all control variables they considered were not significant other than the mother's AFQT score, though they do not present these estimates. They disregard possible simultaneity bias, although they do acknowledge that it might be a problem.

Locard, et al.(1992) looked at over 9,000 five year old children and found that very obese children were only statistically significantly different from normal weight children in that they watched a lot of television, snacked between meals, but slept relatively fewer hours at night. This study did not impose any controls in its methodology, and so the usefulness of their results is very limited.

Klesges, et al.(1991) studied the interaction of nutritional choices of 53 children with parental monitoring. Children were allowed to select from a wide range of food items. Compared to the selection chosen by their mothers, that of the children was found to contain twice as many calories as preferred by their mothers. Further, the children's selection was heavy in sugar, saturated fats, and sodium. Neither the weight of the child nor that of the mother were significant variables in the results. The main drawbacks of this study are the tiny sample size considered and the fact that the sample was not random. The researchers simply put an ad in the paper to solicit mother-child pairs. All but two percent of the participants were white and over 80% were middle or upper income. The study seems to say that child obesity is operating through other avenues than nutrition if most parents do care about the nutrition of their children and if there really are no significant differences between quantity or nutritional value of food between normal weight and overweight children even when parental monitoring is absent, such as genetics or activity levels. However, the study also suggests that an altruism model is inappropriate, and actually a paternalistic model would be more realistic.

3 Theory

A rational choice framework is briefly explicated in this paper in order to better understand what may be driving the empirical results in the following section. We will use a one-period model that wedges Becker's altruism framework with Philipson's model of adult obesity. Parents are assumed to be perfectly, positively matched so that we need only look at a utility function representing the entire household. Food intakes are represented in terms of total calories consumed within the period. Further, calories directly affect child weight, which also directly affects utility in turn. Weight is assumed to be strictly increasing and concave in calorie intake. There is some optimal child weight, W^* , at which the child's utility function is maximized. Additional weight gained on top of this optimal weight leads to decreasing child utility. This optimal weight may be entirely subjective or may coincide with the weight that is optimal for child health. If we are interested in a peer effect, then W^* could simply be average weight of children in the child's age-sex group. There is one other good representing all other goods consumed aside from food. For now, we will abstract from adult obesity to focus only on that of the child's. Both the parent's and child's utility functions satisfy the usual neoclassical conditions. The altruism parameter, denoted $a(n)$, is a positive but decreasing function of number of children in the household. All children are assumed to have identical preferences and equal weight. Also, we will abstract from fertility decisions and assume the number of children in the family is exogenous. We will assume full additive separability for simplicity. The problem, with first and second order conditions, is as follows:

$$\begin{aligned} \max \quad & u(c_p) + a(n)n[v_c(c_k) + v_x(x) + v_w(W(x))] \\ \text{s.t.} \quad & c_p + nc_k + pn x = M \end{aligned}$$

$$\begin{aligned} \text{FOC:} \quad & (i) \quad u'(c_p) = a(n)v'_c(c_k) \\ & (ii) \quad v'_x(x) + v'_w(W(x))W'(x) = pv'_c(c_k) \end{aligned}$$

$$\begin{aligned} \text{SOC:} \quad & (i) \quad u''(c_p) < 0 \\ & (ii) \quad a(n)v''_c(c_k) < 0 \\ & (iii) \quad v''_x(x) + v''_w(W(x)) \cdot (W'(x))^2 + v'_w(W(x))W''(x) < 0 \end{aligned}$$

Obesity, denoted $W(x)$, is strictly increasing in food consumption x . A sufficient condition for $\text{SOC}(iii)$ to be negative is $v'_w W'' \leq 0$. Consumption other than food is denoted c_i for the i th person. The felicity functions $u(\cdot)$ and $v_i(\cdot)$, $i = c, x$ are continuous, strictly increasing, and strictly concave. Function $v_w(\cdot)$ is continuous, strictly concave, and strictly increasing up to some weight \bar{W} , after which it is strictly decreasing. Number of kids in the household is denoted n . Altruism is $a(n)$, where $a'(n) < 0$. We derive qualitative predictions for obesity with respect to family income, food price, and number of children below:

$$\begin{aligned} \frac{dW(x)}{dM} &= \frac{W'(x)}{\Psi} > 0, \\ \frac{dW(x)}{dn} &= \frac{-(px + \frac{a'(n)v'_c(c_k)}{SOC(i)})W'(x)}{\Psi} < 0, \\ \frac{dW(x)}{dp} &= \frac{-[pnx - (\frac{1}{SOC(i)} + \frac{n}{SOC(ii)})a(n)v'_c(c_k)]W'(x)}{p\Psi} < 0, \end{aligned} \tag{1}$$

where $\Psi \equiv \left(\frac{SOC(ii)}{SOC(i)} + n \right) \frac{SOC(iii)}{SOC(ii)} \frac{1}{p} + pn > 0$.

So, the theory predicts child obesity should be positively related to family income, and negatively related to number of children within the household and food prices. This is not surprising considering the specification of full separability. Under Philipson's model, obesity was not separable from consumption, and this yielded the prediction that obesity and income had an upside-down U shape. In this case, modifying the above model by assuming that obesity and consumption are non-separable in the child's utility function yields no predictions without further assumptions.

Similarly, without further structure on the weight function, $W(\cdot)$, adding a time allocation variable will not permit us to say anything about child obesity. However, suppose we have $W(x, t)$, and the following conditions hold:

$$\frac{\partial^2 W(x, t)}{\partial x^2} = \frac{\partial^2 W(x, t)}{\partial t^2} = \frac{\partial^2 W(x, t)}{\partial t \partial x} \equiv 0$$

The variable t is the fraction of time spent in indolent activities. We will simply assume that weight is a function of $t \in [0, 1]$, because the passive leisure time allocation reduces the active leisure allocation one-for-one. Further, the weight function is assumed to be linear in both food consumption and time spent in low calorie activities. Food consumption and leisure time are assumed independent in producing weight. By adding this structure, we find that the following relationships hold:¹¹

$$\begin{aligned} \frac{dW(x, t)}{dM} &> 0, \\ \frac{dW(x, t)}{dn} &< 0, \\ \frac{dW(x, t)}{dp} &< 0. \end{aligned} \tag{2}$$

Thus, the qualitative relationships still hold under the previous model with the above additional restrictions on BMI. Increases in food consumption due to cheaper real prices or a rise in real

household incomes will be partially offset by a restriction in time spent in passive activities. That is, the larger utility cost associated with the deviation from optimal weight caused by increased food intake is lessened by reallocating time toward more physical activities. Similarly, if we modeled leisure activities as produced by inputs of capital, e.g., video games, and time, we might suppose that, for example, decreases in video game or computer prices would lead to an obesity gain from diverting time away from physical activities that would be partially offset by a reduction in calorie intake. That is, the relative price effect might swamp the income effect for food consumption due to the jump downward in the marginal utility of food from the deviation away from optimal weight caused by the increased time devoted to video games, the computer, etc. Likewise, a reduction in real food prices would lead to a decrease in time spent in passive activities due to the drop in the marginal utility of video gaming from the rise in calorie intake leading to the subsequent increase in weight.

In regard to technological improvements in the medical treatment of the morbidity and mortality associated with obesity, there are two opposing responses that follow. On the one hand, the return to the parent of keeping their kid healthy, and thus closer to the medically optimal BMI, jumps up with the technological innovation. Marginal utility in adulthood improves (via the improvement in morbidity) and the probability that the child will experience that marginal utility improvement also improves (via the decrease in mortality). The desire to smooth utility intertemporally translates into a desire to keep a child healthier. However, the cost of being obese will be reduced with better medical treatment, and this will tend to increase child obesity as a result.

4 Data Sources

The 1997 PSID Child Supplement is a single cross-section of 3,563 children of parents interviewed within the full PSID dataset. All children are between the ages of zero to twelve years old. The Child Supplement collected very detailed data on time allocations among various activities undertaken by the child using time diaries. These diaries cover one weekday and one weekend for all children between the ages of three and twelve. Unfortunately, these data are only one side of the equation when it comes to studying child obesity, because the dataset contains very little information on child nutrition. In addition, since the dataset is only a single cross-section, idiosyncratic fixed effects will be a serious problem. Family fixed effects can still be dealt with, however, by looking at siblings within families.

The Third National Health and Nutrition Examination Survey was conducted over the period from 1988 to 1994. It is composed of a pooled cross-section of 13,944 children ranging up to age seventeen. The benefit of this survey is that it includes diet diary information that can be used to look at food consumption by children in detail. However, the only data the NHANES has on time allocations is a question on time spent watching television on the day previous to the interview. We will also make use of the NHANES I conducted in 1971-75, which has diet diary data as well on 7,104 children.

The NLSY Child Supplement contains panel data on 43,369 child-year observations. The children were surveyed in two year intervals from 1986 to 2000. Despite the fact that the NLSY has no data on caloric intake and limited data on time allocations, the NLSY will still allow us to look at the relationship between obesity, household characteristics, and food prices over time. Further,

the dataset will allow us to deal with individual fixed effects.

Finally, the Cycle II National Health Examination Survey is a cross-section of 7,119 children between the ages six to eleven years old. Because it was conducted over the period 1963 to 1965, the NHES will give us a baseline to understand what has happened to child obesity over the last few decades.

5 Empirical Results: Explaining the Trend

5.1 Income and Hours Worked

We will first test the hypothesis that changes in income and hours worked by each parent are behind the growth in child obesity. Our theory suggests, for instance, that child obesity should be positively related to family income. Also, both parents working would be expected to negatively impact the possible time allocations a child could choose, such as, for example, going to a distant park to play. This would be especially important to obesity during school vacations or if there are no opportunities for physical activity during school time.¹³

As far as explaining the trend in child obesity, neither labor nor non-labor income are found to be important. This can be garnered from glancing at Table 5, which uses PSID and NLSY data. Even when not statistically insignificant, the income elasticities for either spouse are simply too small to be relevant. For example, from Column (1) of Table 5, a 10% increase in the mother's wages should result in children that are only 0.2% thinner for the average family. Of course, this would go counter to the trend in child obesity as well. Similarly, hours worked by either parent are not important. Children surveyed under the PSID who have mothers working an additional

10 hours per week, for instance, are expected to be only about 0.01% heavier. Using NLSY and correcting for fixed effects in Column (3) of Table 5 does not improve the economic significance of either income or working hours as far as explaining the trend in child obesity is concerned.

In Table 6, we run BMI on dummy variables using PSID and NLSY data, respectively. The income dummy variables are defined according to decile with the highest decile being excluded. A dummy variable was included for zero labor income for both husbands and wives.¹⁴ The relationship of the father's labor income with child BMI basically has a flat profile, so movement between most of the income deciles will not change child obesity by very much. As far as the mother's labor income is concerned, the PSID elasticities imply that lower wage income mothers tend to have heavier kids. Given labor hours worked, the wage gains made by women over the years would work against increasing child obesity. Only with the dummy variable specification in Table 6 using PSID data do we get significant empirical results for hours worked, though not with the NLSY data. For the former dataset, we find is that obesity is strongly and positively associated with number of hours worked by mothers and negatively associated with hours worked by fathers in a week. The problem with attributing changes in hours worked with the increase in child obesity, however, is that these changes would have had to occur relatively recently. In fact, the distribution of hours worked for either parent has hardly changed between the 1986 NLSY and 1997 PSID surveys.

5.2 Education of Parents

We know that educational levels have been increasing within the U.S. over the time period we are interested in, so we also want to look at parental years of education in order to see if better educated parents have superior understanding and information concerning the health impacts of

obesity on children. There should be a negative relationship between a child's BMI and number of years of education undertaken by his parents. The elasticities we estimate in Table 5 generally bear this out. However, our estimates show that the education level of either parent is generally of small significance in determining child BMI. An additional year of education by the average mother, for example, only results in children that are 0.6% thinner (see column (1) of Table 5). The elasticity with respect to the education level of the husband is of an even smaller magnitude. The dummy variable specifications in Table 6 also generally bear out negative relationship between either parent's education level and their child's BMI.

Considering that both fathers and mothers are more educated than they were in the 1960s, these results will work against the upward trend in childhood obesity, though not very strongly. There has been an increase in parental education by about two and a half years each from the Cycle 2 sample in the 1960s to the NLSY and PSID samples in the 1990s. The quadratic specification implies no change in child BMI for PSID data, and about a one percent decrease using NLSY data (refer to Table 5). As can be gathered from Table 15, there has been, not surprisingly, a large shift away from having less than a high school education towards having a college education. However, the predicted change in BMI with these educational shifts is still relatively small. Specifically, the dummy specifications indicate changes in education reduced BMI by 1.5% and 2% with the PSID and NLSY elasticities, respectively. Finally, though, we must note that changes in parental education between the 1980s and 1990s are much too small to be a significant factor in affecting trends in childhood obesity.

5.3 Medical Technological Development

We can use the panel format of the NLSY to see if advancements in the treatment of certain medical problems have a measurable impact upon child obesity. Because there are only have eight two-year periods in the data, though, we are limited in the extent to which we can tie technical change in health technology with child obesity. Be that as it may, this paper will look at dramatic changes that occurred in the pharmacological treatment of asthma. There were no new significant developments in drug treatment for asthma for twenty years until a new class of drugs, called leukotriene receptor antagonists, was introduced into the market beginning in 1996. Asthma is a disease in which periodic inflammations in the respiratory tract causes the airway passage to become constricted, leaving the asthma sufferer gasping for breath. There is no way to predict when an asthma attack will occur. Until 1996, the response to an asthma attack would generally be the use of fast acting, short term bronchodilators that would be introduced into the airways via an inhaler. This would, as the name implies, dilate the airways, and thus give quick relief from the attack. The big shortcoming of treatment was that bronchodilators responded effectively to asthma attacks but there was no treatment to reduce the frequency of attacks until leukotriene receptor antagonists came on the scene. The first of these, called Accolate¹⁵, was introduced in September of 1996. Now, by taking a pill regularly, an asthma sufferer could reduce both the number of asthma attacks and their severity, though one still needed to carry a bronchodilator in case an attack did occur. The vehicle through which this technology would affect child obesity is by allowing a reallocation of time across activities.¹⁶ For example, because asthma could be triggered by dust or pollen, this would affect the amount of time spent in outdoor play by the child.

To see if the introduction of these new drug treatments had a discernible impact upon obesity after 1996, we will interact the year dummies with indicator variables denoting whether the child had asthma or not. The results are displayed in Table 12. As we can see, the evidence is weak. Statistically, only the interaction terms for 1998 and 1996 are significant. We should expect that, for the years 1998 and 2000, the difference in BMI between asthma sufferers and other children will narrow, so the coefficient on the interaction terms should be negative. However, in 1998, children with asthma were actually four percent heavier than non-asthma sufferers. The difference in BMI between the two groups of children was only smaller in the year 2000, but even this elasticity was a statistically insignificant -0.2%.

Now, the NLSY does have data on the number of hours of TV watched every week by children from 1990 to 2000. Presumably, children afflicted with asthma would tend to stay indoors and watch more TV than children without asthma problems. Table 13 presents regression results with hours watched of TV as the dependent variable, year dummies, year dummies interacted with whether the child has asthma or not, and other characteristics. We should expect to find that, first, children with asthma do watch more TV, and, for the years 1998 and 2000, the coefficient on the interactive terms will be negative, so that the amount of TV watched by asthma sufferers converges to the amount watched by other kids. The first item to note is the lack of statistical significance in television watching between the two groups of children. The best estimate in terms of statistical significance is for the 1998 interaction term with a p-value of 8%. Putting this issue aside for the moment, there is mixed support for the hypotheses that, first, asthma sufferers watch more television than non-asthma sufferers, and, second, the innovation in asthma medication reduced the amount of television watched. In every year except 1994 and 1998, children with asthma tended

to watch at least an hour more of television weekly in the years 1990, 1992, and 2000, and more than three hours more per week in 1996. Also, television watching by asthma sufferers dropped substantially in 1998. One period before, they had been watching a little over three hours of TV more every week than other children, but in 1998, asthma sufferers were watching 3 hours of TV *less* than other kids. However, problems with the estimation are not only the general lack of statistically significant differences between asthma and non-asthma sufferers, but in the year 2000, the estimates show television watching by asthma sufferers jumped right back up to an hour and a half more TV than other children's TV consumption.

In summary, changes in medical technology over the last couple decades may be important in explaining obesity trends, but the data, limited though it is, show lackluster support for this. Looking at the medical treatment of children affected with asthma was ideal in that the innovation in treatment was relatively discrete in time and important in its impact on quality of life. However, either parents are inelastic in changing their child's consumption allocation in response to medical technological progress or we just need more data.

5.4 Time Allocations

The PSID data is the only dataset that has detailed activity data from time diaries, but it only has data on a family's total food expenditures, while the NHES data does have detailed diet data but virtually nothing on a child's daily activities. The NLSY has no useful food intake data and very limited information on daily activities. Differencing across siblings to eliminate family fixed effects such as genes, we can estimate how a child's BMI relates to his various time allocations, using instrumental variables to deal with endogeneity. Getting these estimates would allow us to

see to what extent changes in how children spend their time explains their trends in obesity.

Activities were classified into three general groups: active, passive, and other. These classifications are loosely based on the PSID's own classification of activities. The active variable includes such activities as sports, walking, running, dancing, etc. Examples of passive activities include computer-related activities, television viewing, listening to the radio, playing games, and conversing. All other activities that were not particularly passive or active were classified as Other. The appendix details what activities were included under Active and Passive. I differenced across siblings and produced the estimates in Table 7.

Look at Columns (1) and (2) of Table 7, first. Column (2) further controls for sex as well as age. These regressions show that every additional hour per week spent in passive activities such as television watching will tend to increase child obesity by about 0.2%. Now we can consider the hypothesis that the blame for child obesity lies on TV and video games. Between the 1997 PSID and the 1963-65 NHES Cycle 2, child BMI increased by 9.4%. The average amount of time spent on TV rose from 13.7 to 18.8 hours per week over this period, also. Using these figures and our estimates from columns (1) and (2), we see that TV should have increased child BMI by about one percent. However, NLSY data actually shows that viewership doubled from the 1965 to 1990, rising to 28.3 hours weekly among kids in the age group considered in the NHES. BMI was already shown to be flat from the 1960s to 1986, but from 1986 to 1990, average BMI grew 4.6%. Using the calculated elasticities for time allocations, the predicted increase in BMI due to television viewership is 3.5%, explaining three quarters of the total increase. The problem is that, after 1990, hours watched does not do a good job explaining the trend in child BMI, especially after 1996 when hours of TV watched actually started to decrease (see Figure 3). Over the entire

decade of the '90s, for example, obesity grew by nearly eight percent, but TV viewership had fallen by six hours per week.

As for video games, in 1997, kids who used their video game systems during the survey spent an average of 6.5 hours per week playing them. However, only 16% of the sample either did not have video game systems or did not use them over the period covered by the time diary. Even doubling this proportion would only translate into a negligible increase in BMI of 0.4%. Similarly, those kids who sat in front of their computers during the period spent an average of 4.6 hours weekly, but only eleven percent of the sample did so. Fortunately, the NLSY does have information on whether a child's household owns a computer or not from the period 1994 to 2000(see Table 16). Ownership of computers skyrocketed from one-third to four-fifths of all households over this period. Using these estimates and interpolating gives us an ownership rate of 57% for 1997. If we use these ownership percentages and the PSID estimate for time spent by kids in front of computers, we find that, out of the 7.8% increase in BMI that occurred from 1994 to 2000 according to the NLSY, the change in computer ownership should explain only a little over six percent of that increase. Even lumping in video game systems with computer ownership will only explain about 8.5% of BMI growth.

We get much larger elasticities when we instrument for the passive and other time use variables, though. Columns (3) and (4) of Table 7 are 2SLS regressions. For instruments, we will use variables on whether doctor diagnosed conditions of asthma, speech, hearing, or sight impairments exist. These can be expected to affect time allocations between active and passive activities but not to affect childhood BMI directly.¹⁷ We find that an additional hour of passive activity results in about a 12% increase in child obesity, which is very large. For example, this means that the increase in TV watching since the 1960s should have raised average body weights by over fifty percent as

opposed to less than a percent using the non-2SLS elasticities. The instrumented elasticities are robust when different subsets of the above instruments are used, though.

Table 8 presents elasticities after dividing the sample by sex. Restricting the sample to only brothers results in the uninstrumented elasticities being very small and statistically insignificant, while instrumenting actually results in large negative elasticities. For sisters only, both the uninstrumented and instrumented elasticities are statistically significant and positive. We should place greater reliance upon the elasticities estimated with the full sample due to both the small sample sizes involved and to the fact that differences in sex are already controlled for in the estimation.

Thus, the empirical results suggest that changes in time allocations may be very important in explaining child obesity. The problem, though, is discovering exactly what if any particular change in time usage by kids explain the recent trend in child BMI. We need more data to go further at this point.

5.5 Changes in Household Composition

Another possible culprit lying behind obesity trends within the U.S. may lie in the dramatic shifts in family structure since the 1960s. For example, according to the NHES Cycle 2, only 10% of American children were living in single parent households in the early 1960s. By the time of the NLSY survey in 2000, nearly 30% of all kids would be found in single mother households. Controlling for income, this might affect child obesity simply through the feasible set of time allocations available to the child that results when there is only one working parent, as has already been discussed. Another avenue for family composition to affect child BMI is through changes in number of siblings within a household. Theoretically, parents should transfer correspondingly less resources per child

for each additional kid in the family due to decreasing altruism. The other way number of siblings can be important is through a child's preferences over time allocations. That is, simply, the more siblings a child has, the greater the number of possible playmates there are for activities that are team-based, which usually tend to be physical. This case also would imply a negative relationship between number of siblings and a child's BMI. Changes in the racial composition of the population will be an important consideration, also, because of cultural differences in food consumption and activities.

The general problem with explaining the BMI trend with compositional changes is that these changes would have had to occur quite recently. For instance, the percentage of kids with single moms in the year 2000 is virtually unchanged from that in 1986 according to the NLSY. Further, we know that single parenthood has been an increasing problem since at least the 1970s, but there was no corresponding change in childhood obesity. Yet another strike against this hypothesis is the relatively small elasticities involved when we look at marital status variables or number of adults listed in the household. In Table 14, we add a dummy variable for whether the mother is single, divorced, separated, or widowed, and other variables controlling for number of adults living within the household and mothers current age. None of the variables are important economically. For instance, we estimate that children with single mothers should be about one percent thinner correcting for fixed effects. Of course, the sign goes in the opposite direction than that needed for this hypothesis.

Also, we consistently find a negative relationship between the number of siblings a child has and his or her BMI, but this demographic change is much too small to be helpful in explaining child obesity trends. For example, using NLSY and PSID data, adding a third child to the household is

expected to reduce the BMI of his or her siblings generally by about 1% for the average family (refer to Table 5), but, even though the average number of children in a household has decreased from 3.8 in the 1960s to 2.3 by 1997, this only means an overall rise in BMI of about 1.5%. However, virtually the entire increase in average BMI among children has occurred since the 1980s when there has been hardly any change at all in family size over this short period.

What do shifts in racial composition mean for child obesity? The short answer is not much. Although both Hispanic and Black children tend to be relatively heavier, the elasticities are too small. Fourteen percent of children surveyed in the NHES were Black, while the PSID and NLSY give percentages of 17 and 16 percent, respectively. Thus, the slight increase in the proportion of kids that are Black resulted in just a marginal rise in childhood BMI of about a fifth of one percent based on the six percent elasticity estimated from the PSID dataset (see Table 5). For Hispanic children, the NHES did not separate them out, so we have to turn to Census data. The proportion of Hispanics climbed from about 4.5% in 1970 to 12.5% in 2000. Despite this meteoric rise, the elasticity of four percent estimated from PSID data means that this racial shift only raised child obesity by about one-third of one percent. The corresponding race elasticities estimated with NLSY data were even less than the PSID elasticities (refer to Table 5).

5.6 Calorie Consumption & the Real Price of Calories

The NHANES gathered diet diary information from children aged from 2 months up to 16 years old, and thus we can use these data to say something about the other determinant of a child's BMI: calories consumed. The major drawback to the data is that the diet diary contains information for only a single day of food consumption. NHANES did ask if the respondent's diet that day was

'typical' or not, so we will restrict the sample to those who answered this affirmatively. Also, we will follow the same empirical procedure used with the time diaries in the PSID, i.e., difference across siblings to eliminate family effects.

Table 9 Columns (1) and (2) present our regression estimates of logged child BMI on logged calories and other regressors after differencing for the full sample. The estimate is actually negative though very small, giving an elasticity of -0.03. Running the specification separately for each sex also results in economically small but negative elasticities. We have to instrument, however, and for this we need instruments that correlate with calorie consumption but do not directly determine childhood BMI. Two instruments will be used. The first one is a subjective measure by the parent as to the general quality of the child's natural teeth. There are five ratings ranging from excellent to poor. The second instrument used was whether or not the child had canker sores within the last year. Canker sores occur within the mouth and can make eating certain foods an uncomfortable experience, especially food items that are particularly salty, like potato chips, or acidic, like orange juice. These sores usually last from eight to ten days. Approximately 20% of the sample has had canker or mouth sores within the last year, which is in line with the rate of incidence for the population as a whole. Obviously, this would negatively affect the consumption of salty junk foods which tend to also be high in calories.

The third column of Table 9 presents 2SLS estimates.¹⁸ Looking at the 2SLS estimates for the full sample in Table 9 Columns (3) and (4), we see that a 10% increase in calories should result in a jump in childhood obesity from about five to six percent. Running the regression for each sex separately now results in positive elasticities of BMI with calorie consumption but they are much smaller than for the full sample (see Table 10).¹⁹

To see what this means to the BMI trend, we have to go to U.S. Department of Agriculture data on total calories produced per capita in the U.S..²⁰ Figure 4 charts total calories available for consumption per capita, which is based on the total food supply available for consumption in the U.S.. Interestingly, after remaining flat from 1970 to 1983, calories per capita started a climb upward as can be seen in Figure 4. So, this certainly fits in with the general time trend in child obesity. Calories per capita grew 11.4% from 1986 to 2000. This figure and the 2SLS elasticity of 0.585 suggest that child BMI should have risen by nearly seven percent. Now, using the BMI data from NLSY, average childhood BMI rose 15.6% from 1986 to 2000. Thus, changes in per capita calories can explain about three quarters of the increase in average BMI as long as the relative share of children's calorie consumption is a constant proportion of total calories per capita.

So, looking at aggregate food supplies only seems to explain a big part of the trend in childhood obesity, but we also have direct, micro-level consumption data from food diary information contained in the 1971-75 NHANES I and 1976-80 NHANES II datasets. Figure 6 plots mean calorie consumption for all kids aged less than eighteen years old along with their mean BMI from NHANES I, II, and III. Interestingly, individual-level caloric consumption of children was on a downward trend throughout the 1970s, and, overall, the average number of calories consumed in the period 1971-74, two thousand calories, is about 120 calories more than the average amount consumed in the 1988-94 NHANES.²¹ In addition, the overall trend in calorie consumption is negative while that of BMI tends to be positive.

Why the discrepancy between aggregate calories per capita and calorie consumption garnered from food diaries? First, there is substantial underreporting of food intake when people are given diaries to fill out. Lichtman, et al.(1992) estimate underreporting of actual food intake by nearly

50%, although they only included a handful of people in their study to derive this figure. If the degree of underreporting is stable across time, then underreporting should not qualitatively affect the overall trend in calories consumed. Second, the calories per capita data based on the U.S. food supply do not adjust for spoilage. If the proportion of food wasted out of total production is convex in the volume of food produced, this would mean actual calories consumed per capita would grow at a perhaps significantly slower rate than calories per capita unadjusted for wastage. An example of this might occur if processed foods are relatively more wasteful and account for a greater share of the increase in food output. Wastage would not matter in explaining the trend in child obesity if it were simply a constant fraction of the total food stock, but if different ways of processing food have different degrees of spoilage, which they do, and the shares of each process vary over time, which they did²², then assuming a constant rate of spoilage is not very reasonable. Another possibility is that most of the increase in food production is going to adults. This does not mean that parents are becoming very selfish in allocating food within their families. However, due to declining family size and lower rates of morbidity and mortality among adults, the ratio of total adult consumption to that of children will have increased, accounting for perhaps a significant proportion of the increase in total calories per capita. Yet another possibility is that total daily calories consumed by a child have not increased. Parents might plausibly be more concerned about the nutritional composition of the food intake of their kids than of themselves. In this case, parents would balance junk food consumption by their children with healthy, low-calorie food items more successfully than parents would do with their own consumption. So, again, adults would consume a large chunk of the additional calories produced in the U.S..

Now, towards the end of finding the link between obesity and food prices, we will use the NLSY

panel, but bear in mind that any empirical results must be tempered by the fact that we only have eight two-year periods with which to estimate the relationship. Two measures of calorie cost will be used. The first will be a *Laspeyres* food index provided by the Bureau of Labor Statistics. We can think of this index as prices of calories weighted by the share of calories provided by a food item out of total caloric intake summed over all food items consumed by a household. That is,

$$CPI_0^{food} \equiv \frac{\sum_f p_{ft} x_{fo}}{\sum_f p_{f0} x_{fo}} = \frac{\sum_f \frac{p_{ft}}{c_f} c_f x_{fo}}{\sum_f \frac{p_{f0}}{c_f} c_f x_{fo}} = \frac{\sum_f \frac{p_{ft}}{c_f} \frac{c_f x_{fo}}{\sum_f c_f x_{fo}}}{\sum_f \frac{p_{f0}}{c_f} \frac{c_f x_{fo}}{\sum_f c_f x_{fo}}} = \frac{\sum_f \pi_{ft} s_{fo}}{\sum_f \pi_{f0} s_{fo}},$$

where π_{ft} is the price per calorie of food f at time t , and s_{fo} is the share of calories out of total calories consumed provided by food f at the baseline year. So, the index is equivalent to the average price of calories consumed by the average household relative to the average price of calories in a base year. By use of the CPI for all consumption goods, we can see how the relative price changes for calories affected childhood obesity over time. For the other food price measure, we will use the actual price data for 133 food items included in the CPI food index, and divide each price by the number of calories provided by each food item in order to calculate the simple mean price of calories over time. The data on calories comes from the USDA National Nutrient Database.²³

Figure 5 presents the real rates of change for both price series with the rate of change of childhood BMI from the NLSY. The CPI price index for all goods except food and beverages was used to calculate the relative rates of change.²⁴ If changes in the price of calories are really expected to affect obesity, we might expect childhood obesity growth rates to be inversely related to fluctuations in price growth rates. However, according to the graph, if anything, BMI is procyclical with food prices, at least until after 1998. This is true even if we suppose that BMI responds to price changes

with a lag. For example, from 1990 to 1992, the real price growth fell from approximately zero to -2.5% to -5% annually, depending on the measure used. Child obesity, though, substantially decelerated in growth from almost 5% to negative 4%. Also, the following two year period, 1992 to 1994, saw relatively little change in the growth rate of prices. Despite this, BMI growth rates rose substantially in the subsequent two years. Finally, the Laspeyres food price index actually did not change very much relative to other prices throughout the entire period, and mean food prices were in decline in real terms for almost every year. The fact that the average BMI for kids was growing in all periods except the early 1990s also lends one to be skeptical of the view that obesity is driven by relatively cheap calories. Further, the food price index seems quite inelastic if one believes substantial technological progress in food production took place in the 1980s and 1990s, which it must have if we are to use it as an explanation of the recent runup in child obesity. Of course, the real mean price of calories did shrink considerably over time, but kids were getting heavy at nearly the same rate as when average prices were shrinking at 15% per year in 1986 as when they were growing at 5% in 1998.

Finally, Column (2) of Table 11 includes the full set of regressors along with a logged CPI real food price index and its square. We find that child obesity and real food prices have the expected negative relationship with an elasticity of about -0.2. However, the elasticity is not statistically significant. Column (3) presents 2SLS estimates using the mean yearly BMI for all the mothers in the dataset to instrument for prices. Column (4) instruments and also corrects for fixed effects. The latter two elasticities are negative and highly statistically significant, but are relatively large with a one percent increase in food prices predicting a 5% decrease in BMI from (3) and a 13% decrease from (4). For comparison, over the period 1986 to 2000, food prices declined by 1.7%

in real terms, while NLSY child BMI increased by 15.6%. The uninstrumented elasticities would mean an increase in obesity of only a third of a percent. The non-differenced, IV estimate predicts a nearly 8% increase, while the differenced IV estimate predicts a 22% increase. If we compare average real food prices for the years 1967-69 with those for 1998-2000, we find that food prices have declined by 5.3% in real terms with corresponding predictions of 1%, 24%, and 70% increases in obesity, respectively. The first thing to note is that, because the demand for food is very inelastic, the uninstrumented elasticities are probably more credible than the instrumented ones due to the large magnitude of the latter. Also, two-thirds of the decrease in real food prices occurred before 1986, and yet, if we recall Table 4 and the Introduction, nearly all of the increase in childhood obesity occurred after 1986. So, in this respect, real food prices do not seem relevant in explaining trends in child BMI. Furthermore, despite the decrease in real food prices, there was little change in childhood obesity between the 1960s and 1980s.

The NHANES datasets also provide information on both when and where meals recorded in the food diaries were taken. Using these data, we can see if there were increases in the frequencies of snacking or eating out by kids, because this is one of the contentions of people who blame junk food and fast food restaurants for child obesity. In the 1971-75 NHANES I, 23% of all meals eaten by children were snacks. Nearly ninety percent of all children had at least one snack during the day, and snack consumption accounted for 26% of their total calorie intake. Nearly twenty years later, the snacking picture seems to have improved significantly. The 1988-94 NHANES III shows that 18% of all meals eaten by the surveyed children were snacks. Also, over a third of children, 36%, did not even have a snack break. Of those who did, snacking accounted for nearly the same percentage of their total caloric intake. What is also interesting is that the snacking is slightly

healthier. Kids who snack in NHANES III are actually eating more snacks by weight, about six percent more, than their peers in NHANES I.²⁵ However, in terms of calories, the snacks the former kids are eating only contain two percent more calories.

In regard to restaurants, including fast food, 4.3% of all meals eaten by kids in the early 1970s were in restaurants. Eighty six percent of children did not eat out for any of their meals. Of those who did eat out, they received a third of their total daily calorie intake from restaurant meals. Not much has changed twenty years later. Now, 5.2% of all children's meals are in restaurants, but nearly 89% of kids did not go to restaurants for any of their meals. Now, of those kids that did go out to eat, their meals were bigger than their peers in the early 1970s. Restaurant meals are sixteen percent heavier in weight now than at the time of NHANES I. Kids eating out now derive 39% of their total daily calorie intake from their restaurant meals. However, as in the case of snacking, restaurant meals are a little healthier in terms of having fewer calories per gram consumed. Specifically, restaurant food calories per gram went from 1.61 to 1.42 between the two periods observed. Further, restricting each NHANES sample to only those who ate in restaurants, children in the NHANES III sample compensate much more vigorously for their larger restaurant meals by cutting down their snacking and meals at home. Children now end up consuming nearly 200 fewer calories in a day than their peers in NHANES I despite the larger restaurant meals.

Other interesting observations are that it is true that fast food meals are more calorie intensive than either home cooking or restaurants as a group. Compared to 1.51 calories per gram of food consumed in fast food joints, home cooking only has 1.19 calories per gram and, as already noted, restaurants altogether have 1.42 calories per gram. Be that as it may, however, fast food only represents about two percent of all meals consumed. Similar to what we found for restaurant meals,

parents reduce their kids' calorie intake from snacking and home cooking to fully compensate for eating fast food, so that total calories consumed by children who eat fast food is only 59 calories more than those kids in the early 1970s who ate at home. Speaking of home cooking, it is also lower calorie now. Home cooked meals were both larger and heavier in calories in the 1970s to the tune of 11% larger in weight and 15% heavier in calories than home cooked meals in the late 1980s and early 1990s.²⁶

We can further refine our scope to specific, junk food items such as sodas. When we do so, however, the snacking picture above repeats itself. Kids surveyed in NHANES I who chose to drink soda had about one, twelve ounce can per day. By the late 1980s, soda drinkers were downing an average of fifteen ounces of soda every day, translating into an extra twenty calories daily. Be that as it may, actually fewer kids are drinking sodas with their meals. Whereas fifty-six percent of children in the early '70s had at least one can of soda during the day, now this has dropped to fifty percent. Furthermore, looking at total daily calories consumed by soda drinkers, we find that, even though kids are drinking more soda by the late 1980s, their total caloric intake is actually 90 calories less than their peers in the early 1970s.²⁷ Another fact is that on a per gram basis, soda products on the whole have fewer calories than in the past. Specifically, twelve ounces of soda had on average 141 calories in NHANES III versus 156 calories in I. So, it does seem that parents are forcing diet sodas, not widely available in the early 1970s, onto their children.

Similarly, although kids surveyed in the NHANES III eat an extra twenty calories daily in potato or corn chips, the total daily calorie intake of these children is still smaller now by ninety calories than it was for their peers in NHANES I. Also like soda, chips on a per ounce basis are a little healthier, having ten fewer calories per ounce than in the early '70s.²⁸

Another junk food example is french fries. In this case, the average serving size is smaller now than before. Roughly speaking, french fry servings have shrunk from a medium size of fries at a typical fast food place to a small size.

Pizza consumption bucks the pattern a bit. Less than six percent of children ate pizza during their survey in NHANES I. Nearly two decades later, this fraction tripled in size, so that, by the late 1980s and early 1990s, one out of five kids were having pizza during the day. Further, pizza servings now have a third more calories than previously, not only because of larger serving sizes but because they are simply more calorie heavy. Compared to 2.5 calories per gram of pizza in the early 1970s, there were now 2.8 calories per gram by the late '80s. Once again, though, when we look at total daily calorie intake figures for pizza eaters between the two periods, kids who eat pizza still manage to cut their total calorie consumption by nearly nine percent below that of children surveyed in the earlier period.²⁹

Finally, for one last example, we look at french fry consumption. While it is true that more kids are eating french fries than in the past, the incidence rate has only increased six and a half percent to twenty percent total. Further, french fry eaters reduced their total intake both in terms of weight and calories between the two periods. Children who eat french fries have reduced their calorie intake from fries by almost fifty calories, or by seventeen percent of the original average calorie intake in the early 1970s. Like the other junk foods we have looked at, when we look at total daily caloric intake by french fry eaters between the two periods studied we find a sizable decrease. Whereas the average french fry eater had total calorie intake of 2,275 per day in NHANES I, this had dropped to 2,064 calories per day by the NHANES III survey.

Thus, what can we conclude from all this about the theory that calorie intake has driven child

obesity in America? Despite the startling rise in total calories consumed per capita that we see from Figure 4, both the micro data on calorie intake and the limited price of calorie information we have do not support this explanation. Moreover, there is evidence that parents do seem to be paying more attention than in the past to what their kids are eating. Children may stuff themselves on calorie heavy fast food, for example, but parents will then cut their kids' food intake from other sources such as snacks to compensate. This seems to be the case regardless of whether we look at soda drinking or pizza consumption. Furthermore, we noted above that some of the food categories and items were healthier now in terms of having fewer calories on a per weight basis than in the past. Thus, while it is true that kids are eating more junk food than in the early 1970s, parents seem to be doing a good job of substituting lower calorie foods into their children's consumption and in compensating for the junk food consumption by cutting intakes of other foods so that total daily calorie intake ends up being actually less than in years past.

6 Conclusion

The recentness of the upward climb in child obesity eliminates many possible explanations, such as parental education, hours worked by parents, family income, and family compositional changes, for instance. We also try to test the hypothesis that child obesity responds to medical technology by looking at the pharmacological innovation in asthma treatment, but nothing definitive was found and further data is needed on this point. We find time allocations to be potentially important determinants of child obesity. However, television viewership does not explain BMI trends. Changes in computer ownership was particularly rapid in the '90s but we need more data on computer and

video game usage over time. Total calories per capita consumed in the US tracks fairly closely with the trend in child BMI, and empirically explains most of the increase in obesity. Unfortunately, the evidence that changes in real food prices lies behind increases in food consumption is weak. Furthermore, direct, micro-level evidence from food diaries contradicts the hypothesis that more calories are being consumed by children overall or that so-called junk foods are behind the child obesity epidemic.

Further research avenues lie in finding better measurements of the relationship between medical innovation and obesity. Also, temporal data on time usage by children is needed in order to see if computer or video game usage is behind the obesity trend.

7 Notes

1. VanItallie, TB(1996).
2. Bray, G.A.(December 1996).
3. Defined by the National Center for Health Statistics(NCHS) as BMI ≥ 27.8 for men and 27.3 ≥ 27.3 for women, corresponding to the 85th percentile.
4. Johnston (1985).
5. Serdula, et al.(1993).
6. Refer to Tables 2 and 3.
7. In order to make the datasets comparable to one another, we will only compare children aged 6 to 11. This is the narrowest age range studied among the three datasets, corresponding to

the age range in the NHES Cycle II. All data are weighted by their sample weights.

8. Children were considered to be low socioeconomic status if neither parent was working, they received reduced price or free school lunches, or the household received welfare assistance(808).
9. Obesity was defined as having a triceps skinfold measurement that is at least in the 85th percentile(808).
10. Overweight defined as having a BMI above the 85th percentile.
11. See Appendix A for details.
12. See Dietz and Bellizzi(1999) for an overview of how BMI compares with other measures of obesity.
13. It has been suggested that hours worked, principally by the mother, would lead to substitution of homemade meals with ready-to-eat meals. The implication is that prepared foods are more calorie intensive than home prepared meals. However, this need not be the case. First, there is a wide range of prepared foods in terms of contained calories and nutrients, so parents may choose if they wanted relatively healthy meals. Even relying on fast food does not mean parents only have Big Macs to feed to their kids. Secondly, what matters is total calories consumed during a period. Parents can control this by restricting portion size or substituting some portion of calorie laden foods with low calorie items. Controlling for hours worked, the labor income variable will allow us to see how changes in the parents' value of time affects child BMI. This basically tests whether each parent values their kids in the same way. For example, if the mother is more altruistic to her children than their father, the elasticity between obesity

and the mother's labor income may be greater than the elasticity with respect to the father's labor income.

14. Ten percent of husbands and forty percent of mothers had no labor income for 1996.
15. Accolate was only approved for children twelve years or older, and it was not until February 1998 until another drug was developed, Singulair, which could be prescribed for children down to six years of age.
16. There is no evidence that appetite or metabolism is significantly affected by the drugs.
17. First stage results of 2SLS are given in Tables 8A and 8B.
18. First stage results are presented in Table 9A.
19. For a comparison of these elasticities with those estimated in the time allocation section, see Appendix C.
20. Refer to Putnam, Allshouse, and Kantor(2002) published by the Economic Research Service of the USDA.
21. The median values are 1,863 and 1,703 calories for 1971-74 and 1988-94, respectively.
22. E.g., with the trend of food consumption moving away from minimally processed to heavily processed foods.
23. USDA NND for Standard Reference, Release 16 (July 2003).
24. Real food prices come from the CPI for urban consumers. Base period is 1982-84.

25. Children who snacked in NHANES I consumed an average 470 grams (about one pound) of snacks daily compared with their peers in NHANES III who ate 498 grams (almost 1.1 pounds).
26. Calories per gram of home-cooked meals consumed dropped from 1.23 to 1.19.
27. Soda drinkers consumed 2,043 total daily calories in NHANES III versus 2,133 calories in NHANES I.
28. An ounce of chips had on average 178 calories in NHANES I.
29. Average total calorie intake of pizza consumers in NHANES I was 2250 compared to 2058 for those surveyed in NHANES III.

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8 Appendix A

Restating the problem, FOC's, and SOC's with the new variable t :

$$\max \quad u(c_p) + a(n)n[v_c(c_k) + v_x(x) + v_t(t) + v_w(W(x, t))]$$

$$s.t. \quad c_p + nc_k + pnx = M$$

$$FOC : \quad (i) \quad u'(c_p) = a(n)v'_c(c_k)$$

$$(ii) \quad v'_x(x) + v'_w(W(x, t))W_1(x, t) = pv'_c(c_k)$$

$$(iii) \quad v'_t(t) + v'_w(W(x, t))W_2(x, t) = 0$$

$$SOC : \quad (i) \quad u''(c_p) < 0$$

$$(ii) \quad a(n)v''_c(c_k) < 0$$

$$(iii) \quad v''_x(x) + v''_w(W(x, t)) \cdot (W_1(x, t))^2 < 0$$

$$(iv) \quad v''_t(t) + v''_w(W(x, t)) \cdot (W_2(x, t))^2 < 0$$

Because the corner solution $t = 1$ is straightforward, we will just consider the interior solution $t < 1$. In this case, differentiating the FOCs and BC with respect to M , n , and p , we eventually solve for:

$$\frac{dx}{dM} = \frac{1}{\Psi} > 0,$$

$$\frac{dt}{dM} = \frac{-a(v_w''W_tW_x)}{SOC(iv)} \frac{dx}{dM} < 0,$$

$$\Rightarrow \frac{dW(x,t)}{dM} = W_x \frac{dx}{dM} + W_t \frac{dt}{dM} = \left[1 - \frac{av_w''W_t^2}{SOC(iv)}\right] W_x \frac{dx}{dM} > 0,$$

$$\text{by } 1 > \frac{av_w''W_t^2}{SOC(iv)} \Leftrightarrow v_t'' < 0$$

$$\frac{dx}{dp} = \frac{-pnx + av_k' \left(\frac{1}{SOC(i)} + \frac{n}{SOC(ii)} \right)}{p\Psi} < 0,$$

$$\frac{dt}{dp} > 0, \text{ by above}$$

(3)

$$\frac{dW(x,t)}{dp} < 0, \text{ by above}$$

$$\frac{dx}{dn} = \frac{-px - c_k - \frac{a'v_k'}{SOC(i)}}{\Psi} < 0,$$

$$\frac{dt}{dp} > 0,$$

$$\frac{dW(x,t)}{dp} < 0,$$

$$\text{where } \Psi \equiv \left(\frac{1}{SOC(i)} + \frac{n}{SOC(ii)} \right) \left(SOC(iii) \cdot SOC(iv) - (a(n)v_w''W_tW_x)^2 \right) \frac{1}{pSOC(iv)} + pn > 0$$

$$\text{and } SOC(iii) \cdot SOC(iv) - (a(n)v_w''W_tW_x)^2 = v_x''SOC(iv) + v_w''v_t''(W_x)^2 > 0.$$

9 Appendix B

HOME COMPUTER RELATED ACTIVITIES

Lessons in computers; playing computer games; other recreational computer activities, "surfing the net"; using the computer for homework, studying, research, reading related to classes or profession; computer communication; work for pay at home using the computer; financial services; shopping; media, reading newspaper, stock quotes, weather reports; library functions; computer work, getting computer programs to work, reading the manual, repairing computer, setting up computer; other.

ELECTRONIC VIDEO GAMES

Nintendo, Sony, Game Boy, Sega

OTHER GAMES

Pretend, dressup; playing card games; playing board games; playing social games; puzzles/word or educational games; played with toys; unspecified play outdoors; unspecified playing indoors; unspecified playing games; watching another person do active leisure activities; other active leisure.

PASSIVE LEISURE

Radio; TV; records, tapes, "listening to music", listening to others playing a musical instrument; reading or looking at books, magazines, reviews, pamphlets, newspapers, or other; being read to, listening to a story; phone conversations; other talking/conversations; arguing; receiving instructions, orders; being disciplined; letters (reading or writing); reading mail; relaxing; thinking, planning; reflecting; passive leisure related travel; other.

SPORTS AND ACTIVE LEISURE

Lessons in dance; lessons in sports activities such as swimming, golf, tennis, skating, roller skating; lessons in gymnastics, yoga, judo, body movement; lessons in music, singing, instruments; other lessons; organized meets, games, practices for team sports; meets, practices for individual sports; football, basketball, baseball, volleyball, hockey, soccer, field hockey; tennis, squash, racketball, paddleball; golf, miniature golf; swimming, waterskiing; skiing, ice skating, sledding, roller skating; bowling; pool, ping-pong, pinball; frisbee, catch; exercises, yoga; judo, boxing, wrestling; weight lifting; gymnastics; hunting; fishing; boating, sailing, canoeing; camping; snowmobiling, dune-buggies; gliding, ballooning, leaping off high buildings, flying; excursions, pleasure drives (no destination), rides with the family; picnicking; walking for pleasure, crawling; hiking; jogging, running; bicycling; motorcycling; horseback riding; photography; working on cars; working on or repairing leisure time equipment; collections, scrapbooks; carpentry, woodworking; preserving foodstuffs; knitting, needle-work, weaving, crocheting (including classes), crewel, embroidery, quilting, quilling, macramé; sewing; care of animals/livestock if not farmer; sculpture, painting, potting, drawing, coloring; literature, poetry, writing (not letters), writing a diary; playing a musical instrument (include practicing), whistling; singing; acting in/rehearsing for a play; non-social dancing; ballet, modern dance, body movement.

The table is derived from PSID Child Supplement codebook. The passive leisure variable is

defined according to all activities listed under the first four rows of above table. The active leisure variable is defined according to the last row. The other time allocation variable is defined as the residual.

10 Appendix C

We can use the calorie intake elasticities and the time allocation elasticities derived previously to see if they are consistent with one another, because time allocations can always be defined in terms of calories burned in the given activity per unit time. First, calories burned in an activity can be represented as multiples of BMR, or Basal Metabolism Rate. BMR is defined as the minimum amount of energy needed by a human body to sustain functioning of organs and maintain body temperature, measured fourteen to eighteen hours after eating when the person is completely at rest though awake. BMR also varies according to sex, weight, height, and age. For example, the BMR for an adult male aged 18-29 with a height of 5'9" and weight of 150 pounds would be about 1,720 calories per day. The average BMR for the children surveyed in the NLSY sample is about 1,704 kilocalories per day.

Now, BMR varies by sex and age, so we use a table and formulas from McArdle, et al.(2001)¹ to calculate BMR in calories per hour for each age-sex group. We can then go back to the BMI-calorie elasticities estimated above to see if the imputed change in calories from changing the time allocation gives us a predicted change in BMI that is consistent with the time allocation elasticities we computed previously. The passive activities as we defined them range in energy expenditure

¹Pages 190 and 191, with Table 9.1.

from approximately 1.2 to 2 times BMR. Physically intensive activities range from approximately 4 to upwards of 6 times BMR. Average BMR in terms of calories burned for children is 71.0 kcal per hour². So, looking at the 2SLS estimates for Column (4) in Tables 7 and 9, we see that, in order to be consistent with one another, shifting one hour of time per week toward a passive activity at the expense of an active activity should be equivalent to increasing calorie intake per day by about 21 percent³. For the average kid⁴, this translates into 395 calories per day, or 2,768 calories per week. Obviously, this is way too large as it implies a difference in BMR magnitudes between passive and active activities of 35 times BMR. Now, let us consider the non-instrumented time allocation elasticity in Column (2) of Table 7 instead of the 2SLS estimates. In this case, an additional hour of passive leisure should be equivalent to a 0.4% increase in daily caloric intake for the elasticities to be consistent with each other. For the average child, this would be an increase of 7.7 calories every day, or 54 calories weekly. This is too small but this uninstrumented, passive time allocation elasticity is much more consistent than the instrumented estimate with the 2SLS elasticity we found for food consumption. However, we would expect a decrease in calories burned by at least two times BMR, or almost 160 kcal, if a child reallocated an hour of time away from exercise intensive activities toward passive activities every week.

11 Figures

²Calculated using NLSY data for all ages below eighteen years old.

³From the time allocation elasticity of 12.5% divided by 0.585% from the calorie consumption elasticity.

⁴Average number of calories consumed in the NHANES dataset is 1,883 daily.

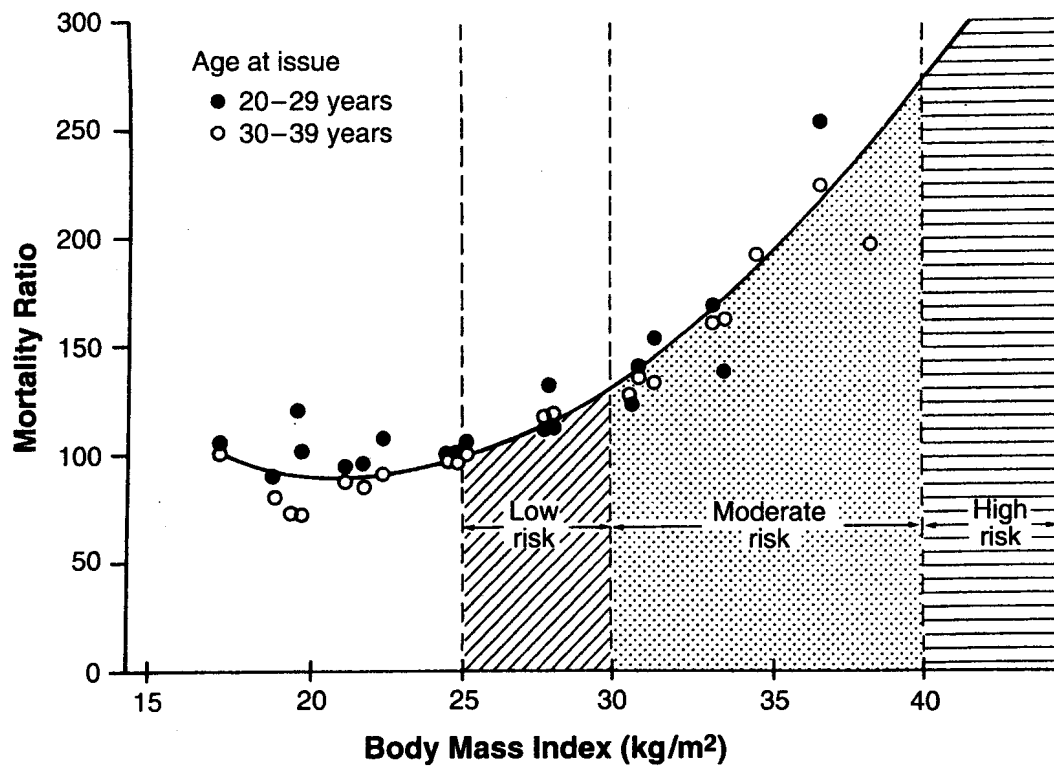


Figure 1: The relationship between BMI & relative risk of mortality for adults. (From Figure 2 in Ryan(1996)).

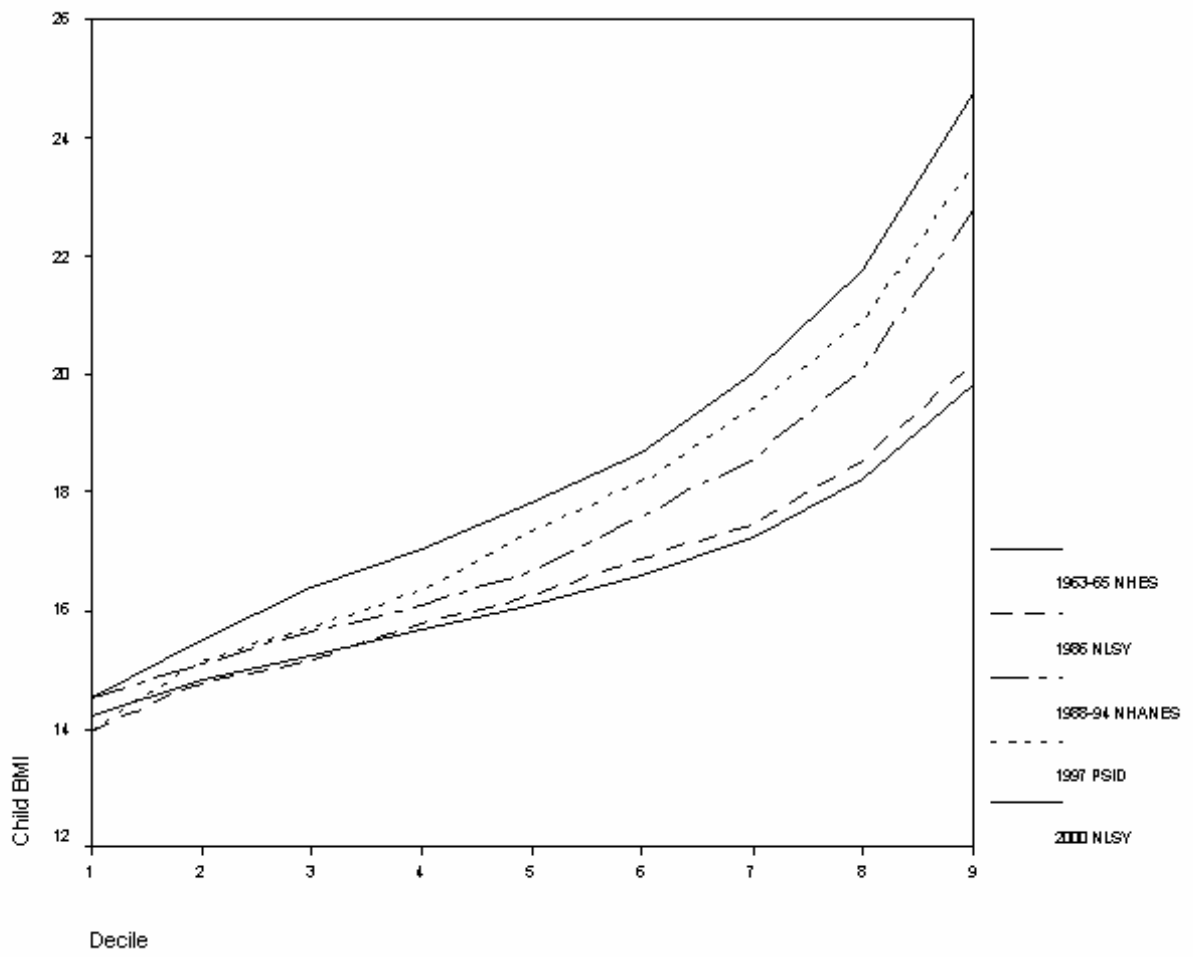


Figure 2: Change in BMI distribution.

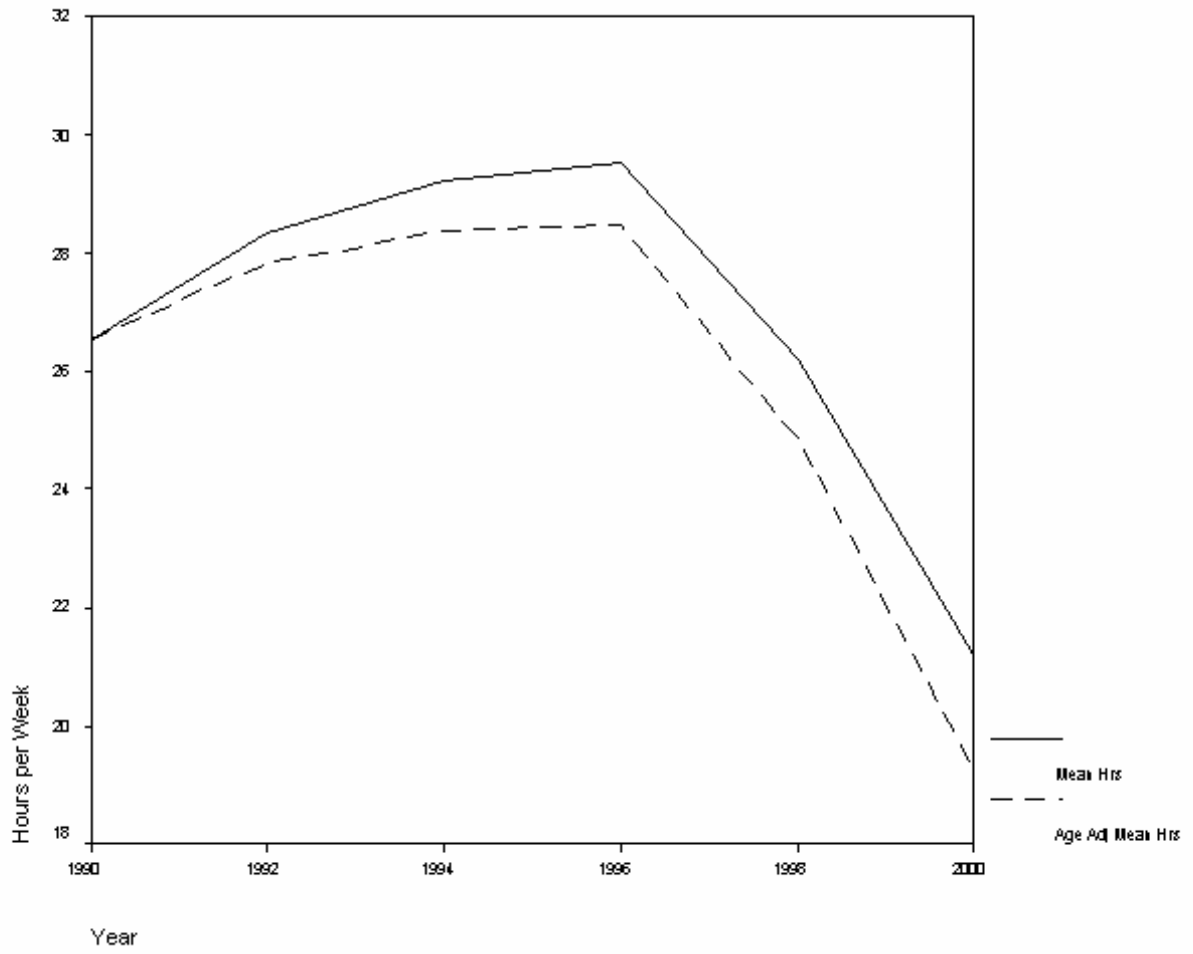


Figure 3: Average hours of TV watched weekly. (NLSY 1990-2000 for children aged three to fourteen).

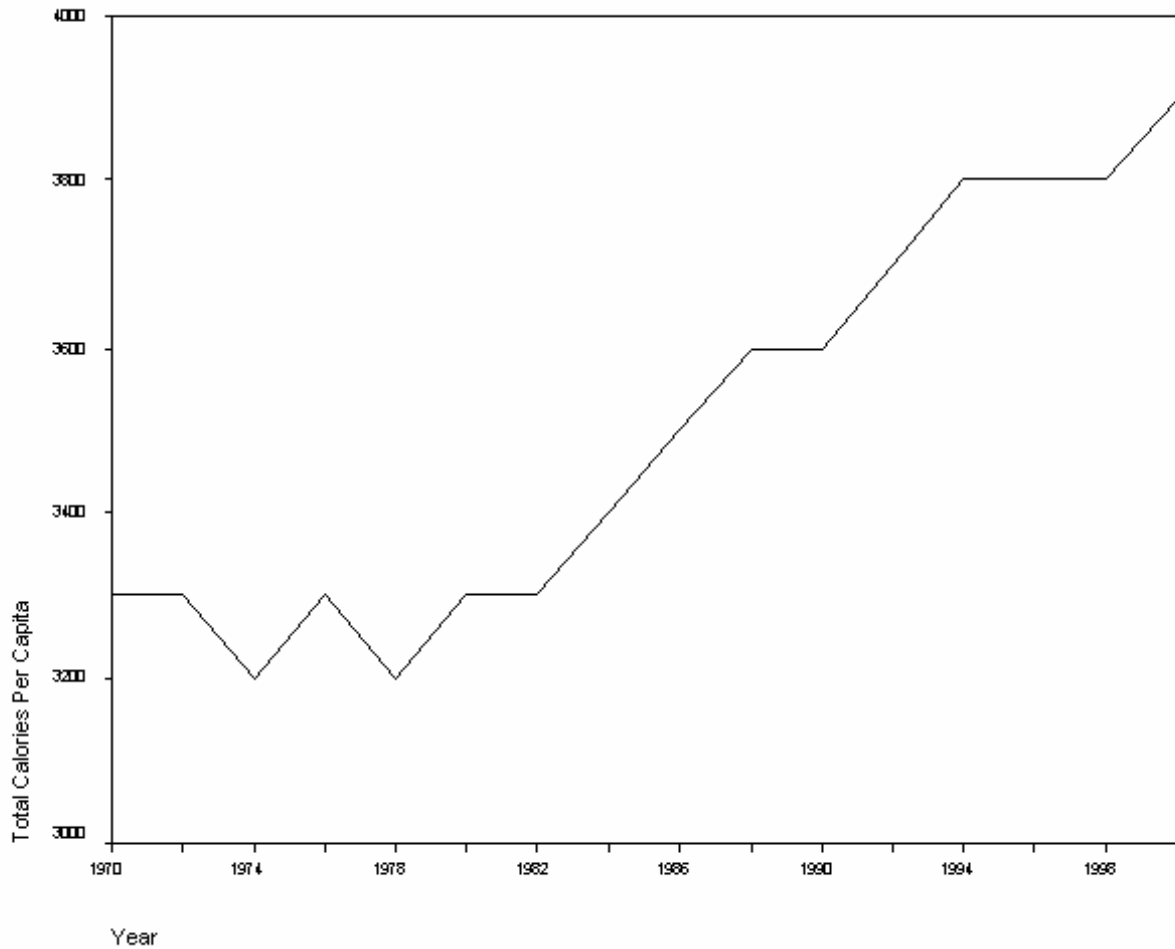


Figure 4: Total calories per capita consumed in the U.S.. This is calculated as the sum of a year's total food production, food inventories at the beginning of the year, and imports minus exports, ending inventories, industrial non-food uses, and farm uses, divided by the total, mid-year U.S. population.

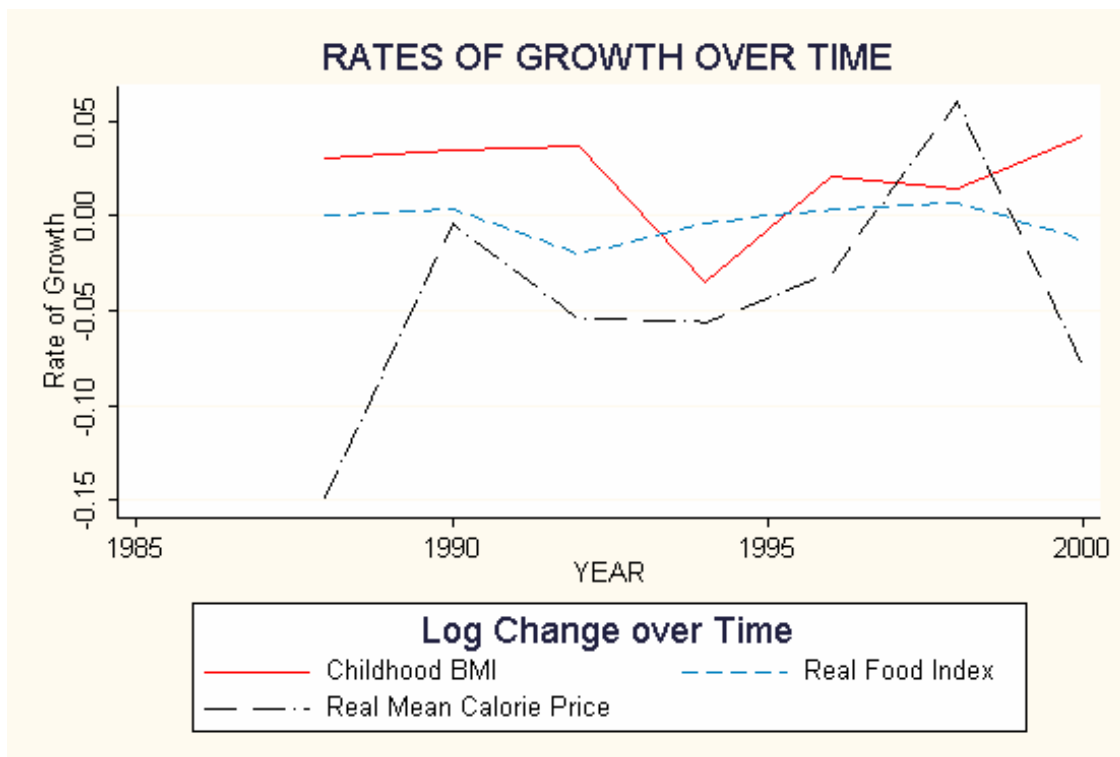


Figure 5: Rates of growth of food prices and childhood BMI.

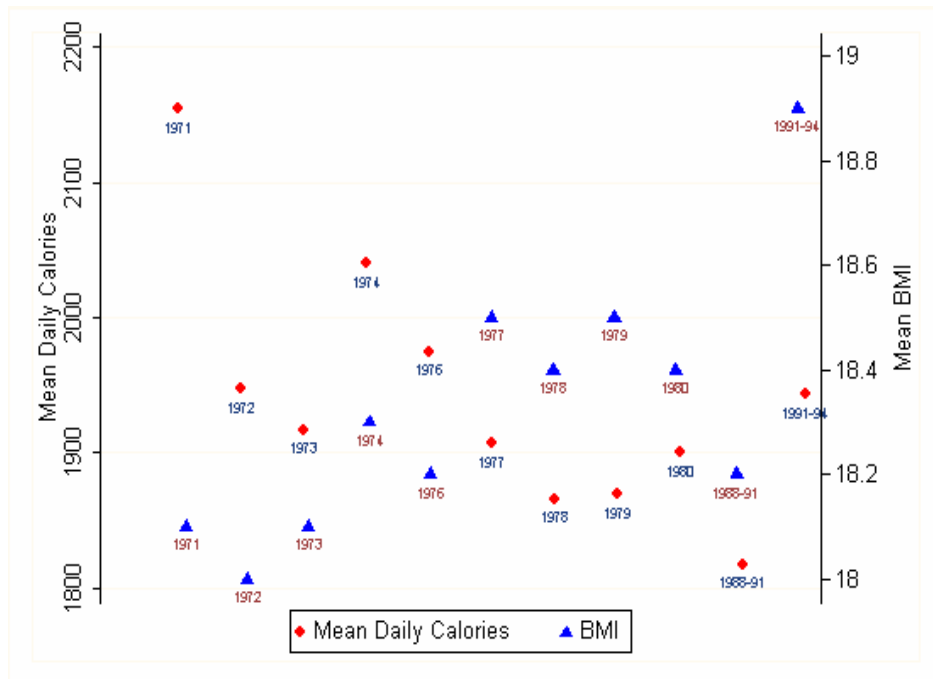


Figure 6: Mean kilocalories consumed and mean BMI of all children found in the 1971-74, 1976-1980, and 1988-94 NHANES datasets.

TABLE 1

<i>Variables</i>	<i>PSID</i>		<i>NLSY</i>	
	<i>N</i>	<i>Means</i>	<i>N¹</i>	<i>Means</i>
Child BMI	1270	17.98 (3.98)	41466	18.10 (4.33)
Mother's BMI			38843	25.68 (5.53)
Family Income	1539	52933 (44528)	34687	49230 (84348)
<i>Median Family Income</i>		43168		35000
Number of Kids	1542	2.3 (1.0)	41383	2.4 (1.1)
Black	1541	0.17 (0.37)	41466	0.16 (0.365)
Hispanic	1541	0.02 (0.15)	41466	0.08 (0.266)
Age of child	1542	6.2	41466	7.1 (4.1)
Male	1542	0.52 (0.5)	41231	.51 (0.5)
Edu of Head	1538	13.3 (2.4)	26051	13.3 ² (2.6)
Edu. Of Wife	1063	13.6 (2.1)	41343	12.7 (2.4)
Head Labor Income	1541	33276 (32333)	24961	35653 ³ (34534)
<i>Median Head Labor Income</i>		27884		30000
Wife Labor Income	1541	10679 (16684)	40113	11601 (33737)
<i>Median Wife Labor Income</i>		3050		6000
Head Labor Hrs (Wkly or Yrly)	1474	41.1 (14.1)	27258	2237 ⁴ (701)
Wife Labor Hrs (Wkly or Yrly)	1300	22.2 (19.4)	40653	1131 (975)
Computing Hours(Weekly)	164	4.57 (3.75)		
Video Gaming(Wkly)	253	6.51 (9.38)		
Active Leisure(Wkly)	1542	4.66 (6.25)		
Passive Leisure (Wkly)	1542	27.48 ⁵ (16.98)		
TV Hours(Wkly)		18.4 ⁶	29685	24.6 ⁶ (24.1)
TV Hours(Wkly)	1197	13.5 ⁸ (9.76)		

¹ Number of person-year observations.

² Education of husband only.

³ Labor income of husband.

⁴ Labor hours worked by husband.

⁵ Includes computing and video gaming hours.

⁶ Reported by parent.

⁷ Based on average number of hours watched on 'typical' weekday and weekend day.

⁸ Reported in time diary.

TABLE 2

<i>Variables</i>	<i>Estimates</i>
Constant	0.130** (0.010)
Parent OverWeight as Teenager	0.820** (0.012)
Adj. R ²	.67
F Stat	4605
N	2753

Dependent variable is dummy for whether BMI is at least one s.d. above mean in 2000.
Independent variable is dummy for whether BMI is at least one s.d. above mean in 1981.
Sample is mothers who were less than 18 yrs old in 1981. Table uses NLSY data.
** and * denote significance at the 1% & 5% levels, respectively.

TABLE 3

<i>Variables</i>	<i>Estimates</i>
Constant	0.508** (0.016)
Parent OverWeight as Teenager	0.486** (0.016)
Adj. R ²	.31
F Stat	895
N	2753

Dependent variable is dummy for whether BMI is at least one s.d. above 1981 BMI mean in 2000.
Independent variable is dummy for whether BMI is at least one s.d. above mean in 1981.
Sample is mothers who were less than 18 yrs old in 1981. Table uses NLSY data.
** and * denote significance at the 1% & 5% levels, respectively.

TABLE 4

	Mean	Std. Dev.	25th	50th	75th	90 th
1963-65 NHES Cycle II	16.66	2.56	15.03	16.07	17.63	19.81
NLSY Child 1986	16.78	3.06	14.85	16.25	17.94	20.20
1997 PSID Supplement	18.22	4.4	15.36	17.33	20.27	23.51
NLSY Child 2000	18.97	5.33	15.91	17.82	20.83	24.74

TABLE 5

<i>Variables</i>	<i>(1)PSID</i>	<i>(2)NLSY</i>	<i>(3)f.e.NLSY</i>
Constant	2.78** (0.004)	2.79** (0.003)	2.79** (0.005)
Lnfaminc	0.010* (0.005)	0.005 (0.003)	-0.007 (0.004)
Sqlnfaminc	0.002** (0.001)	-0.0004 (0.001)	-0.001 (0.001)
Numkids	-0.010** (0.002)	-0.006** (0.002)	-0.010** (0.003)
Sqnumkids	-0.0003 (0.001)	0.0001 (0.001)	-0.002 (0.001)
Agekid	0.024** (0.001)	0.022** (0.001)	0.026** (0.001)
Agekid2	0.004** (0.0001)	0.004** (0.0001)	0.004** (0.0001)
Agekid3	-0.0007** (0.0001)	-0.0003** (2E-05)	-0.0003** (2E-05)
Male	0.038** (0.003)	0.009** (0.003)	
Black	0.066** (0.006)	0.014** (0.005)	
Hispanic	0.041** (0.012)	0.001 (0.006)	
Edu. Of Head	-0.001 (0.001)	-0.002** (0.0007)	-0.0001 (0.003)
Sq. Edu of Head	0.002** (0.0003)	0.0002 (0.0001)	-0.0001 (0.0002)
Edu. Of Wife	-0.006** (0.001)	-0.002** (0.0007)	-0.002 (0.003)
Sq. Edu of Wife	0.001** (0.0002)	0.00005 (0.0002)	-0.0004 (0.0005)
Wkly Hours Work by Hd	-0.0004** (0.0001)	2.4E-04* (1.1E-04)	1.4E-05 (1.3E-04)
Hd Hrs Sq.	2.0E-05** (4.1E-06)	6.8E-06* (3.3E-06)	5.0E-06 (3.5E-06)
Wkly Hours Work by Wf	0.001** (0.0002)	3.1E-04* (1.4E-04)	-1.8E-04 (1.7E-04)
Wf Hrs Sq	-1.5E-05** (4.2E-06)	2.5E-06 (2.5E-06)	5.3E-06 (3.8E-06)
Ln Labor Inc of Head	0.004 (0.004)	0.0008 (0.003)	0.003 (0.004)
Sq Ln labor inc of Hd	0.0005 (0.0004)	0.0002 (0.0003)	0.0005 (0.0004)
Ln labor Inc of Wf	-0.021** (0.002)	0.005** (0.002)	0.004 (0.003)
Sq Ln labor Inc of Wf	-0.003** (0.0002)	0.0006** (0.0002)	0.0004 (0.0003)
Lnmbmi		0.145** (0.007)	0.009 (0.021)
Sqlnmbmi		-0.021 (0.026)	0.237** (0.049)
Within R ²			.26
B/W R ²			.13
Adj. R ²	.13	.22	
F Stat	90	233	214
N	822	19336	19336

Dependent variable is child lnmbi. Column (1) uses PSID data. Columns (2) & (3) use NLSY data, and include year dummies. Column (3) is fixed effects model. All quadratic spec variables defined as deviations from mean. ** and * denote significance at the 1% & 5% levels, respectively.

TABLE 6

<i>Variables</i>	<i>(1)PSID</i>	<i>(2)NLSY</i>	<i>(3)NLSY</i>	<i>(4) f.e.NLSY</i>
Constant	2.92** (0.022)	2.87** (0.018)	2.94** (0.018)	2.94** (0.029)
Male	0.039** (0.003)	0.011** (0.003)	0.008** (0.003)	
Black	0.064** (0.007)	0.032** (0.005)	0.017** (0.005)	
Hispanic	0.057** (0.012)	0.013** (0.005)	0.004 (0.005)	
Numkid2	-0.034** (0.004)	-0.013** (0.004)	-0.010** (0.004)	0.003 (0.006)
Numkid3	-0.023** (0.005)	-0.009* (0.004)	-0.010* (0.004)	-0.009 (0.008)
Numkid4	-0.030** (0.008)	-0.043** (0.006)	-0.038** (0.006)	-0.030** (0.011)
Numkid5	-0.129** (0.014)	-0.003 (0.009)	-0.006 (0.009)	-0.007 (0.015)
Numkid6	-0.027 (0.015)	-0.014 (0.018)	-0.009 (0.018)	-0.088** (0.028)
Numkid7	-0.062* (0.029)	-0.008 (0.025)	-0.059* (0.029)	-0.041 (0.038)
Numkid8	-0.172** (0.061)	-0.050 (0.045)	-0.124* (0.055)	-0.126* (0.061)
Numkid9		-0.089 (0.049)	-0.111* (0.048)	-0.220** (0.056)
Famincome1	0.144** (0.015)	-0.007 (0.015)	-0.025 (0.015)	0.049** (0.017)
Famincome2	0.001 (0.013)	0.002 (0.012)	-0.008 (0.012)	0.039** (0.015)
Famincome3	0.018 (0.012)	0.0003 (0.011)	-0.014 (0.011)	0.028* (0.011)
Famincome4	0.032** (0.011)	0.008 (0.010)	-0.003 (0.010)	0.022 (0.012)
Famincome5	-0.046** (0.010)	0.009 (0.009)	0.004 (0.009)	0.032** (0.011)
Famincome6	0.015 (0.010)	0.010 (0.008)	0.004 (0.009)	0.033** (0.010)
Famincome7	-0.003 (0.009)	-0.002 (0.008)	-0.008 (0.008)	0.015 (0.010)
Famincome8	-0.032** (0.008)	0.008 (0.007)	0.0005 (0.007)	0.016 (0.009)
Famincome9	-0.003 (0.007)	-0.003 (0.006)	-0.008 (0.006)	-0.002 (0.007)
HuseduHS	-0.030** (0.006)	-0.024** (0.004)	-0.022** (0.005)	0.007 (0.014)
HuseduSC	0.004 (0.007)	-0.032** (0.005)	-0.030** (0.005)	-0.001 (0.016)
HuseduCol	-0.011 (0.008)	-0.037** (0.006)	-0.029** (0.006)	-0.021 (0.020)
WfeduHS	0.008 (0.007)	-0.007 (0.004)	-0.008* (0.004)	-0.007 (0.019)
WfeduSC	-0.027** (0.008)	-0.013** (0.005)	-0.014** (0.005)	-0.019 (0.020)
WfeduCol	-0.036** (0.008)	-0.018** (0.006)	-0.021** (0.006)	0.006 (0.025)
Huswage0	-0.063** (0.010)	0.017 (0.009)	0.021* (0.009)	-0.007 (0.013)
Huswage10		0.027 (0.019)	0.038 (0.020)	-0.020 (0.021)

Huswage20	-0.081** (0.013)	0.004 (0.010)	0.002 (0.010)	-0.037** (0.014)
Huswage30	-0.046** (0.011)	0.018 (0.009)	0.011 (0.010)	-0.019 (0.013)
Huswage40	-0.054** (0.010)	0.015 (0.010)	0.009 (0.010)	-0.014 (0.013)
Huswage50	-0.055** (0.010)	0.003 (0.008)	-0.0005 (0.009)	-0.017 (0.012)
Huswage60	-0.065** (0.009)	0.008 (0.008)	0.002 (0.008)	-0.016 (0.011)
Huswage70	-0.046** (0.008)	0.011 (0.008)	0.008 (0.008)	-0.003 (0.011)
Huswage80	-0.012 (0.008)	0.011 (0.007)	0.010 (0.007)	0.002 (0.010)
Huswage90	-0.052** (0.007)	0.002 (0.007)	-0.003 (0.007)	-0.005 (0.009)
Wfwage0	0.039* (0.017)	-0.007 (0.009)	-0.006 (0.009)	-0.013 (0.011)
Wfwage40		-0.010 (0.008)	-0.010 (0.008)	-0.028** (0.011)
Wfwage50	0.051** (0.009)	-0.011 (0.008)	-0.012 (0.008)	-0.016 (0.010)
Wfwage60	0.042** (0.008)	-0.001 (0.007)	-0.001 (0.007)	-0.011 (0.010)
Wfwage70	-0.010 (0.008)	-0.006 (0.007)	-0.003 (0.007)	-0.015 (0.009)
Wfwage80	-0.019** (0.007)	-0.002 (0.007)	-0.003 (0.007)	-0.005 (0.009)
Wfwage90	-0.016** (0.006)	0.012* (0.006)	0.013* (0.006)	0.014 (0.007)
Hushrs1to20	-0.013 (0.015)	0.014 (0.014)	0.022 (0.015)	-0.011 (0.015)
Hushrs21to39	-0.016 (0.013)	0.016 (0.013)	0.024 (0.013)	-0.010 (0.014)
Hushrs40	-0.088** (0.012)	0.013 (0.013)	0.018 (0.013)	-0.012 (0.014)
Hushrs41to60	-0.045** (0.011)	0.015 (0.013)	0.022 (0.013)	-0.015 (0.014)
Hushrs61to80	-0.178** (0.016)	0.031* (0.014)	0.039** (0.014)	-0.017 (0.016)
Hushrs80+	-0.089** (0.020)	0.063** (0.020)	0.046* (0.021)	0.022 (0.023)
Wfhrs1to20	0.071** (0.015)	0.003 (0.006)	0.003 (0.006)	0.007 (0.007)
Wfhrs21to39	0.112** (0.016)	0.001 (0.007)	0.001 (0.007)	0.001 (0.008)
Wfhrs40	0.123** (0.016)	0.010 (0.008)	0.004 (0.008)	-0.003 (0.009)
Wfhrs41to60	0.112** (0.016)	0.016* (0.008)	0.013 (0.008)	0.001 (0.009)
Wfhrs61to80	0.142** (0.025)	0.005 (0.016)	0.007 (0.016)	0.023 (0.017)
Wfhrs80+	0.121** (0.025)	0.050 (0.030)	0.060* (0.031)	0.032 (0.034)
Mombmi10			-0.085** (0.006)	-0.008 (0.014)
Mombmi20			-0.084** (0.006)	-0.034** (0.013)
Mombmi30			-0.084** (0.006)	-0.045** (0.012)

Mombmi40			-0.068** (0.006)	-0.039** (0.012)
Mombmi50			-0.058** (0.006)	-0.043** (0.011)
Mombmi60			-0.055** (0.006)	-0.043** (0.010)
Mombmi70			-0.053** (0.006)	-0.040** (0.010)
Mombmi80			-0.029** (0.006)	-0.033** (0.009)
Mombmi90			-0.026** (0.006)	-0.030** (0.008)
Within R ²				.28
B/W R ²				.14
Adj. R ²	.21	.23	.24	.20
F Stat	56	78	73	65
N	822	20381	19368	19369

Dependent variable is child ln**mbmi**.

Column (1) uses PSID Child Supplement data. Columns (2)-(4) use NLSY data.

Year dummies included with NLSY regressions. Age dummy variable results not shown.

Column (4) corrects for fixed effects.

** and * denote significance at the 1% & 5% levels, respectively.

TABLE 7

<i>Variables</i>	<i>(1)</i>	<i>(2)</i>	<i>(3) IV</i>	<i>(4) IV</i>
Constant	0.011** (0.004)	0.011** (0.004)	-0.121** (0.038)	-0.123** (0.040)
Diffpassive	0.0024** (0.0004)	0.0024** (0.0004)	0.121** (0.034)	0.125** (0.040)
Diffother	0.0019** (0.0004)	0.0019** (0.0004)	0.067** (0.027)	0.070* (0.032)
Diffage	0.006** (0.001)	0.006** (0.001)	0.034** (0.009)	0.035** (0.010)
Diffsex		-0.001 (0.004)		0.018 (0.044)
Adj. R ²	.01	.01		
F Stat	17	12		
N	386	386	383	383

Dependent variable is child Difflnbmi.
 Models (3) & (4) are 2SLS regressions.
 *Statistically significant at the 5% level.
 **Statistically significant at the 1% level.

TABLE 8

<i>Variables</i>	<i>Males</i>		<i>Females</i>	
	<i>(1)</i>	<i>(2) IV</i>	<i>(3)</i>	<i>(4) IV</i>
Constant	-0.002 (0.006)	-0.027 (0.036)	0.020** (0.006)	-0.025 (0.013)
Diffpassive	0.0004 (0.001)	-0.12** (0.034)	0.004** (0.001)	0.023** (0.005)
Diffother	0.001 (0.001)	-0.071** (0.020)	0.003** (0.001)	0.011** (0.003)
Diffage	0.003 (0.002)	-0.080** (0.028)	0.007** (0.002)	0.001** (0.003)
Adj. R ²	.002		.02	
F Stat	1.2		20.3	
N	192	191	194	192

Models (1) and (2) restrict sample to males.
 Models (3) and (4) restrict sample to females.
 Models (2) and (4) are 2SLS.
 *Statistically significant at the 5% level.
 **Statistically significant at the 1% level.

TABLE 7A: First Stage of 2SLS: DiffPassiveTime

<i>Variables</i>	(3)	(4)
Constant	1.577 (0.254)	1.646** (0.255)
Diffage	-0.330** (0.068)	-0.317** (0.068)
DiffAsthma	-1.46** (0.427)	-1.679** (0.432)
DSpeech	0.628 (0.443)	0.474 (0.445)
DEar	0.095 (0.679)	0.120 (0.678)
DSight	-4.45** (0.77)	-4.415** (0.77)
DSex		0.742** (0.226)
Adj. R ²	.01	.01
F stat	13.8	13.3

Dependent variable is Diffpassive.

*Statistically significant at the 5% level.

**Statistically significant at the 1% level.

TABLE 7B: First Stage of 2SLS: DiffOtherTime

<i>Variables</i>	(3)	(4)
Constant	-0.974** (0.272)	-1.169** (0.271)
Diffage	0.109 (0.073)	0.073 (0.073)
DiffAsthma	1.47** (0.457)	2.09** (0.461)
DSpeech	-1.319** (0.474)	-0.884 (0.474)
DEar	0.947 (0.097)	0.878 (0.723)
DSight	6.99** (0.824)	6.89** (0.82)
DSex		-2.10** (0.24)
Adj. R ²	.01	.02
F stat	17.7	27.6

Dependent variable is Diffother.

*Statistically significant at the 5% level.

**Statistically significant at the 1% level.

TABLE 8 A: First Stage of 2SLS: DiffPassiveTime

<i>Variables</i>	<i>(2) Males</i>	<i>(4) Females</i>
Constant	0.356 (0.361)	2.25** (0.361)
Diffage	-1.04** (0.096)	0.235* (0.10)
DiffAsthma	-2.28** (0.565)	-0.231 (0.637)
DSpeech	0.292 (0.536)	1.380 (0.766)
DEar	-4.65** (0.853)	5.66** (1.10)
DSight	-1.90 (1.07)	-5.65** (1.11)
Adj. R ²	.04	.02
F stat	35.4	14.0

Dependent variable is Diffpassive.

*Statistically significant at the 5% level.

**Statistically significant at the 1% level.

TABLE 8B: First Stage of 2SLS: DiffOtherTime

<i>Variables</i>	<i>(2) Males</i>	<i>(4) Females</i>
Constant	-1.19** (0.389)	-0.481 (0.381)
Diffage	0.676** (0.103)	-0.378** (0.104)
DiffAsthma	3.45** (0.609)	-0.746 (0.671)
DSpeech	0.515 (0.578)	-3.84** (0.808)
DEar	3.92** (0.921)	-2.86** (1.16)
DSight	2.74* (1.15)	10.42** (1.17)
Adj. R ²	.03	.03
F stat	22.4	27.3

Dependent variable is Diffother.

*Statistically significant at the 5% level.

**Statistically significant at the 1% level.

TABLE 9

<i>Variables</i>	<i>(1)</i>	<i>(2)</i>	<i>(3) IV</i>	<i>(4)IV</i>
Constant	-0.001** (0.0001)	-0.001** (0.0001)	-0.002** (0.0003)	-0.012** (0.001)
DiffLnCalories	-0.030** (0.0001)	-0.030** (0.0001)	0.465** (0.006)	0.585** (0.012)
Diffage	0.030** (1E-05)	0.030** (1E-05)	0.012** (0.0002)	0.010** (0.0004)
Diffsex		0.001** (0.0001)		-0.118** (0.003)
Adj. R ²	.20	.20		
N	2907	2907	1028	1028

Dependent variable is child Difflnbmi.

Data are NHES.

Models (3) & (4) use 2SLS.

*Statistically significant at the 5% level.

**Statistically significant at the 1% level.

TABLE 10

<i>Variables</i>	<i>Males</i>		<i>Females</i>	
	<i>(1)</i>	<i>(2) IV</i>	<i>(3)</i>	<i>(4)IV</i>
Constant	-0.004** (0.0001)	-0.011** (0.0003)	0.002** (0.0001)	0.044** (0.0002)
DiffLnCalories	-0.035** (0.0001)	0.054** (0.001)	-0.017** (0.0002)	0.186** (0.002)
Diffage	0.029** (2E-05)	0.033** (0.0001)	0.030** (2E-05)	0.018** (0.0001)
Adj. R ²	.21		.19	
N	1456	514	1451	514

Dependent variable is child Difflnbmi.

Data are NHES.

Models (2) & (4) use 2SLS.

*Statistically significant at the 5% level.

**Statistically significant at the 1% level.

TABLE 9 A: First Stage of 2SLS

<i>Variables</i>	<i>(1)</i>	<i>(2)</i>
Constant	0.041** (0.0003)	0.048** (0.0003)
DTeeth	-0.033** (0.0003)	-0.008** (0.0002)
DSores	-0.008** (0.0002)	-0.005** (0.0004)
Diffage	0.035** (0.0001)	0.031** (0.0001)
DiffSex		0.213** (0.0003)
Adj. R ²	.02	.09
N	1028	1028

Dependent variable is DiffIncalories.

*Statistically significant at the 5% level.

**Statistically significant at the 1% level.

TABLE 10A: First Stage of 2SLS

<i>Variables</i>	<i>(2) Males</i>	<i>(4) Females</i>
Constant	0.153** (0.0004)	-0.059** (0.0003)
DTeeth	-0.037** (0.0002)	0.019** (0.0002)
DSores	0.139** (0.001)	-0.093** (0.001)
Diffage	0.033** (0.0001)	0.033** (0.0001)
Adj. R ²	.03	.03
N	514	514

Dependent variable is DiffIncalories.

*Statistically significant at the 5% level.

**Statistically significant at the 1% level.

TABLE 11

<i>Variables</i>	<i>(1)</i>	<i>(2)</i>	<i>(3)</i>	<i>(4)</i>
Constant	2.79** (0.004)	2.79** (0.004)	2.82** (0.002)	2.75** (0.002)
Lnfoodprice	-0.215 (0.171)	-0.181 (0.142)	-4.51** (0.160)	-12.58** (0.215)
Sqlnfoodprice	5.48 (12.90)			
Ln momLI	0.005 (0.002)	0.005 (0.002)		
Ln momLI2	0.0005* (0.0002)	0.0005* (0.0002)		
LnhusLI	-0.002 (0.004)	-0.002 (0.004)		
LnhusLI2	-0.0001* (0.0004)	-0.0001* (0.0004)		
Momhrs	7E-06* (3E-06)	7E-06* (3E-06)		
SqMomhrs	8E-10 (2E-09)	8E-10 (2E-09)		
Hushrs	6E-06* (2E-06)	6E-06* (2E-06)		
SqHushrs	3E-09 (2E-09)	3E-09 (2E-09)		
Lnrealfaminc	0.004 (0.004)	0.004 (0.004)		
Sqlnrealfaminc	-0.001 (0.001)	-0.001 (0.001)		
Numkids	-0.006** (0.002)	-0.006** (0.002)		
Sqnumkids	-0.0001 (0.001)	-0.0001 (0.001)		
Agekid	0.022** (0.001)	0.022** (0.001)		
Sqagekid	0.004** (0.0001)	0.004** (0.0001)		
Cubeagekid	-0.0003** (2E-05)	-0.0003** (2E-05)		
Male	0.009** (0.003)	0.009** (0.003)		
Black	0.013** (0.005)	0.013** (0.005)		
Hispanic	0.001 (0.004)	0.001 (0.004)		
Mother's Edu.	-0.002** (0.001)	-0.002** (0.001)		
Sqmomedu	0.0001 (0.0002)	0.0001 (0.0002)		
Husband's Edu.	-0.002** (0.001)	-0.002** (0.001)		
Sqhusbedu	0.0002 (0.0001)	0.0002 (0.0001)		
Ln mombmi	0.143** (0.008)	0.143** (0.008)		

SqLnmbmi	-0.020 (0.030)	-0.020 (0.030)		
Adj. R ²	.22	.22		
F stat	161	167	794	
N	19363	19363	41465	41470

Dependent variable is child lnmbi.

Columns use NLSY data with robust standard errors.

Columns (3) & (4) instrument for food prices.

Column (4) instruments & corrects for fixed effects.

** and * denote significance at the 1% & 5% levels, respectively.

Table 11A: First Stage 2SLS: Ln Food Price

<i>Variables</i>	<i>(1)</i>	<i>(2)</i>
Constant	0.240** (0.0001)	0.214** (0.002)
Aggregate Ave. Mother's BMI	-0.010** (0.0001)	-0.009** (0.0001)
Adj. R ²	.41	.39
F Stat	29000	10671
N	41465	41470

Dependent variable is child LnFoodPrices.

** and * denote significance at the 1% & 5% levels, respectively.

TABLE 12:

<i>Variables</i>	<i>(1)</i>
Constant	2.76** (0.006)
Year2000	0.073** (0.007)
Intasth2000	-0.002 (0.016)
Year98	0.041** (0.006)
Intasth98	0.042** (0.015)
Year96	0.031** (0.006)
Intasth96	0.029* (0.013)
Year94	0.011 (0.006)
Intasth94	0.020 (0.013)
Year92	0.045** (0.006)
Intasth92	0.020 (0.013)
Year90	0.032** (0.006)
Intasth90	-0.003 (0.013)
Year88	0.018** (0.006)
Intasth88	0.023 (0.015)
LnmomLI	0.002 (0.002)
LnmomLI2	0.0002 (0.0002)
LnhusLI	-0.004 (0.003)
LnhusLI2	-0.0002 (0.0003)
Momhrs(Yrly)	7E-06** (3E-06)
SqMomhrs	7E-10 (1E-09)
Hushrs(Yrly)	6E-06** (2E-06)
SqHushrs	2E-09 (1E-09)
Lnfaminc	0.003 (0.003)
SqInfaminc	-0.0006 (0.001)
Numkids	-0.007** (0.002)

Sqnumkids	-0.0001 (0.001)
Agekid	0.021** (0.001)
Sqagekid	0.004** (0.0001)
Cubeagekid	-0.0003** (2E-05)
Male	0.009** (0.003)
Black	0.016** (0.005)
Hispanic	0.002 (0.006)
Mother's Edu.	-0.003** (0.001)
Sqmomedu	0.00005 (0.0002)
Husband's Edu.	-0.002** (0.0007)
Sqhusbedu	0.0002 (0.0001)
Lnmbmi	0.133** (0.007)
Sqlnmbmi	-0.023 (0.026)
Adj. R ²	.23
F Stat	154
N	19336

Dependent variable is child lnmbmi.

Models use NLSY data & include year dummies.

** and * denote significance at the 1% & 5% levels, respectively.

TABLE 13:

<i>Variables</i>	<i>(1)</i>
Constant	21.62** (0.53)
Year2000	-0.140 (0.695)
Interact Asth2000	0.490 (3.46)
Year98	2.64** (0.599)
Interact Asth98	-3.95 (2.27)
Year96	4.91** (0.622)
Interact Asth96	2.06 (2.80)
Year94	3.63** (0.575)
Interact Asth94	-2.75 (2.22)
Year92	2.53** (0.596)
Interact Asth92	0.028 (2.27)
Ln Family Inc	-1.22** (0.235)
Sq Ln Fam Inc	-0.051 (0.046)
Num of Kids	-1.54** (0.212)
Sq Num of Kids	0.287* (0.129)
Age of Kid	0.414** (0.140)
Sq Age of Kid	-0.186** (0.012)
Cubed Age of Kid	0.03** (0.01)
Male	0.59 (0.36)
Black	12.1** (0.661)
Hispanic	5.05** (0.593)
Mother's Edu.	-1.65** (0.121)
Sqmomedu	0.01 (0.03)
Husband's Edu.	-0.24** (0.10)
Sqhusbedu	0.01 (0.02)
Lnmbmi	4.04** (0.96)
Sqlnmbmi	-1.09 (3.60)
R ²	0.19
F Stat	145
N	14284

Dependent variable is number of hours per week TV watched by child. Models use NLSY data.

TABLE 14:

<i>Variables</i>	<i>(1)</i>	<i>(2) f.e.</i>
Constant	2.77** (0.01)	2.79** (0.01)
Not Married	0.006 (0.004)	-0.012** (0.005)
Num of Adults	0.006* (0.003)	-0.002 (0.003)
Sq Num of Adults	-0.0004 (0.001)	0.0004 (0.001)
Num of Kids	-0.011** (0.001)	-0.006** (0.002)
Sq Num of Kids	0.001* (0.0006)	-0.0005 (0.001)
Male	0.005* (0.002)	
Black	0.020** (0.003)	
Hispanic	0.009** (0.003)	
Ln Mother's BMI	0.144** (0.007)	0.001 (0.014)
Sq Ln Mother's BMI	0.035 (0.024)	0.269** (0.033)
Mother's Edu.	-0.004** (0.001)	-0.002 (0.002)
Sqmomedu	-0.0001 (0.0001)	-0.001** (0.0004)
Ln Family Inc	-0.001 (0.002)	-0.004 (0.002)
Sq Ln Fam Inc	-0.0005 (0.0004)	-0.0004 (0.0005)
Mother's Age	0.001 (0.001)	-0.008* (0.004)
Sq Mother's Age	2E-07 (0.0001)	0.0001 (5E-05)
Age of Kid	0.022** (0.001)	0.035** (0.004)
Sq Age of Kid	0.004** (0.0001)	0.004** (0.0001)
Cubed Age of Kid	-0.0003** (0.00002)	-0.0003** (0.00001)
R ²	0.23	0.28
F Stat	283	582
N	32523	32523

Dependent variable is child ln**mbmi**. Table uses NLSY data. Column (2) is fixed effects model. ** and * denote significance at the 1% & 5% levels, respectively.

TABLE 15: Education Breakdown of Data in Percent

	<i>Cycle II</i>		<i>PSID</i>		<i>NLSY</i>	
	<i>Mothers</i>	<i>Fathers</i>	<i>Mothers</i>	<i>Fathers</i>	<i>Mothers</i>	<i>Fathers</i>
HS Dropout	43	45.3	7	10.4	20	9
HS Degree	41	31.8	37	34.8	40	43
Some College	9	8.2	27.1	21.7	23	20
College Grad	6.7	14.7	28.9	33	18	46

TABLE 16: Percentage of Children with a Computer

<i>Year</i>	<i>Percent</i>
2000	79
1998	64
1996	49
1994	35

From NLSY Child Supplement data.

TABLE 17: Daily Calorie Intake by Source for NHANES I & III

Food Consumed	<i>Children who eat (at):</i>					
	<i>Snack</i>		<i>Restaurant</i>		<i>Home</i>	
	<i>I</i>	<i>III</i>	<i>I</i>	<i>III</i>	<i>I</i>	<i>III</i>
Snack	532	542	659	482	494	458
Restaurant	104	131	735	811	95	113
Home	1555	1325	1164	876	1552	1290

TABLE 18

<i>Mean Age</i> <i>(s.d.)</i>	
<i>NHANES I:</i>	
1971	9.2 (4.9)
1972	9.3 (4.9)
1973	9.3 (4.9)
1974	9.5 (4.7)
<i>NHANES II:</i>	
1976	9.3 (5.0)
1977	9.5 (5.0)
1978	9.4 (5.0)
1979	9.0 (5.0)
1980	9.0 (5.0)
<i>NHANES III:</i>	
1988-91	8.5 (4.8)
1991-94	8.6 (4.8)