CHILDHOOD DISADVANTAGE AND OBESITY:
IS NURTURE TRUMPING NATURE?

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## **ABSTRACT**

Obesity has been one of the fastest growing health concerns among children, particularly among disadvantaged children.  For children overall, obesity rates have tripled from 5% in the early 1970s to about 15% by the early 2000s.  For disadvantaged children, obesity rates are closer to 20%.  In this paper, we first examine the impact of various measures of disadvantage on children's weight outcomes over the past 30 years, finding that the disadvantaged have gained weight faster.  Over the same period, adult obesity rates have grown, and we expect parental obesity to be closely tied to children's obesity, for reasons of both nature and nurture.  Thus, examining changes in the parent-child correlation in BMI should give us some insight into the ways in which the environment that parents and children share has affected children's body mass, or into how the interaction of genes and environment has changed. We find that the elasticity between mothers' and children's BMI has increased since the 1970s, suggesting that shared genetic-environmental factors have become more important in determining obesity.  Despite the faster weight gain for the disadvantaged, there appears to be no clear difference for by disadvantaged group in either the parent-child elasticity or in identifiable environmental factors.  On average, the increases in parents' BMI between the early 1970s and the early 2000s can explain about 37 percent of the increase in children's BMI.  Although common environmental/genetic factors play a larger role now than in earlier time periods, child specific environments such as schools and day care play a potentially important role in determining children's health status.

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# I. Introduction

Obesity has been one of the fastest growing health concerns among children, particularly among disadvantaged children. Childhood obesity has risen starkly over the last three decades. For children overall, obesity rates have tripled from 5% in the early 1970s to about 15% by early 2000s. For disadvantaged children, the rates of obesity are even higher. For example, in the years 1999-2004, nearly 18% of low income children qualified as obese.

Obesity carries with it both short term and long term consequences. Obese children have higher incidences of type II diabetes, for example, and lower quality of life scores. In addition, obese children are much more likely than normal weight children to become obese adults, and obese adults are more likely to suffer disability during their prime working years, and have adverse health outcomes like hypertension, heart attack and cancer. While the precise impact of obesity on mortality remains a matter of debate, there is little debate that obesity increases morbidity and its attendant health care costs. Since disadvantaged children and adults have higher rates of obesity than other segments of the population, they suffer more of the short term and long term consequences of this condition.

Understanding the increase in childhood obesity is important for devising policies to deal with this health problem. Although recent research and policy activity surrounding this issue has focused particularly on the food available to children through schools, there is a gap in our knowledge when it comes to the impact of home environment on children's obesity. To address this question, we ask, how does parental obesity relate to children's obesity? Is this different for disadvantaged families? Have these relationships changed over time? Parental obesity is very closely tied to children's obesity, for reasons of both nature and nurture. First, there is a strong genetic component to body composition. Second, parents and children share many

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environmental characteristics. Additionally, parents have a great deal of influence over what their children, particularly their young children, eat and how much energy they expend. Since genetics are unlikely to have changed dramatically over the past thirty years, if the correlation between parents' and children's obesity has changed over time, then it is likely that the environment and/or parental behavior has changed. If the overall correlation has increased over time, it suggests that something in the common environment (or decisions made by the family) is affecting all family members. On the other hand, if the correlation has decreased over time, then it suggests a larger role for something unique to the environment that children – but not their parents – face, for example in child care settings and public schools.

In addition, there may be important differences in the relationship between parents' and children's obesity for disadvantaged children. For example, if the parent-child correlation is lower for the disadvantaged, then it suggests that the child-specific environmental factors may be relatively more important for disadvantaged children. Again, if the relationship is changing over time, it provides clues as to changes in the environment that may be contributing to the changes in obesity. Thus, this paper focuses on a fundamental component of health status, obesity, for which disadvantaged children have particularly poor outcomes. It sheds light on how parents' health status is related to children's health status, and how that relationship differs for disadvantaged and advantaged children, and how the relationship changes over time.

We find that the parent-child correlation in weight outcomes has increased substantially since the early 1970s. This suggests that the importance of the shared family environment or genetic-environmental interactions has increased over time. Despite the fact that disadvantaged groups have higher obesity rates, parent-child weight outcomes are similar for advantaged and

disadvantaged groups. On average, the observed increase in parents' body-mass-index[1] (BMI) can explain about 37 percent of the increase in children's BMI since the early 1970s. Thus, for advantaged and disadvantaged groups, genetic tendencies toward obesity and how these tendencies interact with the common environment are important, and increasingly so as obesity rates have risen over time. Nonetheless, for both advantaged and disadvantaged children there is an important role in their health status is played by child-specific environments, which suggests that policies affecting schools, day cares, playgrounds and the like may have an effect on children's obesity.

## II. Previous Research in this Area

Economic research on obesity has focused on changes in the (implicit) prices of food and exercise that have increased caloric intake and reduced energy expenditure (Cutler, Glaeser and Shapiro, 2003; Lakdawalla and Philipson, 2002). While this work is important, it is not clear how well it applies to children, especially young children, who typically do not select the menu of food presented to them, nor do they have complete control over how they spend their time. Thus, work on childhood obesity has focused on changes in children's environment that may have tilted their energy balance toward consuming more calories and expending less energy. For example, work has focused on changes in maternal employment (Anderson, Butcher, Levine 2003), and changes in the food available to children through schools (Anderson and Butcher 2006b; Schanzenbach 2005), and how these changes in the two institutions, families and schools, in which children spend most of their time, may have affected obesity.

There is, of course, a substantial literature outside economics on childhood obesity. Much of this focuses on whether children who consume more of specific types of foods (e.g. fast food, soda) or engage in particular activities (e.g. television watching, video game playing) are more

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[1] Weight in kilograms divided by height in meters squared.

likely to be obese (see Anderson and Butcher, 2006a for a summary of this literature). In addition, there is a large literature documenting that children from disadvantaged backgrounds are more likely to have weight problems than others (see e.g. Strauss and Pollack, 2001). Finally, there is a large literature documenting that there is an important genetic component of obesity (see e.g. Stunkard et. al., 1990).

## III. Our Approach

What we believe is missing is a better understanding of whether the obesity epidemic in children is simply part and parcel of the obesity epidemic in adults, or whether it represents a related, but separate, phenomenon. If we see a high correlation between children's and parents' weight outcomes, then it suggests that increases in parents' weight – or that the same factors that led to parents' weight gain -- can explain a large fraction of the increase in children's weight. This would be consistent with an explanation for the rise in children's obesity where both parents and children are faced with an adverse environment that leads to more obesity, or that parents have always made determining choices about food and exercise that have affected their own and their children's weight – and in recent years these choices have been poor choices.

On the other hand, if we see that children's weight outcomes are not highly correlated with their parents, then it suggests that there are other, non-shared, factors that determine children's weight outcomes. At any given point in time, there may be differences between advantaged and disadvantaged children in how closely related children's weight is to parents' weight. For example, a parent who lives in a well-off neighborhood may walk to work for exercise and may encourage her children to walk to school as well. On the other hand, a parent in a disadvantaged neighborhood may feel comfortable walking to work herself, but may be less comfortable with her children walking to school due to safety concerns (traffic safety or criminal

activity, for example). We would expect parents and children in the well-off neighborhood to have more similar weight outcomes; while in the disadvantaged neighborhood parents and children would have less closely linked weight outcomes. Parent-child correlations may change over time, and these changes may also differ between advantaged and disadvantaged children. For both advantaged and disadvantaged children, we will discuss how much of the change in children's weight outcomes can be explained by parents' weight outcomes, given the correlation between the two in a given time period.

To address the question of how the parent-child correlation in BMI has changed over time for disadvantaged and non-disadvantaged groups, we rely on data from the National Health and Nutrition Examination Survey (NHANES). The nationally representative data were collected in 1971-1974 (NHANES 1), 1976-1980 (NHANES 2), 1988-1994 (NHANES 3). Beginning in 1999, the NHANES became a continuously running survey collected in 2-year panels. We pool the surveys from 1999-2000, 2001-2002 and 2003-2004 and refer to them (for comparison's sake) as "NHANES 4." (See data appendix for more details).

The NHANES are the most frequently used data for tracking obesity trends in the U.S. population. The data collection procedures include an examination component, so the children in our sample were weighed and measured by trained personnel. These measurements go into our calculation of BMI for children. For each of the first three NHANES, reported height and weight is consistently available for the parents of examined children under age 12. Thus, we focus on children 2-11 years old. In addition to the restrictions imposed by data availability, questions about the impact of parental choices on children's BMI may be particularly germane for this group since adults are likely to have a greater influence over these children's food and exercise options than would be the case for teenagers. In the fourth NHANES, reported parental

height and weight are not available, however, for both the third and fourth NHANES there are enough children in the sample who have an adult household member who is also in the sample that we can match children and adults within households and use measured heights and weights for both children and adults.[2] The Data Appendix describes more fully how we do the within-household matching and how this matched sample compares with the parental report sample in NHANES 3, where both are available.

## IV. Changes in Obesity and BMI

### A. Measures of Obesity

Obesity for adults is typically defined as having a BMI of 30 or above. Children are classified as obese if they have a BMI above the 95th percentile of an age-sex specific BMI distribution (calculated with data that pre-dates the current increase in obesity).[3]

One can also examine obesity using alternative measures of body composition. BMI does have drawbacks – in particular, muscular individuals may have a higher body-mass-index than someone of the same build who is less muscular, and higher BMI in this case presumably does not indicate a poor health outcome. Ideally, one would like a measure of "fatness" that we know is related to poorer health outcomes both within and across individuals. Alternative measures to BMI, for example, subscapular skin-fold measurements are available in the NHANES. While the levels of obesity sometimes differ by these different measures, the trends in obesity over time show similar increases regardless of the measure chosen to define obesity (Burkhauser et al., 2007).

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[2] We can also match children to adults within the household in the first two waves of the NHANES, but this results in losing about ¾ of our sample.

[3] The nomenclature in the medical literature is different for children and adults. Children whose BMI is above the 95th percentile for their age-sex distribution are called "overweight" and those above the 85th percentile are called

Most researchers have chosen to focus on BMI since there tend to be very high correlations between obesity rates as measured by any of the available outcomes, and BMI is relatively easily measured in surveys. A recent study on measuring obesity in children found no additional information was gleaned from subscapular skinfold measurements once BMI was accounted for (Mei, et. al., 2007). Thus, we focus on BMI and obesity rates as defined by BMI in our analysis below.[4]

### B. Measures of Disadvantage

Disadvantage is difficult to define, but has many correlates. Our approach is to examine BMI and obesity outcomes by several different potential measures of disadvantage. We examine differences in weight outcomes between race and ethnic groups, by educational attainment of adults in the household, and by income-to-poverty line measures. In general, these measures give similar pictures of obesity levels and trends and the parent-child correlation in BMI for advantaged and disadvantaged children.

### C. Trends in Obesity

Before turning to our investigation of children's weight outcomes, it is worth establishing how quickly adult obesity has been spreading throughout the United States. Figure 1 shows maps for 1990, 1995 and 2005 created by the Centers for Disease Control and Prevention using data from the Behavioral Risk Factor Surveillance System (Centers for Disease Control and Prevention, 2007). In 1990, in almost all states less than 15 percent of the adult population was obese. By 2005, many states have more than 25 percent of their adult population that is obese. Given our interest in the role of disadvantage, it is important to note that states known to have a

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"at-risk-for-overweight." In order to simplify the discussion, we will use the adult terminology for both children and adults.

[4] We have also conducted many of our analyses using subscapular skinfold measurements and get very similar results for the effect of disadvantage and for our parent-child correlations in outcomes.

relatively large poor population appear to be the leading edge of the obesity wave.  In 1990, it is states such as West Virginia, Louisiana and Alabama that have over a 15% obesity rate.  Similarly, by 2005, these same states, along with their neighboring states, have exceeded a 25% obesity rate.

Turning now to children, Figure 2 uses NHANES data to illustrate the differences across time in obesity rates for children age 2 to 11 (the focus of our study), as well as demonstrating the differences by the level of disadvantage for each time period.  Here we are using the income-to-poverty line ratio as our indicator of level of disadvantage, examining obesity rates for children from families who fall into each quartile of the income-to-poverty-line ratio.[5]  This figure clearly demonstrates the increase in obesity among children between 1971-1975 and 1999-2004.  As seen in the last set of bars, the overall percentage obese almost triples from 5 percent in the earliest years to about 15 percent in the more recent years.  We can also see that this increase over time applies to children in all family income groups.  Although children in each of the family income groupings have increased their obesity rates, the gains have not all been the same.  By the last period there is a clear gradient to children's obesity, with the lowest family income group having the highest fraction obese and children in the highest family income group having the lowest fraction obese.  However, this gradient is not as clear in the earlier years.

Focusing on the fraction obese may mask some important features of changes in obesity, though. While an adult will be labeled as obese with a BMI greater than or equal to 30, it seems unlikely that someone with a BMI of 30.1 will have much worse health outcomes than someone with a BMI of 29.9.  Thus, if the increase in obesity is driven solely by small increases in BMI

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[5] Information on family income itself is only available in bracketed form, but a continuous income-to-poverty ratio measure is available in each year, which is a measure of reported family income relative to the poverty line.  Thus, anything above 1 indicates the family's income is above the poverty line for that time period, and as it increases, families are better off.

that push individuals across a (somewhat) arbitrary threshold that labels obesity, then we might not worry much about the future health consequences of these changes. Since children's obesity status is determined by a similar comparison to a (in this case age and gender specific) threshold, we want to examine what is happening to BMI among the obese, not just to the fraction of children whose BMI is above the cutoff.

In fact, the obesity epidemic is generally characterized by an increase in weight among those who are already heavy (Cutler et. al. 2003, Anderson et. al. 2003b). Figure 3 shows how average BMI among obese children has changed over time and by family income groups. Average BMI among all obese children increased from 22.5 to 23.6 during this time period (about a 5 percent increase). In addition, by the later period we see a family income gradient emerge in average BMI among the obese. In the first period, there is if anything a reverse gradient – that is, the average BMI of the more advantaged obese children is higher than that of disadvantaged obese children. In the middle years, there is little systematic difference in the BMI between advantaged and disadvantaged obese children. By the later period, though, obese children in the poorer families are heavier than obese children in wealthier families. As differences in BMI get larger between the obese and the non-obese, we expect that long term health outcomes between them will also get larger. It is important to keep in mind, however, that differences in the age and sex composition across these groups and over time could be driving to these changes in BMI levels. In the next section we turn to regression analysis that allows us to hold constant age and gender as we examine the changes in BMI by different measures of disadvantage.

*D. Changes in BMI over Time by Measures of Disadvantage*

We first present simple regressions of children's log BMI on various measures of disadvantage for our four time periods. These regressions control for a cubic in age, gender, and interactions between these variables.[6] This should account for the fact that as children age, we expect their BMI to increase, and to increase at different rates for boys and girls. We control for age and gender in the regressions so that differences in BMI by measures of disadvantage will not be due to differences in the age-sex composition across these groups. Heteroskedasticity-robust standard errors are in parenthesis below the estimated coefficients.

The top panel of Table 1 presents regressions of log BMI on racial and ethnic categories. The second panel shows regressions of log BMI on the highest grade of the household reference person.[7] The third panel shows the results for the income-to-poverty ratio. The fourth panel includes all measures of disadvantage simultaneously. In all years, there is some evidence that disadvantaged children are heavier, although the strength of that correlation varies with the measure and the time period. For example, black children do not have significantly higher BMIs than white children prior to 1999, although in the 1988-1994 wave of the NHANES the point estimate implies black children have an almost 1.0 percent higher BMI. By the 1999-2004 wave, that difference had increased to 2.1 percent and was statistically significant. Currently, the median 8-year-old girl stands at 4'2" and weighs 56 pounds, so we would expect her to be 1.2 pounds heavier if she were black. Hispanic children have significantly higher BMIs than white non-Hispanic children in all but the first time period, and in the time periods when the difference is statistically significant it is stable at about 3.5 percent. For the same median 8-year-old girl then, we would expect her to be about 2 pounds heavier if she were Hispanic.

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[6] Note that sample sizes do not match those from later tables, as for these regressions we use all possible data. Regressions run on consistent samples are not substantively different from those shown here.

[7] For NHANES 4, we define the reference person as the father, if present, otherwise, it is the mother. For the other three panels, the reference person is defined within the NHANES. See the Data Appendix for more details.

Being from a household where the reference person has a higher level of education is statistically significantly negatively correlated with BMI in all four time periods, increasing from a 0.2 percent lower BMI for each additional year of parental education in the early 1970s to a 0.6 percent reduction for each additional year by the early 2000s. Prior to 1980, children from families with higher income-to-poverty ratios did not have significantly lower BMIs. After 1988, however, that correlation is negative and statistically significantly different from zero, and becoming steeper over time. By the last period, a from a family with income at the poverty line would be about 2.4 percent heavier than one with income three times the poverty line, compared to just 1.8 percent heavier in the previous period.

When we include all measures of disadvantage simultaneously, we find that once the income-to-poverty ratio and education are held constant, being black is no longer significantly positively correlated with BMI in any of the years, but being Hispanic is still associated with a higher BMI in all but the first period. Each additional year of education of the household reference person is associated with a 0.1 to 0.5 percent reduction in BMI, but the effect is no longer increasing over time, and in fact is not significantly different from zero in the final period. Finally, in all but the third wave, the income-to-poverty ratio is significantly correlated with children's BMI, however in the first two periods the effect is positive. Overall, then, these regressions show that disadvantaged children are generally heavier, using a variety of measures of disadvantage. Additionally, for most of these measures, the effect of disadvantage has been growing over time.

**V. Parent's and Children's Weight Outcomes**

A. *Mothers and Fathers*

Parents and children share common genetics, and, if they live together, share a common environment. Thus, we expect to see a strong correlation between the BMI of parents and children. Note that the literature on nature versus nurture typically uses the standard behavioral genetics model to carry out a decomposition of these two effects by assuming that an outcome is a linear combination of the independent effects of genetics, shared family environment, and unexplained factors. Given that, in fact, genetics and the shared environment are unlikely to be independent, it is typical to interpret the genes component as incorporating both the direct effect of genes and the effect of the gene-environment interaction (Sacerdote, 2007). Thus, while we do not expect that the underlying genetic predisposition toward obesity will have changed over the twenty or so years that childhood obesity has been increasing, examining changes in the parent-child correlation in BMI should give us insight into the ways in which the environment that parents and children share has affected children's body mass, or into how the interaction of shared genes and the environment has changed.

We investigate the parent-child correlation in BMI by running regressions of log(Child's BMI) on log(Parent's BMI). The coefficient on log(Parent's BMI) tells us the elasticity of children's BMI with respect to their parents – a one percent change in parent's BMI is correlated with what percent change in children's BMI. A larger elasticity implies a greater role for the common environment and genes that parents and children share; a smaller elasticity implies a greater role for environments that children do not share with their parents.

Table 2 presents regressions of the log of child's BMI on his or her mother's log BMI (column 1), his or her father's BMI (column 2), and both simultaneously (column 3). These regressions control for race, ethnicity, education of the reference person, the family's income-to-

poverty ratio, and a cubic in the child's age fully interacted with gender.[8]  The results in the first panel show that a 10 percent increase in either mothers' or fathers' BMI is correlated with about a 1.4 percent increase in child's BMI.  When both parents' BMI measures are in the regression the individual coefficients decline somewhat – as is to be expected since mothers' and fathers' BMI are correlated as well – but both are statistically significant.  If children, on average, shared the same amount of common genetics with their mothers and their fathers, but shared more common environment with their mothers we might expect that mothers' BMI would be more important in explaining children's BMI than fathers' – as mothers' BMI would pick up both the effect of common genetics and common environment.  These results suggest that the common environment shared by mothers and their children and fathers and their children is similar.  One caveat to this is that fathers' reported BMI is more likely to be missing than mothers. Thus, one might say that the parent-child correlation in BMI is about the same for fathers and mothers, *when the father is present*.  Since including fathers' BMI in the regression does not make much difference to the estimated mother-child correlation in BMI and we lose observations when we require non-missing father BMI data (and those children who are missing fathers' BMI data are more likely to be disadvantaged), we focus on mother-child BMI elasticities in the analyses that follow.

Looking down the panels in Table 2 we can see how the mother-child BMI elasticity has changed over time.  Between the first and second wave of the NHANES, recall, children's obesity rates and BMIs do not change very much.  Here we see that the mother-child BMI elasticity is nearly identical in these two periods as well, implying that a 10 percent increase in mothers' BMI is correlated with a 1.4 percent increase in children's BMI.  In the third wave of the NHANES, the mother-child elasticity has grown larger. Now, a 10 percent increase in

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[8] Results look similar if the child's own birth weight is included as a control variable.

mothers' BMI is correlated with a 2.1 percent increase in children's BMI. In the fourth wave, the mother-child elasticity remains larger than in the earlier periods, with a 10 percent increase in mothers' BMI being correlated with about a 2 percent increase in children's BMI.

We can use the parent-child elasticity to estimate how much of the increase in children's average BMI over time would be predicted, given the mothers' average increase over the same time. We use the average BMI for mothers and children shown in Appendix Table 1 to calculate the percentage change in children's BMI and the percentage change in mothers BMI between the first and fourth period. Using the standard midpoint formula, this would imply that mothers' BMI grew 13.7 percent and the children's BMI grew 7.3 percent.[9] However, even using the larger, later period, elasticity, the growth in mothers' BMI would imply less than a 3 percent growth in children's BMI.[10] The growth in mothers' BMI can explain 37.5 percent of the increase in children's BMI between the beginning and the end periods. Clearly, there are other factors besides the shared genetic-environmental factors of mothers and children that are driving the increase in children's BMI.

*B. Interpreting the BMI Elasticity*

As noted above, the parent-child BMI elasticity cannot be used to isolate pure genetic and pure environmental determinants of weight status. The observed increase in mother-child BMI elasticity is best interpreted as reflecting an increase in the shared environment of mothers and their children, or the effect of the interaction of shared genes with the environment.[11] Using a sample of adult adopted and genetic siblings collected in 2004, Sacerdote (2007) estimates that the shared environment explains 30.8 percent of the variance in BMI, genes (and their correlation

[9] This is obtained by dividing the change between the first and last period by the average of the two periods.

[10] Percentage change in children's BMI = (elasticity* percentage change in mothers' BMI) = (0.2*13.7 percent)

with the environment) explain 11.5 percent, and the rest (57.7 percent) is unexplained.

Interestingly, this decomposition is very close to the one he calculates for family income, even though this is an outcome that typically might not be considered to be as genetically determined as BMI. That said, the role of genetics in the mother-child BMI elasticity is made clear when comparing estimates using the adopted children to those using the biological children.[12] Running a regression similar to that shown in column (1) of Table 2 using these data[13], one obtains an elasticity (standard error) for the biological children of 0.221 (0.045), but only 0.025 (0.025) for the adoptees. Thus, the elasticity for biological children for this sample is similar to our later elasticities, but it is unlikely to reflect current shared environment. The implication is that the interaction of shared genes and the general, not just the intra-household, environment is likely very important. Thus, suppose that some parents and children are pre-disposed to eat too much junk food, and vending machines go from being rare in the early period to being ubiquitously available in the later period both at work and in schools. We would observe an increase in BMI in both parents and children for the genetically pre-disposed that we would attribute to shared genetic-environmental factors. However, it is not necessarily the case that these shared environmental factors are solely within the walls of the household.

Finally, recall that although BMI has increased for everyone, the BMI for the obese has increased more than for the median person, which corresponds to a model of obesity such that there may be some people who are particularly susceptible to weight gain when the environment

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[11] One concern is whether the increase in elasticity is real, or an artifact of changes in measurement error in BMI over time. We find that changes in measurement error would have to be implausibly large to be the underlying driver of the observed increase in parent-child BMI elasticities. See data appendix for a more detailed explanation.

[12] Recall that these "children" are actually adults – the average age is 28 for the adoptees and 32 for the biological children. Thus, it is unlikely that mother and child are literally sharing an environment at this point.

[13] We thank Bruce Sacerdote for making his data available to us.

is right, and in the last two decades, the environment has been right.[14]  Thus the impact of common genetic and environmental factors may be different for children who occupy different points in the BMI distribution.  Given that we have seen that disadvantaged kids are heavier, it is important to investigate whether the mother-child elasticity is constant throughout the BMI distribution or not.  Thus, we estimate models identical to column (1) from Table 2, but using quantile regression rather than OLS.  We estimate elasticities that fit at each of 20 quantiles of the BMI distribution (versus OLS, which fits at the mean).

Figures 4-7 plot these quantile regression estimates and 95 percent confidence intervals for the mother-child BMI elasticity for NHANES 1-4. First, we see that the elasticity is generally higher at higher quantiles of the BMI distribution, suggesting that for heavier children, the shared environmental-genetic component captured by mothers' BMI is more important in determining their BMI.  Second, this relationship seems to have gotten steeper over time.  That is, while the elasticity is around 10 percent for the lowest quantiles in all periods, in the earlier periods it does not rise much above that level until increasing sharply at about the 80th percentile.  By contrast, in the later periods the elasticity rises smoothly throughout the quantiles.[15] The heavier the child, the more important the common family genes and environment appear to be in determining her weight outcomes.

*C. Differences in Mother-Child Elasticities by Measures of Disadvantage*

Since disadvantaged children are heavier, the results in the preceding section might lead one to believe that parent-child elasticities would be higher for disadvantaged groups. Table 3

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[14] Note that how this all works is far from settled science.  Recent evidence suggests that the obese may have different body chemistry such that for a given number of calories ingested, their digestive tract can extract more calories for use (Weill Medical Center, 2007).

[15] Note, of course, that children at the middle quantiles in NHANES 3 and 4 have BMIs that would have placed them farther up the quantile distribution in the earlier year, but since we are using ln(BMI) the shift is not as stark as it would be in levels. Thus, across NHANES 2 and 3, median ln(BMI) only increases from 2.77 to 2.78, while 75th percentile ln(BMI) increases from 2.85 to 2.88.

investigates whether the mother-child elasticity, which has increased on average, has changed in similar ways for advantaged and disadvantaged groups. The regressions control for a cubic in age fully interacted with gender, and all measures of disadvantage. Interactions between mother's log BMI and specific measures of disadvantage are entered separately. The top panel shows the interaction of log of mothers' BMI with race/ethnicity; the second panel shows the interaction with the reference person's highest grade completed; the third panel shows the interaction with the income-to-poverty-line ratio. The results for the different waves of the NHANES are reported in the four columns.

Looking down the panels we see, in general, that there is little consistent role for disadvantage in terms of its effect on the mother-child elasticity.[16] In the first panel, the majority of the point estimates imply that the mother-child elasticity for blacks and for Hispanics is smaller than for white non-Hispanics, but the estimates are not significantly different from zero and are not consistently negative in all periods. In the second panel, using the household reference person's education as a measure of disadvantage, there is again no real impact on the elasticity. In the final panel, when we use perhaps the most straightforward measure of disadvantage - the income-to-poverty-line ratio - the majority of the point estimates imply a lower elasticity for the disadvantaged, but again the difference is not significantly different from zero.

Nonetheless, Table 4 uses the point estimates from Table 3 to calculate the implied mother-child BMI elasticity for different groups. Doing this calculation makes clear that this elasticity has increased over time for most groups, but indicates that it may have decreased slightly (albeit not significantly) for the more advantaged groups in the last period. Recall from Table 1 that the effect of disadvantage on children's BMI was positive and growing over time.

That is, that the disadvantaged were increasingly heavier than the non-disadvantaged.   However, it does not appear that this increasing effect of disadvantage can be attributed to differences in the shared genetic-environmental factors of these mothers and their children.

These results are somewhat puzzling when contrasted with the quantile regression results in the previous section.  We expected that since heavier children show higher parent-child BMI elasticities, and disadvantaged children tend to be heavier, that we would find higher parent child elasticities for the disadvantaged.  However, the quantile regression results suggest that common genetic-environment factors play a larger role for heavier children, and that this difference is better captured by one's place in the log BMI distribution itself than by coarse measures of disadvantage that are simply correlated with one's place in the BMI distribution.

## VI. Common Parent-Child Environmental Factors

The above analysis suggests that, overall, common-parent child factors are becoming increasingly important in determining weight outcomes. Ideally, we would like to be able to observe the factors that determine BMI – caloric in-take and expenditure – and correlate these underlying factors for both adults and children.  We would like to be able to do this for different measures of disadvantage in order to see whether it is differences in food consumed or exercise that determines differences in BMI across disadvantaged groups.  Further, we would ideally be able to observe the environments in which the advantaged and disadvantaged live and examine whether these differences affect caloric in-take or expenditure.  In this section, we present some evidence on these environmental factors, while also discussing the many challenges in examining these direct determinants of BMI.

*A. Caloric In-take by Disadvantage*

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[16] There continues to be no significant interaction effect when using quantile regression.

Since we know that BMI is higher for disadvantaged groups, it seems likely we should find that caloric in-take is higher for these groups or energy expended is lower. However, it is very difficult to find evidence of these differences. First, over time, relatively small caloric imbalances can result in a relatively large amount of weight gain. In fact, Cutler, Glaeser and Shapiro (2003) estimate that the weight gain observed for the median adult male between the early 1970s and the early 2000s can be entirely accounted for by just a daily 150-calorie imbalance. Thus, just one extra 12 ounce can of non-diet soda per day is sufficient to cause the increase in the median man's weight gain seen in recent years. Even fewer excess calories can explain the increase in obesity for children (Schanzenbach, 2005). Second, good data on caloric intake and expenditure are difficult to obtain. While food diary data (as is available in the NHANES), is generally considered fairly reliable, the caloric intake is still likely measured with error. Caloric expenditure data tends to be more problematic, since accurately describing the intensity of exercise can be difficult. As a result, analyses of caloric imbalance will likely have difficulty precisely estimating differences in calorie intake that are as small as those required to explain a great deal of weight gain over time.

Nonetheless, we will use the food diary data from the NHANES to try to get a feel for eating patterns over time and by disadvantage status. We will investigate caloric intake overall, and from a set of food types that may be markers of better or worse nutritional habits. In particular, we focus mainly on food away from home, fast food and fruits and vegetables. Table 5 presents a regression of log(calories+1) for each of these food types on the income-to-poverty ratio along with a cubic in age fully interacted with gender.[17] Thus, these models are identical to those shown in the third panel of Table 1, but using calories instead of BMI in the

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[17] We add one to all of the calorie counts so as not to have to drop observations with no consumption in a specific food group. See the Data Appendix for further details.

dependent variable. The regressions using total calories show an inconsistent pattern in the correlation between the income-to-poverty line ratio and total calories. In the first wave, the relationship is positive, but insignificant. In the second wave, it is positive and significant – meaning that children from wealthier families report consuming more calories. In the third wave it is negative and insignificant; in the fourth, it is very small and insignificant. Thus, if anything, we observe a change between NHANES 2 and 3 in which disadvantage no longer implies fewer calories, but rather more calories, but that in more recent years, all children have similar caloric intake.

While total calories (if well-measured) are what ought to matter for weight gain or loss, we also examine calorie in-take from fruits and vegetables and calories consumed away from home. Consumption of fruits and vegetables may be viewed as a "marker" of healthful eating habits, and thus may give us some insight into how disadvantage may correlate with poor nutrition. Similarly, we examine calories from food consumed away from home. One popular explanation for the rise in obesity is that consumption of food from fast food and other restaurants has increased. The notion is that with professionally prepared food individuals are less aware of the calorie content of what they are eating, have less control over portion size, and may find the food more palatable, than food prepared at home. Note that French fries are included in this measure of fruits and vegetables – for some of the years we can separate out certain food types and food venders and we will do that when we examine parent-child correlations in calorie consumption.

For now, note that the second column of Table 5 indicates that in the first three NHANES, higher income is associated with significantly more calories from fruits and vegetables. In the last two NHANES, higher incomes are associated with significantly more

calories consumed away from home.  Thus, there is some evidence that advantaged and disadvantaged children consume calories from different sources, and that these relationships may have changed over time. However, with these broad measures of caloric in-take it is hard to see how this may translate into differences in BMI.

*B. Mother-Child Calorie Elasticies*

Having seen that the intra-household correlation in BMI has increased over time, but with no real pattern by disadvantaged, we now turn to looking more closely at intra-household correlations in eating patterns.  With NHANES 3 and 4, we have enough data to look at children matched to an adult household member.  The matched data looks very similar to the total data that are both available for NHANES 3.  Appendix Table 2 repeats the relevant panel of Table 5, and then replicates it using the matched data.  Additionally, the more detailed food codes allow us to remove French fries and potato chips from the fruit and vegetable category, and to identify fast food consumption.  The final columns, then, reflect these two additional food groups.  Note that without French fries and chips, the effect of disadvantage on fruit and vegetable consumption becomes more important.  Similarly, while the more advantaged do eat more fast food, the effect is much smaller than for the broader food-away-from-home category, and is not significantly different from zero.

Having seen that the matched data is similar to the full data, we now use NHANES 3 and 4 data to estimate intra-household calorie elasticities in a manner identical to those calculated for BMI in Table 2. For each of the five food groups (i.e. total calories, fruit and vegetable calories, calories away from home, fruit and vegetable calories without fries and chips, and fast food calories), we regress the child's log(calories+1) on first the mother's log(calories+1), then the father's log (calories+1), and then both.  The results are in Table 6.  For total calories, the results

show patterns that are similar to the BMI estimates. That is, both the mother and the father matter; estimates are similar whether the parents are included together or separately; and there is no significant difference between mother and father. Additionally, unlike for the BMI elasticity, the point estimate of the calorie elasticity increases somewhat between the two later periods.

For the specific food groups, the mother elasticity is always larger, and is sometimes significantly different from the father elasticity when included together. In the earlier period, while mother is not significantly different from father for all fruits and vegetables, once we remove French fries and chips there is a marginally statistically significant difference. When looking specifically at fast food, the maternal elasticity of 0.319 is twice the size of the paternal elasticity of 0.151. This finding is consistent with a story where fathers have opportunities to eat fast food that do not include their children -- for example, lunch on a work day. In the second period, however, only the elasticity of calories from fast food shows a statistically significant difference between the mother and father.

Some interesting patterns emerge when comparing the food subgroup results across periods. For the main markers of poor diet – food away from home and fast food, the calorie elasticity increases over time, while for both measures of fruits and vegetables, the calorie elasticity decreases, as does the total calorie elasticity. This pattern holds for both the mothers and the fathers. Recall that the data with which we can calculate parent-child calorie elasticities come from only the last two NHANES. In these data, the parent-child BMI elasticity was higher than in the earlier two periods, but about the same between these two years. Thus, it is not necessarily a conflict that we do not see an increase in, for example, the total calorie elasticity between these two years.

While it appears that both mother's and father's eating patterns influence children, mothers seem to play the dominant role. Thus, as was the case with the BMI elasticities, we will just focus on the maternal elasticity when investigating the role of disadvantage.

Table 7 is parallel to the bottom panel of Table 3, in that it uses the income-to-poverty ratio as the measure of disadvantage, and interacts it with the maternal log(calorie+1) measure. Here, each column is a different food group. Looking first at column (1), we see that the elasticity of total calories is larger for the more disadvantaged, but this difference is only marginally statistically significant in the last period. This point estimate may be due to the fact that by this time period advantaged mothers are more likely to work (and work longer hours) than disadvantaged mothers (Anderson et. al., 2003). Advantaged mothers may thus be spending more time away from their children than disadvantaged mothers, leading to a lower elasticity in calories consumed. Overall, though, as was the case with Table 3, there little evidence of a statistically significant difference in the mother-child calorie elasticity for the disadvantaged.

*C. Other Environmental Factors*

Above we examined reported caloric in-take patterns for insight into the shared environment between mothers and their children that might affect their BMI. This intake is clearly a small subset of the complex environmental factors that affect body mass, however. One would clearly like information on calories expended as well as calories consumed. The measurement problems that attend collecting information on calories consumed are increased for calories expended – not only does one need information on the number of minutes spent running, for example, but the intensity of that activity. Carefully controlled medical studies are able to document how calories expended and consumed affect BMI, however these necessarily give us

little insight into who chooses to participate in different caloric in-take and expenditure behavior and why they make those different decisions.

In order to explain differences in body mass between the advantaged and disadvantaged, many social science researchers have turned to documenting differences in the living environment that each group faces. We examined county business pattern data for 1980-1986 and 1988-1994 (years which roughly correspond to the second and third waves of the NHANES data – the period when we see an initial jump in children's obesity rates) in order to see whether the environments in which the disadvantaged live have changed differentially from the environments in which the advantaged live in ways that are likely to affect body mass. The results showed no consistent pattern between changes in the availability of restaurants, grocery stores or recreation outlets and various measures of disadvantage.

There are several problems with this approach. First, the establishment definitions are coarse, for example many types of restaurants are grouped together. Growth in the count of restaurants in an advantaged area may represent growth in establishments that cater to a health conscious crowd, while in a disadvantaged area it may represent growth in fast food restaurants. Thus, it is perhaps not surprising that we found no consistent pattern in changes in establishment types over time by county level measures of disadvantage.

A second problem is more fundamental, and would be a problem even if we had very detailed and accurate measures of establishment type linked to specific geographic areas. Ideally, we would like to examine whether an exogenous increase in concentration of fast food restaurants, for example, increases caloric in-take and increases body mass. Similarly, we would like to know whether an exogenous increase in recreational space, for example, increases exercise, and reduces weight. Without that exogenous variation, it is very hard to say whether

fast food restaurants cause people to eat unhealthful foods, or whether restaurant owners simply open franchises near where they perceive their customers to live – e.g., obese individuals would likely consume fast food whether or not there is one around the corner, but franchise owners, in an attempt to get customers into their *particular* store, may try to locate very close to the customers. Given the importance for policy purposes of understanding whether location of fast food restaurants, for example, has a *causal* impact on obesity, research with a design that credibly answers this question is a high priority.

## VII. Conclusion

Designing good policies to affect childhood obesity will require an understanding of how the environment that children face is related to their body mass. Although our attempts to directly measure changes in the food and exercise environments faced by advantaged and disadvantaged children were not very fruitful, our earlier results on child-parent BMI elasticity indirectly suggest that shared environment/genetics do play an important role. In fact, we find that the shared mother-child component of BMI became increasingly important over this time period, reflected in the increase in the mother-child BMI elasticity. However, if the only determinants of children's body mass were their genes and the environment they share with their parents, we would expect to find that increases in parents' BMI can go a long way toward explaining the increases we have seen in children's BMI over the last few decades. Instead, on average, based on our estimates of the mother-child BMI elasticity, the 13.7 percent increase in mothers' average BMI over the 30-year period we examine can explain at most 40 percent of the 7.3 percent increase in children's average BMI.

The title of this paper asks whether in determining children's obesity, "nurture" is trumping "nature" and whether that is different for disadvantaged children. While we find that common parent-child factors are becoming more important in determining children's weight outcomes, this result is consistent with *both* an increasing role for "nurture" *and* an increasing role for "nature." For example, there is either an increasing role for the shared environments that affect both parents' and children's body mass, or an increasing role of the overall environment for certain genetic profiles (which may be shared by parents and their children). The former situation might fit what we loosely mean by "nurture" – parental choices that involve such things as increasingly sharing meals and snacks made up of unhealthful foods or sharing more sedentary, rather than vigorous, activities. The latter situation might fit what we loosely think of as "nature"-- or at least how one's natural susceptibility towards obesity interacts with one's environment -- and might include eating increasingly available unhealthful foods in separate environments (say at school for children, at work for parents) that have similar effects on those with the same genetic susceptibility to weight gain. Note that Anderson and Butcher (2006b) finds evidence for a different effect of "environment" on those with a genetic susceptibility toward weight gain since that study finds that the availability of junk food in schools results in higher weights only for those adolescents who have an overweight parent.

Overall, it appears that the parent-child elasticity is similar for advantaged and disadvantaged children, for both it has increased over time, but, as noted above, that changes in parents' BMI still does not explain most of the change in children's BMI. Regardless of whether one wants to interpret the finding that the elasticity has increased as evidence for increasing importance of parental choices for children ("nurture") or the increasing importance of the environment for both children and parents who share a similar genetic susceptibility to

weight gain ("nature" interacting with the environment), there appear to be different factors that affect parents' and children's body-mass. Child-specific environments like schools, childcare facilities, and playgrounds may play an important role in determining children's weight outcomes. This is an important implication since it suggests policies targeted at changing children's environment outside the family, rather than solely targeting parental choices, may be an effective way to address childhood obesity.

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**Data Appendix**

*A. Variable Definitions and Parent-Child Match*

All of the data used in the paper are from the National Health and Nutrition Examination Survey (NHANES). Most of these data are publicly available from the National Center for Health Statistics (NCHS), Centers for Disease Control and Prevention (CDC) at their website (http://www.cdc.gov/nchs/nhanes.htm). We use four waves of this survey. The first, known as NHANES 1 was collected between 1971 and 1975, the second, NHANES 2, was collected between 1976 and 1980 while the third, NHANES 3, was collected between 1988 and 1994. With sampling weights, these data are representative of the U.S. population at the midpoint of the survey years. For each of these three waves, we extract publicly available information from the youth examination files on professionally measured body mass index (BMI) for all children age 2 to 11. For each of these children reported BMI is also publicly available for each of their resident parents, along with basic demographics such as age, gender and race/ethnicity. For an adult household reference person, we also have completed education in years, and for the family as a group we have the ratio of family income to the poverty line for a family of that size in that year.

Also publicly available for the children is information from a 24-hour recall food diary. Each food item consumed is separately recorded, giving the food category, along with key nutritional information such as calories. The exact food codes used differ slightly over the waves, but we can always identify whether the food item was consumed at home or away, and the major food group (e.g. fruits and vegetables; dairy products, etc.). For consistency across waves, we choose to look at total calories, calories from the major category, "all fruits and vegetables" and calories consumed away from home. For each individual, then, we aggregate

the calories from food items in those categories to obtain a per-person measure of total calories consumed, calories consumed from fruits and vegetables, and calories consumed away from home. We then add one to each of these aggregates. While one calorie is fairly incidental in an individual's diet, this addition allows us to take logs of our calories subgroups, even if an individual did not eat any calories from that group in the 24 hours captured by the diary.

The fourth wave is slightly different from the first three. First, what we will refer to as NHANES 4 is actually made up of 3 separate sub-waves named NHANES 1999-2000, NHANES 2001-2002 and NHANES 2003-2004. These data can pooled and appropriate six-year sample weights computed such that the data are also representative of the U.S. population at the midpoint of the survey years. As with the other waves, we extract publicly available data on professionally measured BMI for children age 2 to 11, along with basic demographics such as age, gender and race/ethnicity. Also available for each child is the income-to-poverty line ratio for the child's family. Unfortunately, the reported parental BMI is not available in these data. As a result, we must create a "matched" data set using a confidential household identifier to compute intra-household BMI elasticities. Since NHANES 3 is also large enough to create a similar matched data set (using the publicly available household identifier), we can evaluate the importance of this alternate approach.

Based on results from NHANES 3, which determined that 89 percent of households had only one adult (age 20-50) male and 88 percent had only one adult female, we settled on a very simple matching algorithm. For each household, the adult females age 20-50, adult males age 20-50 and children age 2-11 were separated out and sorted by age. The first adult female in the household was then assigned to be the child's "mother" and the first adult male was assigned to be the "father." Based on the reported parental information in NHANES 3, it appeared that only

4 percent of the matched mothers had a BMI and age that were inappropriate. As a result models identical to those in Tables 2 and 3 run using the matched NHANES 3 data looked very much like those presented using the larger data set with reported parental BMI.

Unfortunately, with NHANES 4, we cannot make this same comparison, but again given the large number of households with only one adult female or adult male, we feel confident that the results are reliable. A few other differences in NHANES 4 are potentially problematic. First, education was not reported for the household reference person. Thus, we must use the matched "mother" and "father" to determine the reference person's education. When we have education for a "father" in the household, we choose that as the reference person's education. Otherwise, we use the "mother" in the household. Second, in NHANES 4, education is not recorded as years completed, but rather is categorical – less than high school, high school graduate, beyond high school. To maintain consistency with the previous waves, we code these categories as 10 years of education, 12 years, and 14 years, respectively. The results from this approximation to continuous years of education do not differ substantively from including the categorical dummies. Appendix Table 1 presents summary statistics for the variables used in our main analyses for all four waves of the NHANES.

As noted above, the 24-hour food recall diaries (used in our secondary analyses) are available for everyone, making it possible to examine the effect of disadvantage on eating patterns for all waves of the NHANES. However, within-household correlations can only be computed when children are matched to adults in the household. While we can follow the matching procedures outlined above for NHANES 1 and 2, the resulting sample sizes are too small to provide useful estimates. Thus, we limit our in-depth analysis of intra-household eating behaviors to the NHANES 3 and 4.
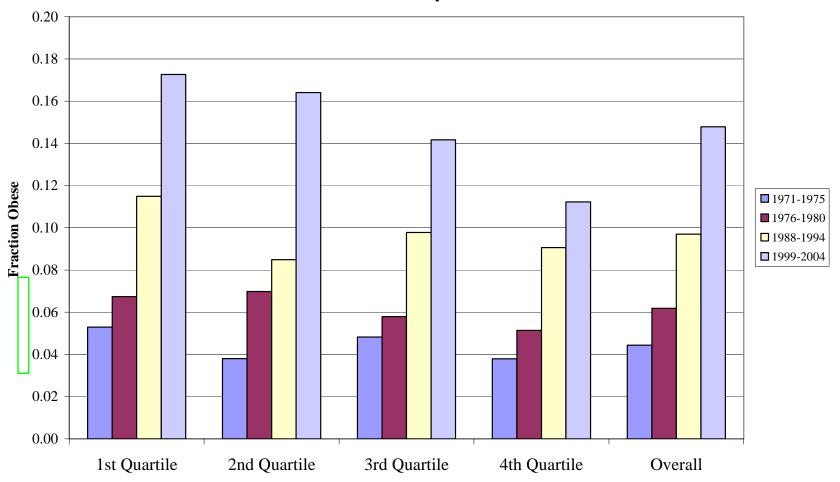
Across these two waves, we try to maintain as much consistency in the definition of food groups as possible. One area where this is not entirely possible is in the definition of fast food calories. In NHANES 3, calories are coded based on the item being described using a set of detailed codes for branded products (e.g. Burger King Whopper, Wendy's Frosty, etc. Thus, "fast food calories" implies having consumed any kind of food from a fast food restaurant, no matter where it was consumed. .In NHANES 1999-2000, we can identify that a food item was eaten at a fast food establishment, but if it was brought home and eaten, it will not be coded as "fast food." In NHANES 2003-2004, we can identify that a food item was obtained from a fast food restaurant, no matter where it was consumed, while in NHANES 2001-2002 we cannot identify fast food at all. As a result, for NHANES 4, "fast food calories" implies calories from food items that were either eaten at a fast food restaurant in the early years or obtained from a fast food restaurant in the later years.

*B. Implications for Estimated Elasticity of Changing Measurement Error in BMI*

While it appears that between the 1971-1980 and 1988-2004 periods there was a marked increase in the parent-child BMI elasticity, a concern with this conclusion may be that, in fact, the true elasticity has not changed, but rather the amount of measurement error in BMI has fallen, allowing us to estimate this true elasticity more precisely (e.g. the signal-to-noise ratio has risen). If one makes the assumption of a constant true elasticity, one can use the estimated elasticities, along with the variance in log mothers' BMI to estimate what combination of constant elasticity and error variance would be consistent with these estimated elasticities. The result is an implausibly large error variance of 0.030 combined with a constant elasticity of 0.52. Note that in NHANES 3, since we have both reported and measured maternal BMI, we can calculate an alternate measure of the error variance (and thus of the true elasticity) from a
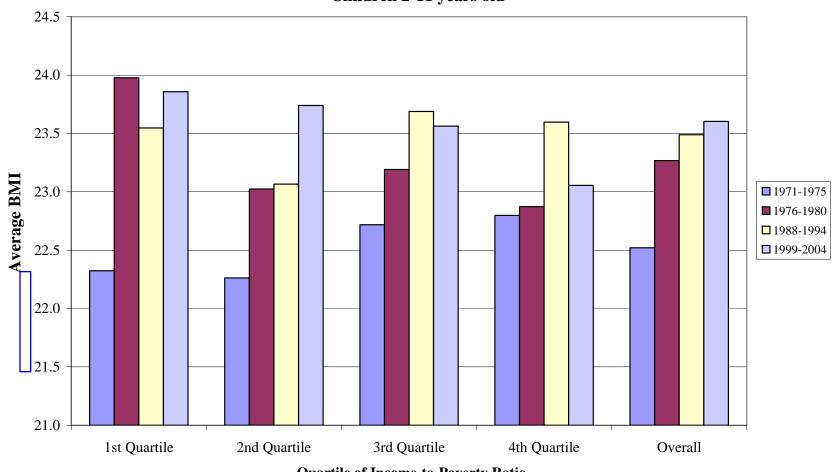
regression of the measured BMI on the reported BMI. This exercise implies a true elasticity of 0.23, with an error variance of just 0.004 in NHANES 3. Thus, we conclude that it is very unlikely that a simple measurement error story is behind the increase in the estimated elasticity, and that there has indeed been an increase in the true elasticity.

**Figure 2: Fraction Obese by Year and Quartile of Income-to-Poverty Ratio: Children 2-11 years old**

**Figure 3: Average BMI Among the Obese by Year and
Quartile of Income-to-Poverty Ratio:
Children 2-11 years old**

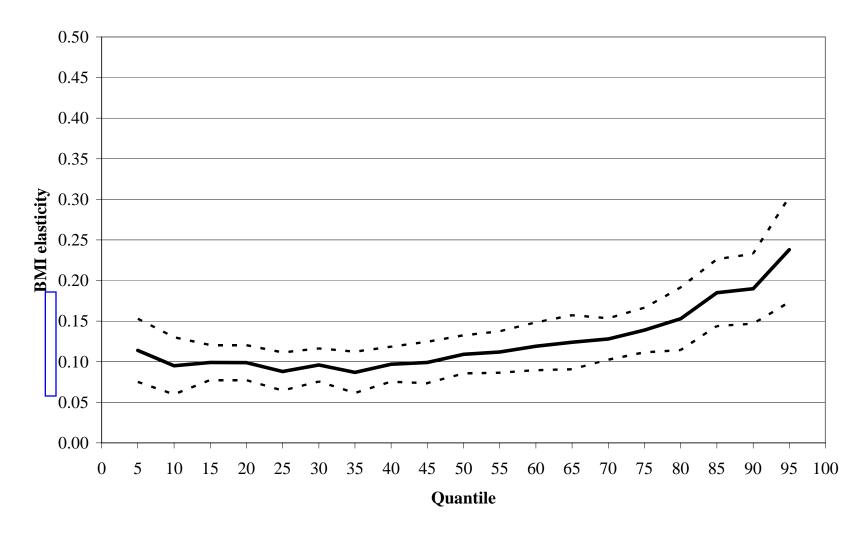**Figure 4: NHANES 1 Mother-Child log(BMI) Relationship**

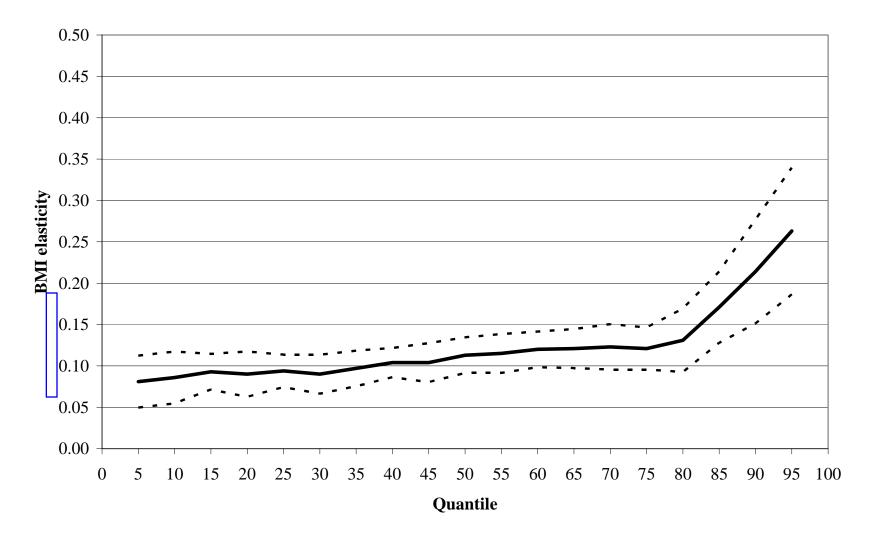**Figure 5: NHANES 2 Mother-Child log(BMI) Relationship**
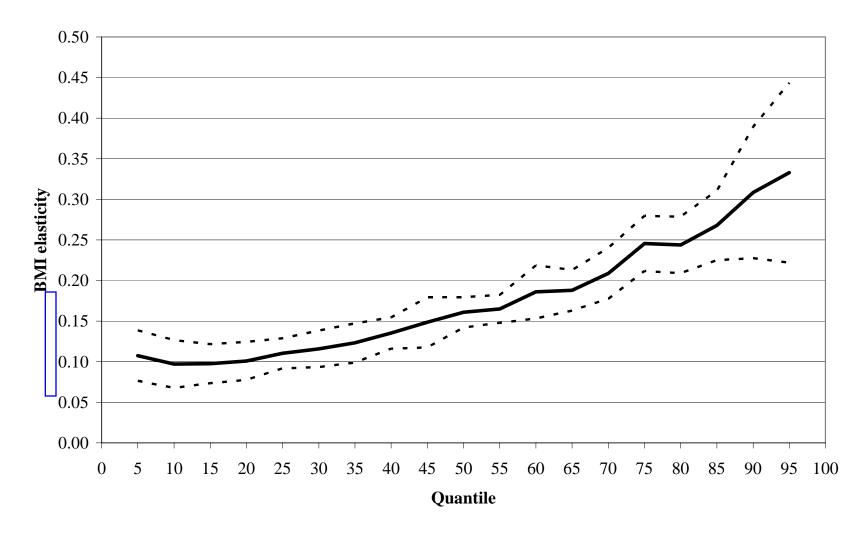
**Figure 6: NHANES 3 Mother-Child log(BMI) Relationship**

**Table 1: Disadvantage and Children's log BMI**

| Race on log BMI | (1) NHANES 1: 1971-75 | (2) NHANES 2: 1976-80 | (3) NHANES 3: 1988-94 | (4) NHANES 4: 1999-2004 |
|---|---|---|---|---|
| Black | -0.008 | 0.001 | 0.008 | 0.021 |
| | (0.006) | (0.007) | (0.006) | (0.006)** |
| Hispanic | 0.016 | 0.034 | 0.037 | 0.035 |
| | (0.009) | (0.010)* | (0.006)** | (0.006)** |
| Other Non-white | 0.006 | -0.016 | 0.004 | -0.002 |
| | (0.020) | (0.014) | (0.012) | (0.013) |
| Observations | 4374 | 4781 | 7694 | 5586 |
| R-squared | 0.16 | 0.18 | 0.17 | 0.20 |

| Education on log BMI | NHANES 1: 1971-75 | NHANES 2: 1976-80 | NHANES 3: 1988-94 | NHANES 4: 1999-2004 |
|---|---|---|---|---|
| Highest Grade for Reference Person | -0.002 | -0.004 | -0.003 | -0.006 |
| | (0.001)** | (0.001)** | (0.001)** | (0.002)** |
| Observations | 4260 | 4688 | 7582 | 2869 |
| R-squared | 0.16 | 0.18 | 0.17 | 0.18 |

| Poverty on log BMI | NHANES 1: 1971-75 | NHANES 2: 1976-80 | NHANES 3: 1988-94 | NHANES 4: 1999-2004 |
|---|---|---|---|---|
| Income/Poverty Line Ratio | 0.002 | -0.002 | -0.006 | -0.008 |
| | (0.002) | (0.002) | (0.002)** | (0.002)** |
| Observations | 4276 | 4637 | 7095 | 5142 |
| R-squared | 0.16 | 0.17 | 0.17 | 0.20 |

| All 3 on log BMI | NHANES 1: 1971-75 | NHANES 2: 1976-80 | NHANES 3: 1988-94 | NHANES 4: 1999-2004 |
|---|---|---|---|---|
| Black | -0.009 | -0.003 | 0.003 | 0.004 |
| | (0.007) | (0.008) | (0.006) | (0.009) |
| Hispanic | 0.011 | 0.026 | 0.024 | 0.033 |
| | (0.009) | (0.011)* | (0.008)** | (0.009)** |
| Other Non-white | 0.000 | -0.018 | -0.003 | 0.016 |
| | (0.024) | (0.014) | (0.012) | (0.019) |
| Highest Grade for Reference Person | -0.002 | -0.005 | -0.002 | -0.001 |
| | (0.001)* | (0.001)** | (0.001)* | (0.003) |
| Income/Poverty Line Ratio | 0.005 | 0.006 | -0.002 | -0.007 |
| | (0.002)* | (0.003)* | (0.003) | (0.003)* |
| Observations | 4175 | 4567 | 7011 | 2646 |
| R-squared | 0.16 | 0.19 | 0.17 | 0.19 |

Notes: All models also include a cubic in child's age fully interacted with gender. BMI is measured. Standard errors are in parentheses. Statistical significance level: *10%, **5%, ***1%. Sample is limited to children age 2-11.

**Table 2: Intergenerational BMI Elasticity**

| NHANES 1: 1971-75 | (1) | (2) | (3) |
|---|---|---|---|
| log Mother's BMI | 0.142 | | 0.131 |
| | (0.014)** | | (0.015)** |
| log Father's BMI | | 0.141 | 0.126 |
| | | (0.016)** | (0.016)** |
| Observations | 3918 | 3609 | 3552 |
| R-squared | 0.20 | 0.19 | 0.22 |
| F test: Mom=Dad | | | 0.04 |
| Prob > F | | | 0.843 |

| NHANES 2: 1976-80 | (1) | (2) | (3) |
|---|---|---|---|
| log Mother's BMI | 0.141 | | 0.120 |
| | (0.016)** | | (0.018)** |
| log Father's BMI | | 0.180 | 0.152 |
| | | (0.021)** | (0.021)** |
| Observations | 4402 | 4173 | 4116 |
| R-squared | 0.22 | 0.22 | 0.24 |
| F test: Mom=Dad | | | 1.05 |
| Prob > F | | | 0.305 |

| NHANES 3: 1988-94 | (1) | (2) | (3) |
|---|---|---|---|
| log Mother's BMI | 0.206 | | 0.178 |
| | (0.020)** | | (0.022)** |
| log Father's BMI | | 0.208 | 0.150 |
| | | (0.023)** | (0.022)** |
| Observations | 6555 | 6016 | 5817 |
| R-squared | 0.24 | 0.22 | 0.26 |
| F test: Mom=Dad | | | 0.66 |
| Prob > F | | | 0.415 |

| NHANES 4: 1999-2004 | (1) | (2) | (3) |
|---|---|---|---|
| log Mother's BMI | 0.196 | | 0.188 |
| | (0.019)** | | (0.025)** |
| log Father's BMI | | 0.201 | 0.126 |
| | | (0.030)** | (0.029)** |
| Observations | 2249 | 1770 | 1394 |
| R-squared | 0.27 | 0.23 | 0.30 |
| F test: Mom=Dad | | | 1.95 |
| Prob > F | | | 0.163 |

Notes: Dependent variable is the log of the child's measured BMI. Parental BMI data are reported for NHANES 1-3, and measured and matched for NHANES 4 (see data appendix for details). All models also control for race, highest grade completed by the household reference person, the household income-to-poverty ratio, and a cubic in child's age fully interacted with gender. Standard errors are in parentheses. Statistical significance level: *10%, ** 5%, ***1%.

**Table 3: Interactions between Disadvatage and Mother's BMI**

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| **Race on log BMI** | **NHANES 1: 1971-75** | **NHANES 2: 1976-80** | **NHANES 3: 1988-94** | **NHANES 4: 1999-2004** |
| log Mother's BMI | 0.155 | 0.146 | 0.211 | 0.190 |
| | (0.016)** | (0.019)** | (0.031)** | (0.025)** |
| Black | 0.156 | 0.036 | 0.162 | -0.064 |
| | (0.122) | (0.120) | (0.112) | (0.126) |
| Hispanic | 0.181 | 0.004 | 0.040 | 0.130 |
| | (0.172) | (0.238) | (0.127) | (0.164) |
| Other race | -0.569 | 0.379 | -0.155 | -0.529 |
| | (0.435) | (0.189)* | (0.177) | (0.316) |
| log Mother's BMI*Black | -0.054 | -0.015 | -0.053 | 0.014 |
| | (0.035) | (0.038) | (0.035) | (0.038) |
| log Mother's BMI*Hispanic | -0.056 | 0.006 | -0.010 | -0.026 |
| | (0.054) | (0.075) | (0.040) | (0.049) |
| log Mother's BMI*Other race | 0.180 | -0.125 | 0.045 | 0.173 |
| | (0.140) | (0.059)* | (0.056) | (0.098) |
| Observations | 3918 | 4402 | 6555 | 2249 |
| R-squared | 0.20 | 0.22 | 0.24 | 0.27 |

| | | | | |
|---|---|---|---|---|
| **Education on log BMI** | **NHANES 1: 1971-75** | **NHANES 2: 1976-80** | **NHANES 3: 1988-94** | **NHANES 4: 1999-2004** |
| log Mother's BMI | 0.172 | 0.177 | 0.172 | 0.371 |
| | (0.053)** | (0.060)** | (0.077)* | (0.135)** |
| Highest grade for Reference Person | 0.008 | 0.006 | -0.011 | 0.048 |
| | (0.015) | (0.016) | (0.022) | (0.036) |
| log Mother's BMI*Highest grade | -0.003 | -0.003 | 0.003 | -0.014 |
| | (0.005) | (0.005) | (0.007) | (0.011) |
| Observations | 3918 | 4402 | 6555 | 2249 |
| R-squared | 0.20 | 0.22 | 0.24 | 0.27 |

| | | | | |
|---|---|---|---|---|
| **Poverty on log BMI** | **NHANES 1: 1971-75** | **NHANES 2: 1976-80** | **NHANES 3: 1988-94** | **NHANES 4: 1999-2004** |
| log Mother's BMI | 0.119 | 0.101 | 0.171 | 0.217 |
| | (0.025)** | (0.030)** | (0.029)** | (0.031)** |
| Income/Poverty Line Ratio | -0.034 | -0.065 | -0.055 | 0.031 |
| | (0.036) | (0.045) | (0.051) | (0.036) |
| log Mother's BMI* Income/Poverty Line | 0.013 | 0.023 | 0.018 | -0.010 |
| | (0.012) | (0.014) | (0.016) | (0.011) |
| Observations | 3918 | 4402 | 6555 | 2249 |
| R-squared | 0.20 | 0.22 | 0.24 | 0.27 |

Notes:  See notes to Table 2.

**Table 4: Mother-Child BMI Elasticity by Group**

|  | (1)<br>NHANES 1:<br>1971-1975 | (2)<br>NHANES 2:<br>1976-1980 | (3)<br>NHANES 3:<br>1988-1994 | (4)<br>NHANES 4:<br>1999-2004 |
|---|---|---|---|---|
| White | 0.155 | 0.146 | 0.211 | 0.190 |
| Black | 0.101 | 0.131 | 0.158 | 0.204 |
| Hispanic | 0.336 | 0.150 | 0.251 | 0.320 |
| Other race | -0.414 | 0.525 | 0.056 | -0.339 |
| Reference Person's Education = 10 | 0.142 | 0.147 | 0.202 | 0.231 |
| Reference Person's Education = 12 | 0.136 | 0.141 | 0.208 | 0.203 |
| Reference Person's Education = 16 | 0.124 | 0.129 | 0.220 | 0.147 |
| Income-to-Poverty Ratio = 1st Quartile Cutoff | 0.135 | 0.126 | 0.188 | 0.207 |
| Income-to-Poverty Ratio = 2nd Quartile Cutoff | 0.144 | 0.142 | 0.206 | 0.198 |
| Income-to-Poverty Ratio = 3rd Quartile Cutoff | 0.155 | 0.165 | 0.226 | 0.181 |

Notes: Calculations based on elasticities reported in Table 4.

**Table 5: Calorie Intake Among Children Age 2-11**

| | (1) | (2) | (3) |
|---|---|---|---|
| **NHANES 1: 1971-75** | log(Total Calories +1) | log(Calories from Fruits & Vegetables +1) | log(Calories Eaten Away from Home +1) |
| Income/Poverty Line Ratio | 0.006 | 0.080 | 0.002 |
| | (0.005) | (0.022)** | (0.037) |
| Observations | 4092 | 4092 | 4092 |
| R-squared | 0.11 | 0.01 | 0.09 |
| **NHANES 2: 1976-80** | log(Total Calories +1) | log(Calories from Fruits & Vegetables +1) | log(Calories Eaten Away from Home +1) |
| Income/Poverty Line Ratio | 0.011 | 0.081 | 0.065 |
| | (0.006)* | (0.030)** | (0.052) |
| Observations | 4466 | 4466 | 4466 |
| R-squared | 0.14 | 0.01 | 0.05 |
| **NHANES 3: 1988-94** | log(Total Calories + 1) | log(Calories from Fruits & Vegetables +1) | log(Calories Eaten Away from Home +1) |
| Income/Poverty Line Ratio | -0.009 | 0.066 | 0.284 |
| | (0.006) | (0.025)** | (0.040)** |
| Observations | 7046 | 7046 | 7046 |
| R-squared | 0.16 | 0.01 | 0.09 |
| **NHANES 4: 1999-2004** | ln(Total Calories + 1) | Ln(Calories from Fruits & Vegetables +1) | Ln(Calories eaten away from home +1) |
| Income/Poverty Line Ratio | 0.000 | -0.026 | 0.357 |
| | (0.006) | (0.024) | (0.042)** |
| Observations | 3099 | 3099 | 3099 |
| R-squared | 0.11 | 0.02 | 0.08 |

Notes: Other covariates include a cubic in age fully interacted with gender. BMI is measured. Standard errors are in parentheses. Statistical significance level: *10%. **5%. ***1%.

**Table 6: Relationship between Parent and Child Calorie Intake**

| | NHANES 3 (1988-1994) | | | NHANES 4 (1999-2004) | | |
|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | (6) |
| **log(Total Calories +1)** | | | | | | |
| log(Mom's Total Calories) | 0.101 | | 0.101 | 0.150 | | 0.116 |
| | (0.021)** | | (0.030)** | (0.029)** | | (0.037)** |
| log(Dad's Total Calories) | | 0.147 | 0.131 | | 0.120 | 0.071 |
| | | (0.034)** | (0.034)** | | (0.038)** | (0.038) |
| Observations | 3342 | 1859 | 1859 | 1229 | 994 | 748 |
| R-squared | 0.17 | 0.18 | 0.19 | 0.14 | 0.15 | 0.15 |
| F test: Mom=Dad | | | 0.39 | | | 0.70 |
| Prob > F | | | 0.532 | | | 0.403 |
| **log(Calories Eaten Away from Home +1)** | | | | | | |
| log(Mom's Calories Away from Home) | 0.288 | | 0.202 | 0.329 | | 0.292 |
| | (0.027)** | | (0.035)** | (0.034)** | | (0.043)** |
| log(Dad's Calories Away from Home) | | 0.179 | 0.145 | | 0.244 | 0.196 |
| | | (0.042)** | (0.042)** | | (0.040)** | (0.047)** |
| Observations | 3342 | 1859 | 1859 | 1229 | 994 | 748 |
| R-squared | 0.16 | 0.11 | 0.15 | 0.23 | 0.19 | 0.29 |
| F test: Mom=Dad | | | 0.92 | | | 1.73 |
| Prob > F | | | 0.339 | | | 0.189 |
| **log(Calories from Fruits & Vegetables +1)** | | | | | | |
| log(Mom's Fruit/Veg Calories) | 0.246 | | 0.248 | 0.111 | | 0.099 |
| | (0.036)** | | (0.049)** | (0.039)** | | (0.048)* |
| log(Dad's Fruit/Veg Calories) | | 0.237 | 0.179 | | 0.077 | 0.009 |
| | | (0.047)** | (0.049)** | | (0.041) | (0.044) |
| Observations | 3342 | 1859 | 1859 | 1229 | 994 | 748 |
| R-squared | 0.07 | 0.08 | 0.13 | 0.05 | 0.03 | 0.04 |
| F test: Mom=Dad | | | 0.79 | | | 1.67 |
| Prob > F | | | 0.375 | | | 0.197 |
| **log(Calories from Fruits & Vegetables, Less French Fries +1)** | | | | | | |
| log(Mom's Fruit/Veg Calories, No Fries) | 0.263 | | 0.270 | 0.165 | | 0.158 |
| | (0.029)** | | (0.042)** | (0.040)** | | (0.048)** |
| log(Dad's Fruit/Veg Calories, No Fries) | | 0.229 | 0.146 | | 0.132 | 0.080 |
| | | (0.038)** | (0.042)** | | (0.041)** | (0.046) |
| Observations | 3342 | 1859 | 1859 | 1229 | 994 | 748 |
| R-squared | 0.09 | 0.09 | 0.16 | 0.06 | 0.04 | 0.07 |
| F test: Mom=Dad | | | 2.98 | | | 1.24 |
| Prob > F | | | 0.084 | | | 0.266 |
| **ln(Calories from Fast Food +1)** | | | | | | |
| log(Mom's Fast Food Calories) | 0.375 | | 0.319 | 0.506 | | 0.447 |
| | (0.038)** | | (0.046)** | (0.037)** | | (0.049)** |
| log(Dad's Fast Food Calories) | | 0.229 | 0.151 | | 0.353 | 0.204 |
| | | (0.041)** | (0.035)** | | (0.038)** | (0.042)** |
| Observations | 3342 | 1859 | 1859 | 1229 | 994 | 748 |
| R-squared | 0.19 | 0.10 | 0.21 | 0.29 | 0.18 | 0.35 |
| F test: Mom=Dad | | | 6.99 | | | 9.67 |
| Prob > F | | | 0.008 | | | 0.002 |

Note: Data are for the matched NHANES 3 (1988-94) and NHANES 4 (1999-2004) samples. Other covariates include a cubic in age fully interacted with gender, race, income-to-poverty ratio and head's education level.

**Table 7: Relationship between Calories, Disadvantage and Mother's Calories**

**Panel A: NHANES 3 (1988-1994)**

|  | (1) log (Total Calories + 1) | (2) log (Calories from Fruits & Vegetables +1) | (3) log (Calories Eaten Away from Home +1) | (4) log (Calories from Fruit/Veg, No Fries +1) | (5) log (Calories from Fast Food +1) |
|---|---|---|---|---|---|
| log(Mother's Calories) | 0.162 | 0.224 | 0.310 | 0.267 | 0.360 |
|  | (0.039)** | (0.064)** | (0.042)** | (0.049)** | (0.063)** |
|  |  |  |  |  |  |
| log(Mother's Calories)* Income/Poverty Line Ratio | -0.030 | 0.011 | -0.011 | -0.002 | 0.007 |
|  | (0.019) | (0.030) | (0.019) | (0.021) | (0.026) |
|  |  |  |  |  |  |
| Observations | 3342 | 3342 | 3342 | 3342 | 3342 |
| R-squared | 0.17 | 0.07 | 0.16 | 0.09 | 0.19 |

**Panel B: NHANES 4 (1999-2004)**

|  | (1) log (Total Calories + 1) | (2) log (Calories from Fruits & Vegetables +1) | (3) log (Calories Eaten Away from Home +1) | (4) log (Calories from Fruit/Veg, No Fries +1) | (5) log (Calories from Fast Food +1) |
|---|---|---|---|---|---|
| log(Mother's Calories) | 0.229 | 0.120 | 0.341 | 0.243 | 0.502 |
|  | (0.048)** | (0.064) | (0.059)** | (0.068)** | (0.063)** |
|  |  |  |  |  |  |
| log(Mother's Calories)* Income/Poverty Line Ratio | -0.041 | -0.004 | -0.003 | -0.038 | 0.002 |
|  | (0.019)* | (0.029) | (0.021) | (0.029) | (0.023) |
|  |  |  |  |  |  |
| Observations | 1229 | 1229 | 1229 | 1229 | 1229 |
| R-squared | 0.15 | 0.05 | 0.22 | 0.06 | 0.29 |

Notes: See Table 6.

**Appendix Table 1: Summary Statistics**

| | (1)<br>NHANES 1:<br>1971-75 | (2)<br>NHANES 2:<br>1976-80 | (3)<br>NHANES 3:<br>1988-94 | (4)<br>NHANES 4:<br>1999-2004 |
|---|---|---|---|---|
| BMI | 16.41 | 16.58 | 17.05 | 17.65 |
| | (2.29) | (2.61) | (3.02) | (3.52) |
| Mother's BMI | 24.14 | 23.93 | 24.90 | 27.68 |
| | (4.96) | (4.91) | (5.54) | (6.89) |
| Father's BMI | 28.68 | 25.62 | 26.12 | 27.86 |
| | (5.30) | (3.65) | (4.05) | (5.39) |
| Black | 0.14 | 0.15 | 0.16 | 0.10 |
| | (0.34) | (0.36) | (0.36) | (0.30) |
| Hispanic | 0.07 | 0.08 | 0.08 | 0.19 |
| | (0.25) | (0.27) | (0.28) | (0.39) |
| Other race | 0.01 | 0.03 | 0.10 | 0.05 |
| | (0.10) | (0.16) | (0.30) | (0.23) |
| Female | 0.49 | 0.49 | 0.49 | 0.49 |
| | (0.50) | (0.50) | (0.50) | (0.50) |
| Highest grade of reference person | 11.54 | 11.87 | 12.60 | 12.49 |
| | (3.16) | (3.25) | (3.05) | (1.61) |
| Income/Poverty line ratio | 2.12 | 1.99 | 2.21 | 2.35 |
| | (1.22) | (1.09) | (1.42) | (1.55) |
| Observations | 4092 | 4594 | 7124 | 2870 |

Note: Standard deviations in parentheses.

**Appendix Table 2: Comparing Reported and Matched NHANES 3 Samples**

| | (1) | (2) | (3) | (4) | (5) |
|---|---|---|---|---|---|
| **NHANES III: Overall Sample** | log (Total Calories + 1) | log (Calories from Fruits & Vegetables +1) | log (Calories Eaten Away from Home +1) | log (Calories from Fruit/Veg. No Fries +1) | log (Calories from Fast Food +1) |
| Income/Poverty Line Ratio | -0.009 | 0.066 | 0.284 | 0.117 | 0.035 |
| | (0.006) | (0.025)** | (0.040)** | (0.026)** | (0.028) |
| Observations | 7046 | 7046 | 7046 | 7046 | 7046 |
| R-squared | 0.16 | 0.01 | 0.09 | 0.01 | 0.00 |
| **NHANES III: Matched Sample** | log (Total Calories + 1) | log (Calories from Fruits & Vegetables +1) | log (Calories Eaten Away from Home +1) | log (Calories from Fruit/Veg. No Fries +1) | log (Calories from Fast Food +1) |
| Income/Poverty Line Ratio | -0.002 | 0.039 | 0.251 | 0.081 | 0.044 |
| | (0.010) | (0.043) | (0.059)** | (0.042) | (0.043) |
| Observations | 3365 | 3365 | 3365 | 3365 | 3365 |
| R-squared | 0.15 | 0.00 | 0.07 | 0.01 | 0.00 |

Notes: See Table 6.