Food for Thought:

The Cognitive Effects of Childhood Malnutrition in the United States

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Abstract:

The U.S. faces two types of childhood malnutrition – the prevalence of overweight children has increased dramatically over the past two decades and the degree of underweight has been unacceptably high. Both forms of malnutrition create public health problems. Less is known about how childhood over- or underweight affects a child's cognitive functioning. We use data from the children of the NLSY79 to investigate the cognitive consequences of child malnutrition. We use several estimation methods to control for various forms of endogeneity. Our results suggest that malnourished children tend have lower cognitive abilities when compared to well-nourished children.

INTRODUCTION

Health is an important dimension of well-being. Not only is it instrumentally significant through its effects on individual productive capacity and income-earning abilities, but it is also "intrinsically" significant as it affects individuals' capabilities to function in society (Dreze and Sen 1989; Sen 1985, 1987, 1999). Further, as nutrition is an important element of health, nutritional deprivations can have adverse effects on well-being. In light of this, this paper addresses one avenue through which malnutrition can affect capabilities, that is through its effect on cognitive development. In particular, we examine the effect of childhood undernutrition and overnutrition on test scores in the United States.

The United States is currently characterized by the coexistence of two forms of childhood malnutrition. On the one hand, the prevalence of overweight children has increased dramatically over the past two decades[1] (Hedley et al. 2004). On the other hand, the degree of underweight among children has been unacceptably high for such a wealthy country (Polhamus et al. 2003). Both forms of malnutrition create public health problems. For example, an overweight child is more likely to be obese as an adult and has a higher probability of suffering from Type 2-diabetes, high cholesterol, high blood pressure, some types of cancer, and heart disease than is a child who is not overweight (Dietz 1998; Schwimmer et al. 2003). Furthermore, the Surgeon General has linked childhood overweight to social discrimination and depression (U.S. Office of the Surgeon General 2001). At the other end of the weight distribution, children who do not get enough to eat are likely to suffer from stunted growth and hindered mental development (Center on Hunger and Poverty 1998). Paradoxically, although stunted growth (low height-for-age) and wasting (low weight-for-

[1] The term overweight is commonly used to refer to obese children as it is less stigmatizing

height) are more prevalent in poor families (Miller and Korenman 1994), many poor children today are overweight. This has led some researchers to describe the problem as one of "misnourishment," where instead of getting the necessary healthy food that their bodies need, children take in excessive amounts of inexpensive fats and calories (Bhattacharya and Currie 2001). The strain that these consequences of child malnutrition will place on the health-care system are worthy of investigation.

Although the adverse effects of undernutrition on the cognitive functioning of children are well documented in the United States and around the world (Alaimo et al. 2001; Brown and Pollitt 1996; Center for Hunger and Poverty 1998; Gardner and Halweil 2000a and 2000b; Pollitt et al. 1996; and Reid 2000), less is known about the effects of obesity. Although it is indirect, there is some evidence from the medical literature that obese children may suffer cognitive deficits. This follows from deficiencies of certain micronutrients such as zinc, iron and iodine (Taras 2005) for which overweight children are at risk (Nead, et al. 2004). This is exacerbated by changes in food technologies and lifestyles in the United States, and surprisingly throughout much of the developing world, which have resulted in what is referred to as the "nutrition transition" (Popkin et al. 2001). This is the process through which the households have access to, and consume more foods that are not only cheap, energy-rich and convenient, but which are also nutrient-poor. The result of this is increasing rates of obesity and micronutrient deficiencies among children.

In this research, we use data from the children of the National Longitudinal Survey of Youth 1979 cohort (NLSY79) to investigate this potential cognitive consequence of childhood malnutrition. For example, in addition to stunted growth, underweight children are also more likely to experience emotional, academic and behavioral problems than are well-nourished

children (Center on Hunger and Poverty 1998; Jyoti et al. 2005; Kleinman et al. 1998). Furthermore, overweight children may suffer taunting from their peers resulting in low self-esteem, low academic achievement and even behavior problems. Thus concerns over the cost of health care are not the only concerns that policy makers should have as they study the misnourishment of children. Because of the potential link to cognitive development, there are legitimate concerns over the future economic effects of malnutrition-induced diminished productivity (Owens, 1989). These have motivated advocates to emphasize the *public* financial costs of malnutrition (obesity in particular) as a strategy to encourage policy makers to address the issue. The role for public policy follows from the externalities associated with malnutrition. As Paul Krugman (2005) succinctly describes it, "many of these costs fall on taxpayers and on the general insurance-buying public, rather than on the obese individuals themselves."

However, in our analysis, we aim to discover whether there are additional *private* costs such as stunted cognitive development. These costs may manifest themselves in the future if children who are currently malnourished are likely to be less productive members of society. For example, researchers have established that obese adults earn lower wages (e.g. Averett and Korenman 1996; Cawley, 2004). The importance of highlighting these private costs is that the primary decision makers for children vis-à-vis food consumption and exercise are parents. Thus, the results from our study may well influence the thinking of parents as they become aware of the possibility that, in addition to healthcare issues, their children's future earning potential may be threatened by their current nutritional status. Further, as these important private costs also translate into social costs such as lower labor market productivity, policy makers will also consider them in addition to the healthcare costs.

Our research on the effects of childhood malnutrition on cognitive ability improves on past research in several ways. First, many previous researchers did not use nationally representative samples, thus limiting the degree to which their results can be generalized. Second, previous researchers often focused on either underweight or overweight rather than both extremes of the distribution. Third, we also examine the depth of malnutrition—for example we find that overweight children are heavier than those children who were overweight 20 years ago. Fourth, we explicitly recognize the potential for at least two sources of endogeneity and we take steps to tackle them. To address reverse causality, we use the method of instrumental variables. To the extent that our instruments are valid, the IV models allow us to obtain causal estimates of the effect of childhood malnutrition on the outcomes we study. This is important because those children who have poor cognitive functioning may over or under eat to compensate for that, thus suggesting the possibility of reverse causality. We also address time invariant unobserved heterogeneity by estimating individual fixed effects models.

The remainder of this paper is organized as follows. We begin by describing our data in section 2. We then provide some background on measurement and trends in child malnutrition in the United States in section 3, before examining the relationship between child malnutrition and cognitive ability and behavior as described in our data and in the literature (section 4). In section 5, we outline the theoretical foundations of our estimation strategy, and use a brief review of the literature on overweight children to motivate our choice of explanatory variables and instruments. Following a discussion of the results in section 6, we close with some concluding remarks.

DATA

To investigate the cognitive effects of malnutrition in children, we use data from the NLSY79, a panel study of approximately 12,000 individuals who were first interviewed in 1979 when they were between the ages of 14 and 22. The female respondents were re-interviewed annually from 1979-92 and bi-ennially since 1994. The data are a nationally representative sample of individuals born between 1957 and 1964 with an oversampling of the black, Hispanic, and low income white populations. Because of this, sampling weights are used when estimating summary statistics. The data include information about economic and demographic behavior and outcomes for the respondents and their families.

Our analysis focuses on the children of the original NLSY79 female respondents and includes data through the 2002 survey year (the latest available to us). At this point, the mothers are between the ages of 37 and 45 and the children range in age from 3 to 15. It is worth noting that the sample of children in the NLSY79 is born disproportionately to younger mothers. This is potentially troubling because these women tend to have lower education and income levels. However, a great deal of information is available about the family circumstances of these children over time (e.g., prenatal care and birthweight, family income, household composition, family structure, and family background). As children who are older than 13 years of age are likely to have more control over their own food choices, we limit our sample to elementary school-age children (i.e. ages 6 to 13 years).

Anthropometric measures of height and weight were recorded biennially for each child beginning in 1986. For approximately 20 percent of the cases, these are reported by the child's mother, not measured. They are therefore likely to suffer from measurement error which may in turn bias our coefficient estimates. Following a thorough cleaning of the data,

we address this potential reporting bias by using Cawley and Burkhauser's (2006) proposed method of predicting heights and weights based on models estimated from the National Health and Nutrition Examination Survey (NHANES III).

The NLSY79 data are ideally suited for this analysis because various developmental measures are available for the children.[2] These child assessments have been administered bi-ennially since 1986 (Baker and Mott 1989; Chase-Lansdale et al. 1991). Thus we have bi-annual data on children from 1986 to 2002. Our measures of cognitive development are the math and reading recognition scores from the Peabody Individual Achievement Tests (PIAT) (Dunn and Barkwardt Jr. 1970), administered to children ages 5 and over. The PIAT is among the most widely used brief assessments of academic achievement and is an individually administered measure of academic achievement. The test can be used with students in kindergarten through the twelfth grade. The PIAT Mathematics assessment begins with early skills (recognizing numerals) and progresses to measuring more advanced concepts. The Reading Recognition assessment measures word recognition and pronunciation ability, which are considered essential components of reading achievement. For both of these tests, we use the standardized score which has a mean of 100 and a standard deviation of 15. Reliability of these tests is quite high. Test-retest reliability for the Reading Recognition test is 0.89 for children from kindergarten through twelfth grade (Baker et al. 1993). The one month test-retest reliability for the PIAT mathematics assessment is 0.74, with lower levels of reliability for children in the lower grades (Dunn and Barkwardt Jr. 1970).

[2] These data have been used extensively to examine the effect of maternal employment on cognitive ability (Ruhm 2004) and the effect of paternal child care on children's cognitive ability (Averett et al. 2005).

MEASUREMENT AND TRENDS OF CHILD MALNUTRITION

A standard for measuring child nutritional outcomes in developed countries is the Body Mass Index (BMI), which is defined as the ratio of weight in kilograms over height in meters squared.[3] A particular child's BMI can be compared to those on tables configured by the Centers for Disease Control (CDC), which established distributions for each sex by age because BMI levels for children in the healthy reference population differ by age and gender. A child is considered likely to be overweight if his/her BMI for age is over the 95th percentile of the healthy reference population,[4] while he/she is considered likely to be at risk for overweight with a BMI for age above the 85th percentile. Children classified as likely to be underweight are those with BMI for age measures less than the fifth percentile of the

[3] Other metrics include stature for age and weight for age for all children (see CDC 2000; and Martorell and Habicht 1986), and overall evaluation of child health by physicians for very young children (Wolfe and Sears 1997). While stature for age is a commonly used measure of chronic malnutrition in developing countries, we do not use it in our analysis as we consider children up to the age of 15. Martorell and Habicht (1986) find that less than 10 percent of the worldwide variance in height can be ascribed to genetic or racial differences among children under the age of five. Genetic factors play a much larger role at older ages and as such, stature for age is not an appropriate measure in our analysis given our sample of children (described in more detail below).

[4] The reference population is based on a sample of healthy children in 2000. See CDC (2000) for a complete discussion of the reference population.

reference population (CDC 2000). In the population, prevalence rates for overweight, at risk of overweight, and underweight are calculated using these criteria.

To compare BMI measurements across age and gender cohorts, normalized BMI z-scores (hereafter BMIZ) are calculated. The BMIZ for child *i* is defined as follows:

$$BMIZ_i = \frac{BMI_i - \mu_{BMI}^{ref}}{\sigma_{BMI}^{ref}}$$

where BMI_i is the child's BMI measurement, μ_{BMI}^{ref} is the mean BMI measurement for the healthy reference population of the same age and gender, and σ_{BMI}^{ref} is the standard deviation of BMI measurements for the healthy reference population of the same age and gender. Since the healthy reference population is distributed normally, the BMIZ for the reference population has a standard normal distribution (CDC 2000). Thus, there is a probability distribution on the expected value of a BMIZ for any given child – a standard normal distribution to be precise. This means that there is a five percent probability that a child from the healthy reference population will have a BMIZ greater than 1.645. In other words, 1.645 is the BMIZ cutoff for the 95th percentile (overweight) in the distribution of BMI for age in the reference population. Similarly, the cutoffs for the fifth (underweight) and the 85th (at risk of overweight) percentiles are -1.645 and 1.0365, respectively.

To illustrate, in Figure 1 we plot the 1986, 1996 and 2002 distributions of the normalized BMI for age measures for children in the NLSY79 dataset, along with a standard normal distribution that represents the reference population. The apparent deviations of the sample distributions from the reference distribution indicate malnutrition (or "misnourishment") among the children in the survey.

To make this point clearer, this figure illustrates that the prevalence of overweight children in this sample rose from 6.7 percent to 18.2 percent between 1986 and 2002 (as seen by the intersections of the distributions and the overweight cutoff).[5] Further it shows that the prevalence of underweight children remained relatively constant from 1986 to 1996 percent but fell from 7.2 percent to 4.5 percent from 1996 to 2002.[6] With regard to overweight children, the figure also highlights a weakness of prevalence measures. Not only has the share of children who are overweight increased, but the degree to which these children are overweight has increased substantively (as seen by the 2002 distribution being considerably lower than the 1986 distribution and the reference distribution in the region to the right of the overweight cutoff).

Discrete measures of over- or undernutrition such as prevalence rates are important for information-dissemination purposes as they are something that the general public can easily comprehend. However, focusing only on specific cutoffs such as being above the 95th percentile or below the fifth percentile can be misleading for two reasons. First, it puts undue

[5] More accurately, the prevalence rates should be recorded as 2.8 in 1986, and 17.7 in 2002, as this represents the difference between the reference distribution and the sample distributions (i.e. 1.7 = 6.7 – 5.0, and 12.8 = 18.2 – 5.0, respectively). Nonetheless, we report prevalence rates for all those beyond the threshold as this is the standard practice.

[6] These estimates are potentially biased because the age distribution in the 2002 sample is weighted toward older children relative to the 1986 sample. The implication of this is that the BMI z-scores of the children at the upper tail of the 1996 distribution are likely to be biased downward, as are the prevalence rates for 2002 compared to 1986. These potential biases reinforce our concerns about child malnutrition trends.

emphasis on the admittedly arbitrary cutoff points. Marginal changes in the cutoff point can lead to categorical changes in the recorded health status of a child whose BMI for age measure is near the cutoff. Second, it ignores the distribution of BMI around the cutoff points. Thus, in our research, we borrow from the poverty literature by estimating not only prevalence rates, but also measures of the depth and severity of malnutrition (for overweight, see Jolliffe 2004; and for underweight, see Sahn and Stifel 2002).

The measures of the prevalence, depth and severity of malnutrition belong to a class of malnutrition measures that we refer to as M_α. These are defined as follows for underweight:

$$M_\alpha = \frac{1}{N}\sum_{i=1}^{N}(cut - BMIZ_i)^\alpha 1(BMIZ_i < cut),$$

and as

$$M_\alpha = \frac{1}{N}\sum_{i=1}^{N}(BMIZ_i - cut)^\alpha 1(BMIZ_i > cut),$$

for overweight, where *cut* is the under- or overweight threshold, and 1(.) is an indicator function that takes on a value of one when its argument is true, and zero otherwise. The parameter, α, can be interpreted as a malnutrition aversion parameter, similar to the poverty aversion parameter in the Foster-Greer-Thorbecke class of poverty measures (Foster et al. 1984). When α is zero, M_0 is the prevalence of malnutrition. When α is one, M_1 is the average malnutrition gap, where a child's gap takes on a value of zero if he or she is not malnourished. We refer to this measure as the depth of malnutrition. M_2 can be interpreted as the severity of malnutrition as it is a weighted average of the malnutrition gaps where the weights are the gaps themselves. The prevalence of malnutrition (M_0) is related to the number of malnourished. The depth of malnutrition considers the distance that the malnourished children are from the threshold, but weights each child equally. The severity puts more

weight on those who are furthest away from the threshold. As α approaches infinity, the social welfare function associated with the malnutrition measure is Rawlsian. In this extreme case, when comparing two distributions, the distribution with the most malnourished child is considered to have more malnutrition. In this paper, we restrict our analysis to the prevalence (M_0) and the depth (M_1) of malnutrition.

In Table 1, we present these types of malnutrition metrics applied to the NLSY79 data. Although underweight is typically thought of as a phenomenon only afflicting developing countries, it clearly occurs in the United States too. Indeed, we estimate about 6.6 percent of children between the ages of six and 13 in our sample had BMI levels that fell below the fifth percentile cutoff in 1986, though this proportion decreased markedly by 2002.[7] The prevalence and depth of underweight children did not change substantially over the decade from 1986 to 1996. This can also be seen in the form of the stable lower tails of the BMI for age distributions that appear in Figure 1.

These estimates of undernutrition outcomes are paralleled in the literature on input measures such as "food insecurity" and hunger. For example, according to the United States Department of Agriculture (USDA 2004), in 1999, 14 million children lived in "food insecure households," which means that their families lacked access to enough food to meet their basic steady state needs (Center on Hunger and Poverty 1999). Another recent survey estimated that approximately 4 million American children experienced prolonged periods of food insufficiency and hunger each year. This is roughly 8 percent of all the children under the age of 12 living in the United States. The same study shows that an additional 10 million

[7] This is consistent with Grigsby's (2003) estimate of an incidence rate less than 10 percent, though her estimate is a measure of protein-energy malnutrition (PEM), not underweight.

children are at risk for hunger (Kleinman et al. 1998). Finally, in a state by state analysis of food insecurity in the U.S., Nord et al. (1999) estimate that 9.7 percent of all households were food insecure during the years 1996-1998.

Not surprisingly, food insecurity is most prevalent in poor families. The Center for Hunger and Poverty estimates that 35.4 percent of families below the poverty line are food insecure compared to only 10.2 percent of households nationwide. Paradoxically, however, children who live in poverty can also be overweight – perhaps because they lack access to healthy, nutritious low-fat foods (Center for Hunger and Poverty 1999) – which adds to the confusion over the causes of under- and overnutrition.

Part of this paradox apparently stems from changes in food technologies and prices. As fast foods become more easily available and as the prices of high-calorie "junk" foods fall more quickly than the prices of fresh fruits and vegetables, the poor may stretch their limited budgets by substituting out of the latter into the former (Bhattacharya et al. 2004; Kennedy and Goldberg 1995). Bhattacharya and Currie (2001) found that in their sample of food-insecure youths nearly 20 percent were overweight, with almost one-third consuming excess amounts of sweets. Even adolescents who are not "food insecure" are likely to be malnourished – a concept Bhattacharya and Currie (2001) refer to as "misnourishment." Indeed, the determinants of food insecurity and malnutrition outcomes (underweight and overweight) are quite different. It is because of this difference and the apparent poverty-obesity "paradox" that Bhattacharya et al. (2004) conclude that, controlling for poverty, food insecurity is simply not a good predictor of poorer nutrition outcomes.

As indicated in Table 1 and in Figure 1, the prevalence of underweight among children in the United States has remained stable until recently when it fell.[8] The same, however, cannot be said for the prevalence and degree of overweight children. Using the NLSY79 data, we find that the share of children who are overweight rose by nearly 6.1 percentage points between 1986 and 1996, and by an additional 5.4 percentage points between 1996 and 2002. Further, the depth of overweight rose from an average of 0.03 standard deviations above the cutoff in 1986 to 0.53 standard deviations in 2002. In other words, not only is there a larger share of children who are considered to be overweight, the degree to which they are heavier has grown substantially.

This rapid rise in overweight children has been particularly pronounced over the past 25 years. A Department of Health and Human Resources report (2002), estimates that for a similar age group (6 to 19), 15 percent (almost 9 million) were overweight in 1999-2000. This is triple the rate in 1980. Among a younger cohort of children between the ages of two and five, over 10 percent are overweight, representing a 7 percent increase from 1994 (Ogden et al. 2002).

[8] Note that although the percentage of children with low weight is no more than we would expect to see in the healthy reference population, the degree to which these weights are low is higher than expected (e.g. the average underweight gap in the sample is 3.3 standard deviations, compared to 2.1 standard deviations for the reference population).

CHILD MALNUTRITION AND COGNITIVE DEVELOPMENT

One often-cited concern about undernutrition in children is that it may have negative consequences for cognitive development presumably because a lack of food deprives the brain of essential nutrients. Although this is generally an issue in the developing world, there is also a fairly sizeable literature on this topic in the medical field for the United States. Corman and Chaikind (1998), for example, find that low-birthweight children score lower on tests of academic performance. Alaimo et al. (2001) report that children aged 6 to 11 in food-insecure households scored lower on arithmetic tests, were more likely to have repeated a grade and to have seen a psychologist, and had difficulty getting along with other children. Winicki and Jemison (2003) also find that food insecurity negatively impacts the academic performance of kindergartners. Recent research provides compelling evidence that undernutrition can have detrimental effects on the cognitive development of children and on their behavior and that this may even impact their later adult productivity (Center On Hunger 1998). Weinreib et al. (2002) report that severe child hunger is correlated with a greater incidence of behavior problems and is also correlated with a greater level of reported anxiety/depression. There is evidence that programs such as providing breakfast to school age children have been effective in mitigating these consequences (Murphy et al. 1998). This has become such a strongly held view that some schools purportedly manipulated the nutritional content of their lunches to improve their test scores (Figlio and Winicki 2002).

At the other end of the weight distribution, there are concerns that overweight children may also suffer from nutrient deficiency (Nead et al. 2004), as well as low self-esteem and that low self-esteem may lead to lower academic performance or a perceived inability to perform well in school (Davison and Burch 2001). For adults, it has been demonstrated that

obese women have lower self-esteem than their non-obese counterparts (Averett and Korenman 1999, 1996). This also appears to be the case for children (Eisenberg et al. 2003), with the effect increasing with age (Strauss 2000). Furthermore, overweight children are more likely to be more socially isolated compared to adolescents who are not overweight (Strauss and Pollack 2003). There is also evidence that overweight children have lower academic performance (Datar et al. 2004), and are more likely to have behavior problems (Datar and Sturm 2004), to act as bullies, and to be bullied (Janssen et al. 2004). The social functioning of overweight children is likely to be reduced so much that Schwimmer et al. (2003) compare their qualities of life to those of children with cancer.

Basic evidence from the NLSY79 data is generally consistent with the literature. As illustrated in Table 2, children who are categorized as obese according to their BMI tend to fare worse vis-à-vis test score outcomes.

As the figures in Table 2 are only for a select number of years, we also plot the evolution of the PIAT math and reading recognition scores by nutritional status for each of the survey years (Figures 2 and 3). The patterns that emerge in these three figures are striking. Cognitive ability improves over time for all nutrition groups. However, there remain distinct differences in these scores at any point in time with healthy kids generally having higher cognitive scores than malnourished children. It is interesting to see that in the past two years, PIAT math scores have been greater on average for those children considered at risk of obesity. This may reflect the fact that there are so many more children in this category over time. The general story, however, is that although test scores have improved among malnourished children in the NLSY79 sample, they remain at a disadvantage relative to healthy children.

THEORY AND ESTIMATION STRATEGY

The theoretical foundations for modeling cognitive ability are based on integrating health and cognitive ability production functions into a common-preference model of household decision-making in the tradition of Becker (1981).

We start with the assumption that all household members have the same preferences. As such, the household can be treated as a single individual who maximizes a quasi-concave utility function that takes as its arguments the consumption of commodities and services, *q*, leisure, *l*, health status, *H* (of which, a child's anthropometric measurement, *n*, is one dimension), and cognitive ability, *C*, of each household member. Without considering the precise household decision-making process, though recognizing that parents make consumption decisions for young children, the household solves the following problem,

$$\begin{aligned} &\max_{q,l,H,C} u(q,l,H,C;X) \\ &s.t. \quad pq + w(l + T_H + T_C) \le wT + y \end{aligned} \tag{1}$$

where X represents individual, household and community characteristics, some of which are not observed. Allocation choices are made conditional on the full-income budget constraint, where p is a vector of prices, w is a vector of household members' wages, T is a vector of the household members' maximum number of work hours, y is sum of all household members' non-wage income, and T_H and T_C are time inputs into the production of health and cognitive ability.

The nutritional status of children, *n*, is determined by a biological health production technology:

$$n_i = n(I_n; X_n, \mu_i), \tag{2}$$

where I_n is a vector of health inputs, and μ_i represents the unobservable individual, family, and community characteristics that affect the child's nutritional outcomes. Specific household and community characteristics (e.g., demographics, educational levels, etc.), X_n, can have an impact on health by affecting household allocation decisions.

Similarly, the cognitive abilities of children, *c*, are determined by cognitive production technologies:

$$c_i = c(I_C, n_i; X_c, v_i) \tag{3}$$

where I_c is a vector of cognitive inputs, and v_i represents the unobservable individual, family, and community characteristics that affect the child's cognitive ability. These production technologies differ, however, in that the child's nutritional status, *n*, is also an input into the production of cognitive ability.

Ideally, we would estimate these production functions. However, the input vectors, *I*, include consumption goods, *q,* which contribute positively to household welfare both directly through *q*, and indirectly through *H* and *C*. They also include time inputs, T_H and T_C, which are choice variables that affect labor earnings and consumption of leisure. As such, the choice of consumption goods and health/cognitive inputs is simultaneous and makes consistent estimation of the production functions impossible in the absence of valid instruments for all of the inputs. Instead, by solving the household's optimization problem, we obtain reduced-form demand functions. For child nutritional status, this can be represented as follows:

$$n_i = n(I(X_n, p_n, w, y), X_n, \varepsilon_i) = \tilde{n}(X_n, p_n, w, y, \varepsilon_i), \tag{4}$$

where ε_i is the child-specific random disturbance term, which is assumed to be uncorrelated with the other elements of the demand function.

For cognitive ability, if we note that inputs and nutritional status are functions of exogenous information,

$$c_i = c(I(X_c, p_c, w, y), \tilde{n}(X_n, p_n, w, y, \varepsilon_i), X_c, \xi_i),$$

then a quasi-reduced form demand function can be estimated,

$$c_i = \tilde{c}(\tilde{n}(X_n, p_n, w, y, \varepsilon_i), X_c, p_c, w, y, \xi_i), \quad (5)$$

where ξ_i is the child-specific random disturbance term, which is also assumed to be uncorrelated with the other elements of the demand function. This is a quasi-reduced form demand function because it is a function of nutritional status, which is represented here by the reduced form function of exogenous information. Note that identification of the effect of contemporaneous nutritional status, *n*, on cognitive ability, *c*, requires differences in functional forms, or that the exogenous characteristics and prices that determine nutritional status, X_n and p_n, differ from those exogenous characteristics and prices that determine cognitive ability, X_c and p_c, respectively.

The basis of our estimation strategy can thus be summarized by the following equation:

$$c_{it} = \alpha + \boldsymbol{X}_{it}'\boldsymbol{\beta} + \boldsymbol{n}_{it}'\boldsymbol{\gamma} + \boldsymbol{w}_t'\boldsymbol{\delta} + \boldsymbol{p}_t'\boldsymbol{\eta} + \varepsilon_{it} \quad (6)$$

where c_{it} is a measure of cognitive ability for child *i* at time *t*,, $\boldsymbol{X}_{it}$ is a vector of individual-level, family-level and community-level observables, and $\boldsymbol{n}_{it}$ is a vector of measures of nutritional status (allowing for malnutrition) for child *i* at time *t*. The vector of parameters of interest is $\boldsymbol{\gamma}$. To allow for differing types of malnutrition (overweight and underweight) to affect cognitive development differently, we estimate three general forms of model (6) in which nutritional status enters as (a) a set of dummy variables indicating underweight or overweight, (b) a BMIZ quadratic, and (c) BMIZ along with the malnutrition gap. We also

present models in which BMIZ is entered linearly for comparison purposes.

Ordinary least squares (OLS) estimates of model (6) provide unbiased estimates of $\boldsymbol{\gamma}$ only if the child's nutritional status is exogenous, that is it is uncorrelated with the error term (i.e. $E(\varepsilon|\boldsymbol{n}) = 0$), and the direction of causality goes from nutritional status to cognitive development. If these conditions do not hold, then the OLS estimator will be biased. There are two general reasons why we might expect such a bias.

First, there may be unobserved characteristics that simultaneously determine cognitive ability and nutritional status. In such a situation, changes in these unobservable characteristics lead to coincidental changes in nutrition and cognitive development. The OLS estimator will be biased here because it attributes this change in cognitive development to the change in nutritional status. An example of one such unobservable is parental behavior. Datar et al. (2004) found that overweight kindergartners were more likely to come from poor families in which the parents did not read to their children or encourage good academic performance. This makes it difficult to determine if being overweight is truly the cause of the poor academic performance, or if poor parenting or some other factor is the cause of both the overweight and the poor academic performance. Davison et al. (2005) report that some family environments are obesigenic. In these families mothers and fathers have high dietary intake and low physical activity and the children in these families are at increased risk of obesity from ages 5 to 7 years. Unobserved school characteristics may also lead to biased estimates as they may be an important determinant of both academic achievement and nutritional status (Crosnoe and Mueller, 2004).

Second, the direction of causality may go both ways independently of unobservables. For example, while being overweight may cause low self-esteem, depression or other adverse

health outcomes and consequently low cognitive development, depression (which may stem from low cognitive ability) may be a cause of obesity (Goodman and Whitaker 2002). In our view, most of the previous research on children's weight and academic performance has not adequately addressed the issue of causality versus correlation. Although clearly not feasible, the ideal experimental design would be to randomly "assign" children to be overweight, underweight, or well nourished. If the assignments were truly random and children who were either over or underweight performed lower on tests of cognitive ability, we could be confident that it was their nutritional status that caused the relatively poor performance.[9]

Given that such an experiment is not feasible, we adopt three empirical methods for dealing with what we perceive to be the two separate and important sources of endogeneity — unobserved heterogeneity and reverse causality. We begin by estimating OLS models of equation (1) as a base of reference using as wide an array of control variables as possible to address potential heterogeneity and to avoid omitted variable bias. Our first method to address the endogeneity of nutritional status is to employ instrumental variables. This two-stage least squares (2SLS) method involves estimating a (set of) first stage equation(s),

$$n_{it} = \theta + \boldsymbol{X}_{it}'\boldsymbol{\varphi} + \boldsymbol{Z}_{it}'\boldsymbol{\lambda} + v_{it} \qquad (7)$$

where Z is a set of instrumental variables that are excluded from model (6). The criteria for suitable instruments are that they are highly correlated with nutritional status but uncorrelated with the error term in model (6). In other words, the only effect that a suitable instrument may have on cognitive ability is indirect, through its effect on nutritional status. Values for

[9] Interestingly, some experimental studies similar in design to this have been carried out and have found that students who fasted before school scored lower on tests of cognitive ability (Pollitt et al. 1998).

nutritional status predicted using the parameter estimates from the first stage estimation (7), $\hat{n}_{it}$, are then used as an explanatory variable in model (6) instead of observed nutritional status, n_{it},

$$C_{it} = \alpha + \boldsymbol{X}_{it}'\boldsymbol{\beta} + \hat{n}_{it}'\boldsymbol{\gamma}_{\text{iv}} + \boldsymbol{w}_t'\boldsymbol{\delta} + \boldsymbol{p}_t'\boldsymbol{\eta} + \varepsilon_{it} \qquad (8)$$

Given appropriate instruments, the IV estimator ($\boldsymbol{\gamma}_{\text{iv}}$) is an unbiased estimate of $\boldsymbol{\gamma}$, the causal effect of nutritional outcomes on cognitive ability. This approach not only addresses the concern of reverse causality, but also, because the instruments are uncorrelated with the error term, it removes biases in the estimator due to unobserved heterogeneity. The difficulty, of course, is finding suitable instruments that explain nutritional outcomes, but not academic achievement.

The strong genetic component of child weight (Cawley 2004; Grilo and Pogue-Geile 1991; Strunkard et al. 1986; Volger et al. 1995) indicates that a potential instrument is the mother's BMI. However, mother's current BMI is likely correlated with other unobservable family-level environmental characteristics that affect the child's cognitive ability.[10] To

[10] Several other potential instruments were explored, but proved fruitless. For example, as policymakers have called for schools to require that students spend more time in physical education (National Association of State Boards of Education 2000; American Academy of Pediatrics 2003), we merged our data with the School Health Policies and Programs Study (SHPPS) data from 1994 and 2000 on state-level physical fitness requirements. Our findings among elementary school children, however, were similar to Cawley et al. (2005) who found no link between state physical education requirements and the probability that a given high school student is overweight. Furthermore, state-level school policies on soft-

minimize the possibility that mother's BMI is correlated with the error term in the second stage, we use an historical measure of mother's BMI from 1981.[11] For nearly all of the cases in our sample, this BMI measurement was taken before the birth of the mother's first child, and as such is more likely to measure the genetic component of child weight than does the contemporaneous measure of mother's BMI. Indeed, a simple regression of mother's contemporaneous BMI on her 1981 BMI reveals that only 42 percent of the variation in current BMI is explained by historical levels. Nonetheless, we also include as control variables proxies for unobserved household environment and mother's unobserved abilities and attitudes that may be more/less favorable to cultivating higher academic achievement. These proxies include a dummy variable indicating if the child was breastfed, average household income since the child was born, and the mother's AFQT score and education level. We note that since these variables are employed as proxies for mother's attitudes and

drink vending policies available in the SHPPS data lacked the variation necessary to serve as a valid instrument for child nutritional status. The NLSY79 also has information on the average time that a child spends watching television. Research by medical doctors generally shows a negative effect of TV on different measures of academic achievement (Borzekowski and Robinson 2005; Chernin and Linebarger 2005) but recent research by economists reports evidence that television has a negligible effect on cognitive ability. (Gentzkow and Shapiro 2006). Thus, the available evidence suggests that television watching is not a suitable instrument for us.

[11] Genetic variation in weight as measured by parental weight status is also used by an instrument for child's BMI by Sabia (2007) in his examination of the effect of adolescent obesity on GPA.

household environment, the parameter estimates for these variables should not be interpreted as indicating a causal relationship. The parameter estimate for the breastfeeding dummy, for example, is not expected to represent the true effect of breastfeeding on cognitive ability. We remind the reader that the object of interest in this analysis is the $\boldsymbol{\gamma}$ parameters, not the potentially biased $\boldsymbol{\beta}$ parameters.

Our second approach to addressing unobserved heterogeneity (but not reverse causality) is to take advantage of the panel nature of the NLSY data and to estimate individual fixed effects (FE) models. Thus model (6) becomes

$$C_{it} = \alpha + \mu_i + \boldsymbol{X}_{it}'\boldsymbol{\beta} + \boldsymbol{n}_{it}'\boldsymbol{\gamma} + \boldsymbol{w}_t'\boldsymbol{\delta} + \boldsymbol{p}_t'\boldsymbol{\eta} + \varepsilon_{it} \qquad (9)$$

where μ_i is a child-specific dummy variable[12], and $\boldsymbol{X}_{it}$ now includes only those explanatory variables that are not fixed over time. The effectiveness of the fixed effects estimator in reducing the bias in $\boldsymbol{\gamma}$ depends on the unobservable characteristics that affect both nutritional outcomes and cognitive development being fixed over time and consequently differenced out.[13] Thus, this model improves on OLS but may not be ideal if factors influencing cognitive

[12] This is also referred to as the "within" estimator as it is equivalent to estimating a model of differences in within-individual means.

[13] We also estimated sibling fixed effects models, the results of which were qualitatively similar to the individual fixed effects models presented here. The motivation for this approach is that differences between siblings remove variance in weight attributable to a shared family environment. However, Cawley (2004) argues that this is not an appropriate way to remove unobserved heterogeneity citing evidence that shared family environments explain a negligible proportion of the variance in weight across siblings. However, others

ability and BMIZ vary over time. Further, as noted earlier, FE models do not eliminate the potential for reverse causality.

In our third approach, we employ an IV method that differs from the standard IV estimator due to the means through which identification is obtained. In this method proposed by Lewbel (2004),[14] the identification of $\boldsymbol{\gamma}$ comes from exploiting the heteroskedasticity of the first-stage equation (BMIZ). To illustrate, begin by defining the first stage equation as

$$n_{it} = \theta + \boldsymbol{X}_{it}'\boldsymbol{\varsigma} + v_{it} \qquad (10)$$

where $\boldsymbol{X}$ can include all or a subset of the explanatory variables in the main (second stage) equation (6). If $Cov(X,v^2)$ is nonzero (i.e. the data are heteroskedastic), then $\boldsymbol{\gamma}$ and the other parameters in the main equation can be estimated consistently without external instruments by an ordinary linear two stage least squares regression in which all of the exogenous right hand side variables and $\left(X - \overline{X}\right)\hat{v}^2$ are used as instruments for the child's BMIZ. We estimate model (6) using this method without external instruments. Breusch and Pagan (1979) tests for heteroskedasticity are applied to the first stage equations to test the identification requirement that $Cov(X,v^2) \neq 0$.

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have noted that an obesigenic family environment is an important predictor of children's changes in BMI (Davison et al. 2005). We do not report these models.

[14] See also Rigobon (2003)

RESULTS

Tables 3 through 7 present the results of our estimated models. Table 3 presents the weighted sample means. Because an important control variable in our model is the mother's wage, we predicted mother's wages for all women in our sample.[15] The sample consists of 20,856 child years. Just under half of the sample is female and the sample of child years is distributed evenly by age except for the oldest age groups (12 and 13) where there are slightly fewer child years. Tables 4 through 6 presents the results from OLS, FE and IV estimates of the determinants of cognitive development as measured by PIAT math and reading recognition scores. The IV models include the standard 2SLS models (hereafter referred to as IV) and the models that use heteroskedasticity to identify the parameters of interest (hereafter referred to as Hetero). The specifications in Table 4 include BMIZ entered linearly and a dummy variable specification for under and over weight. In table 5, BMIZ is entered as a quadratic,[16] while in table 6 we use BMIZ and the overweight gap as our measure of malnutrition. These tables only include the parameters of interest – the effects of nutritional status – from the OLS, IV, FE, and Hetero models. Table 7 presents the first stage estimates for the IV models,

[15] Predictor variables for this regression were age and education (both measured in years), their squares and an interaction between them and mother's AFQT score. Details of this regression are available upon request from the authors.

[16] Because we enter BMIZ nonlinearly (as a quadratic) and since the distribution of z-scores for a healthy population has a standard normal distribution, we first shift the BMIZ distribution by 10 points. While this does not change any of the information in the distribution of BMIZ, it does avoid confusion over how to interpret the squared value of a negative z-score.

while Table 8 presents the parameter estimates for the other control variables in the main models.

Focusing first on the results for PIAT math scores in Table 4 (top panel), in the OLS dummy variable specification we find that underweight children score a statistically significant 1.27 points lower than children whose weight is in the recommended range all else equal. Given a standard deviation of 15 for this test, the effect is equivalent to scores that are one eighth of a standard deviation lower. Overweight children do not have significantly different math scores in the OLS model. Although the pattern of significance is similar, the coefficients in the FE models are smaller, indicating that there is some unobserved heterogeneity.

Recall that the OLS and FE models do not account for possible reverse causality. Hence we turn to the IV and Hetero models. To determine the validity and relevance of our instruments in the IV model, we report the p-values for the *F*-test of joint significance of the excluded instruments (mother's BMI and BMI squared), and two tests of weak instruments – the Cragg-Donald statistic[17] and the p-value for the Anderson-Rubin test[18]. Despite the fact

[17] This is the multiple equation analog to the *F*-statistic used in the Stock-Yogo (2005) test, and is available as an option in Stata's *ivreg2* command. This statistic is used to test if the instruments are weak. The critical value is the 5 percent value compiled by Stock and Yogo (2005) and reported by *ivreg2*. The interpretation is that a Cragg-Donald statistic above this critical value rejects the null hypothesis that the instruments are weak a the 5 percent level of significance. See also Murray (2005).

[18] The Anderson-Rubin *F*-statistic is used to test the null hypothesis that all of the endogenous variables are jointly insignificant.

that the parameter estimates for the IV models are much larger than for the OLS and FE models, these test statistics indicate that we can reject the null hypothesis that the instruments are weak, and that our IV estimates are not much biased. Because our models are just identified we cannot rely on standard tests of overidentification to determine if the instruments can be legitimately excluded from model (8). Hence, we focus on the intuition that there is a strong genetic component to weight as referenced above, and that by using an historical measure of the mother's BMI, the instrument should be less correlated with the child's current home environment.[19] Experiments were conducted using alternative instruments such as sibling nutritional status and district-level fast-food prices. The results of these estimates are similar to those presented here and are available upon request from the authors.

The IV models clearly indicate that children at both ends of the nutrition spectrum — underweight and overweight — have lower test scores on average. Underweight children have PIAT math scores that are nearly one and a quarter standard deviations lower than those of children with BMIZ scores in the recommended range (18.76/15). Overweight kids have PIAT math scores that are about six-tenths of a standard deviation lower than their well-nourished peers, all else equal (9.21/15). In the Hetero models, only the effect of overweight

[19] As an informal test of the intuition of the instruments, we estimated reduced form regressions with the instrumental variables as the explanatory variable and test scores as the dependent variables. The instrumental variables have coefficients that are significantly different from zero and have signs that support the genetic-component identification story – positive for mother's BMI and negative for mother's squared BMI. (See Murray 2006)

is statistically significant, with a magnitude that is similar to the IV model. We reject the null hypothesis of no heteroskedasticity using a standard Breush-Pagen test.

The bottom panel of table 4, the PIAT reading recognition scores, tells a slightly different story. In the dummy variable specification using OLS, we see that PIAT reading scores are significantly lower for overweight children but not for underweight children (the opposite of the case for the PIAT math scores). This pattern is the same for the FE models though the coefficient is slightly smaller. The IV and Hetero results are similar across math and reading scores in that they are much larger than the OLS and FE coefficients, though the effect of malnutrition on PIAT reading recognition scores in the Hetero models is considerably smaller than the IV models (0.2 standard deviations lower for the former compared to 1.5 standard deviations lower for the latter).

Turning to the models in table 5 where BMIZ is entered as a quadratic, a clear pattern emerges for the PIAT math scores (top panel). The positive and significant coefficients for BMIZ and negative and significant coefficients for squared BMIZ indicate that as BMIZ scores rise, PIAT math scores first rise and then fall. This pattern is consistent in sign and significance across the estimation procedures used. The only difference is that the magnitude of the coefficient estimates in the OLS and FE models is considerably smaller than for the IV and Hetero models.

To facilitate interpretation of the non-linear effect of the BMIZ quadratic in table 5, we calculate marginal effects at points of interest in the BMIZ distribution (i.e. at the underweight, at-risk of overweight and overweight thresholds) and test if these effects are significantly different from zero. These marginal effects reveal a consistent statistically significant effect for children at the underweight threshold. An improvement in nutritional

status beyond this threshold improves test scores. For example, a 1 standard deviation increase in BMI for age at the underweight threshold leads to PIAT math scores increasing from 0.036 standard deviations in the FE model (0.55/15) to 0.82 standard deviations in the IV model.[20] Although the marginal effects calculated at the at-risk of overweight and overweight thresholds are not statistically significant for the OLS and FE models (though the coefficient estimates are), they are for the IV model and for overweight in the Hetero model. For the IV model, a 1 standard deviation increase in BMIZ leads to between a 0.43 (for at risk of overweight) and 0.71 (for overweight) standard deviation decline in math scores.

A different pattern exists for the coefficients on BMIZ and BMIZ-squared for PIAT-reading recognition scores (Table 5, bottom panel). The parameter estimates are statistically significant with the expected signs for the OLS, IV, and Hetero models, but are not statistically different from zero for the FE model. As before, the IV coefficients are considerably larger. The marginal effects, however, are statistically significant in the IV models only for at-risk of overweight and overweight kids but not for underweight kids. Because the Breusch-Pagan χ^2 test statistic is small, indicating that we cannot reject the null hypothesis of homoskedasticity, the parameter estimates for the Hetero reading recognition model are suspect.

As noted in section 3, children who are classified as overweight are heavier than they were even a decade ago. To understand how the depth of overweight affects test scores, the

[20] The marginal effects in Table 5 are interpreted as the average change in test scores for a 1 standard deviation increase in BMIZ. To interpret these effects in terms of test score standard deviations, one needs to divide the marginal effect by the reference population standard deviation of the test scores (i.e. by 15).

results of estimates in which BMIZ and the overweight gap are used as our regressors are reported in Table 6.[21] In this model, the BMIZ in linear form controls for the nutritional status of the entire population while inclusion of the overweight gap controls for the depth of overweight. The BMIZ and the overweight gap parameter estimates in the OLS, IV, and Hetero models all suggest that overweight children have lower math and reading scores. The coefficient estimates in the FE models were not statistically significant, and as with the previous estimates, the null hypothesis of homoskedasticity for the Hetero reading recognition model is not rejected. In Table 6, we assist the interpretation of parameter estimates on BMIZ and the overweight gap by simulating the effect of a 0.1 standard deviation increase in BMIZ on the dependent variables. This is done by applying the parameter estimates to the adjusted sample distribution of BMIZ, keeping all other factors constant. Note that for children with BMIZ scores less than 0.1 standard deviations below the 95 percentile threshold initially, their overweight gaps take on positive values in the simulation. The averages of the resulting predicted changes in PIAT scores are then reported for all kids and for overweight kids. Because these are simulations, we do not have tests of significance.[22] Our estimates and simulations reveal a negative effect of being overweight on cognitive ability. The magnitude of these effects ranges from test scores that are 0.12 to 3.03 standard deviations lower due to a 0.1 standard deviation increase in BMIZ.

The parameter estimates for the other explanatory variables are generally as we might expect (Table 7). In particular, birth weight exerts a positive and statistically significant

[21] We also experimented with using the underweight gap but this was the best fitting model.

[22] Test statistics can, however, be formed by bootstrapping these simulations to create standard errors.

effect on cognitive ability. The low birth weight literature generally establishes a strong negative correlation between birth weight and cognitive ability (Hack et al. 1991; Corman and Chaikind, 1998; Boardman et al., 2005 and Almond et al., 2002) consistent with our results. Hispanic children score lower on tests of math than white children, but higher on reading recognition when compared to white children. Black children score lower on both math and reading tests when compared to white children. Birth order is an important predictor of cognitive ability with first born children scoring better on the cognitive tests. Household size is also an important predictor of cognitive ability with children from larger household having lower scores on the math and reading recognition tests. Mother's age is not an important predictor of a child's cognitive ability but children with more educated mothers have higher cognitive ability. Average household income is also an important predictor in the expected directions. Children in families with more economic resources have higher scores on math and reading recognition. Again, we caution that strict interpretation of these estimates can be misleading as they were also included in the models as proxies for unobserved mother's behavior and household environment. The urban dummy is not an important predictor of cognitive ability. Finally, as we saw in the raw data, scores on tests of cognitive ability rise over time.

Naturally the validity of our IV results depends on how well our instruments perform. We present the first stage results of our IV estimation in table 7. The mother's BMI and BMI squared are significant predictors of a child's BMIZ score both individually and jointly. The p-values on the F-statistics testing their joint significance are all less than 1 percent. Further, the coefficient estimates in the first stage for the IV model are of the expected signs and magnitudes. Nonetheless, the R-squares in the first stage regressions are low ranging from

0.07 to 0.11. Since the magnitude of the bias of the IV estimator is inversely related to the r-squared from the first stage, the IV estimates in Tables 4, 5 and 6 could be substantively biased. Indeed it can be shown that when the R-squared from the first stage estimation is low, even a small correlation between the error and the instrument can lead to a large bias (Murray, 2005). Thus, we cannot rule out that a lack of explanatory power may be causing the IV estimates to become large. Nonetheless, the large Cragg-Donald statistics and Anderson-Rubin *F*-statistics suggest that IV estimation is predictable enough to provide relatively unbiased parameter estimates (Murray 2006; Stock and Yogo 2005).

To summarize, we find evidence that child malnutrition as measured by BMIZ exerts a negative effect on cognitive abilities as measured by the PIAT math and reading recognition scores. In FE specifications using dummy variables to control for malnutrition, we find evidence that overweight children have lower reading recognition test scores and that underweight children have lower math scores. The IV models using mother's historical BMI as an instrument, and the heteroskedasticity-identification models, suggest a negative effect of being overweight on both math and reading test scores, and often find a negative effect of being underweight. While none of these methodologies provides a "silver bullet", they do yield qualitatively similar results that collectively provide evidence that deviating from "normal" weight lowers academic ability as measured by these test scores.

CONCLUSION

The prevalence of overweight among children has reached near epidemic proportions in the United States over the past twenty five years. Our research documents that the prevalence and depth of childhood overweight has increased over time, and although the incidence of underweight children has declined, the depth of underweight remains higher than would be expected in a healthy reference population. Our primary research question is whether childhood malnutrition, as measured by BMIZ scores, is an important causal predictor of cognitive ability in elementary school aged children. This is an important question for parents, school administrators and policymakers. If overweight and/or underweight children perform poorly on tests of cognitive ability, and if this persists into adulthood, they are likely to be less productive as adults. This lower productivity has both private and public costs that arise in addition to the medical costs associated with malnourishment.

We use data from children born to the women in the NLSY79 to address whether under- or overweight children have lower cognitive ability. We are particularly interested in establishing if such malnutrition is a cause of low cognitive ability. Endogeneity, however, is an important concern with regard to any statistical estimate of this relationship. In particular, we are concerned about two sources of endogeneity – reverse causality and unobserved heterogeneity. Therefore, in addition to using standard OLS estimation procedures, we estimate individual fixed effects models as well as two different types of IV models. Although standard tests confirm the validity and relevance of our instruments, the explanatory power of our first stage models is modest.

Although there are issues related to each of the estimation methods, the collective weight of the evidence provided in this analysis suggests that childhood malnutrition (either underweight or overweight) has a negative effect on cognitive abilities as measured by the PIAT math and reading recognition scores. Further, we find evidence that the degree to which a child is overweight matters. While these effects are generally statistically significant, the range of the magnitude of our coefficient estimates makes it difficult to pin down the precise impact. Nonetheless, the consistent results of these three estimation procedures indicates a robustness of our findings. Thus, our research suggests that parents should be particularly vigilant in monitoring a child's nutritional status whatever the proximate cause of under or overweight might be.

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Table 1: Malnutrition Among American Children between Ages 6-13

Year	Underweight	At Risk of Obesity	Obese
Incidence			
1986	6.6	20.1	6.7
1996	7.2	26.3	12.8
2002	4.5	35.6	18.2
Reference	*5.0*	*15.0*	*5.0*
*Average Gap (standard deviations)**			
1986	4.1	10.0	2.4
1996	5.3	16.9	5.1
2002	3.3	23.6	7.4
Reference	*2.1*	*7.8*	*2.1*

Source: Authors' calculations from NLSY

* Multiplied by 100

Table 2: Behavioral/Cognitive Outcomes by Probable Nutritional Status
Mean standardized scores for children of age 6-13

		PIAT	
	Behavioral Problems Index	Math	Reading Recognition
All Years			
Underweight	104.8	100.9	104.2
Healthy	105.2	103.1	105.5
At Risk of Obesity	106.4	103.1	105.2
Obese	106.9	101.5	103.6
1986			
Underweight	106.4	99.6	102.6
Healthy	109.6	99.7	103.2
At Risk of Obesity	109.1	100.1	104.6
Obese	111.5	100.9	103.2
1996			
Underweight	104.5	101.7	105.0
Healthy	105.2	104.0	105.6
At Risk of Obesity	107.8	103.8	105.4
Obese	107.3	101.2	103.6
2002			
Underweight	100.0	107.6	111.1
Healthy	101.3	107.4	108.9
At Risk of Obesity	102.1	108.8	109.2
Obese	103.6	104.4	106.2

Source: Authors' calculations from NLSY

Table 3: Models of Determinants of Cognitive Development
- *Sample Means*

NLSY79 Children of Age 6-13

	Mean	Std. Dev.
PIAT Math	102.80	13.32
PIAT Reading Recognition	105.14	14.17
BMI for age (z-score)	0.27	1.18
Mother's BMI in 1981	22.09	3.60
Dummy: Child is female	0.48	0.50
Dummy: Child is 7 years old	0.13	0.33
Dummy: Child is 8 years old	0.13	0.34
Dummy: Child is 9 years old	0.13	0.33
Dummy: Child is 10 years old	0.13	0.33
Dummy: Child is 11 years old	0.13	0.33
Dummy: Child is 12 years old	0.11	0.32
Dummy: Child is 13 years old	0.11	0.31
Birthweight (ounces)	119.80	18.30
Birthweight missing	0.04	0.20
Child born early (no. of weeks)	0.70	1.67
Dummy: Child was breastfed	0.51	0.50
Dummy: Hispanic	0.07	0.26
Dummy: African American	0.16	0.37
Dummy: Child is 2nd Born	0.34	0.47
Dummy: Child is 3rd Born	0.13	0.34
Dummy: Child is 4th Born	0.04	0.20
Dummy: Child is 5th Born	0.01	0.11
Dummy: Child is 6th or higher	0.01	0.07
No. of HH members age 0-2	0.18	0.43
No. of HH members age 3-5	0.30	0.52
No. of HH members age 6-11	1.42	0.84
No. of HH members age 12-17	0.66	0.84
Mother's age (years)	34.31	4.62
Mother's AFQT score in 1989	43.78	27.44
Mother's highest grade - some HS	0.17	0.38
Mother's highest grade - completed HS	0.40	0.49
Mother's highest grade - college+	0.39	0.49
Mother's wage (predicted)	7.50	1.38
Mother's wage squared	58.11	22.59
Avg real annual HH income (1000) since birth	39.90	48.77
Share of poor households in county	0.22	0.16
Dummy: Urban area	0.58	0.49
Dummy: 1988	0.09	0.28
Dummy: 1990	0.10	0.30
Dummy: 1992	0.11	0.31
Dummy: 1996	0.14	0.35
Dummy: 1998	0.15	0.35
Dummy: 1998	0.14	0.35
Dummy: 2000	0.12	0.33
Dummy: 2002	0.11	0.31
Number of observations	20,856	

Table 4: Models of Determinants of Cognitive Development
- Effects of Nutritional Status (BMIZ & Dummies)

NLSY79 Children of Age 6-13

	OLS				Individual Fixed Effects				IV				Heteroskedasticity Identification			
	Linear		Dummies		Linear		Dummies		Linear		Dummies		Linear		Dummies	
	Coeff	t-stat	Coeff	t-stat	Coeff	t-stat	Coeff	t-stat	Coeff	t-stat	Coeff	t-stat	Coeff	t-stat	Coeff	t-stat
PIAT Math																
BMIZ	**0.27**	2.68 ***			**0.19**	1.82 *			**-1.92**	-5.92 ***			**-1.61**	-3.16 ***		
Dummy variables																
Underweight (bmiz < -1.6449)			**-1.27**	-3.21 ***			**-0.94**	-2.81 ***			**-18.76**	-2.13 **			**10.19**	1.40
Overweight (bmiz > 1.6449)			**-0.19**	-0.46			**0.05**	0.14			**-9.21**	-4.44 ***			**-8.92**	-2.72 ***
R-squared	0.22		0.22		0.08		0.08		0.14		0.13		0.11		0.10	
F-test of excluded instruments (p-value)									0.000		0.000					
Weak identification statistics																
Cragg-Donald statistic									446.8		13.22					
- Stock & Yogo critical value									*19.33*		*7.03*					
Anderson-Rubin F-test (p-value)									*0.00*		*0.01*					
Breusch-Pagan test of heteroskedasitcy; $X^2(1)$													225.0		223.2	
PIAT Reading Recognition																
BMIZ	**0.05**	0.39			**0.05**	0.54			**-2.76**	-7.77 ***			**-1.35**	-4.30 ***		
Dummy variables																
Underweight (bmiz < -1.6449)			**-0.58**	-1.36			**-0.08**	-0.25			**-14.24**	-2.72 ***			**2.59**	1.30
Overweight (bmiz > 1.6449)			**-0.67**	-1.68 *			**-0.51**	-1.72 *			**-22.38**	-5.71 ***			**-2.84**	-3.31 ***
R-squared	0.19		0.19		0.06		0.06		0.14		0.15		0.12		0.11	
F-test of excluded instruments (p-value)									0.000		0.000					
Weak identification statistics																
Cragg-Donald statistic									441.6		11.72					
- Stock & Yogo critical value									*19.33*		*7.03*					
Anderson-Rubin F-test (p-value)									0.000		0.006					
Breusch-Pagan test of heteroskedasitcy; X2(1)													44.9		41.8	
Number of observations	20,856		20,856		20,856		20,856		20,856		20,856		20,856		20,856	

Note: Mother's BMI and BMI-squared in 1981 used as instruments

Table 5: Models of Determinants of Cognitive Development
- Effects of Nutritional Status (BMIZ quadratic)

NLSY79 Children of Age 6-13

	OLS		Individual Fixed Effects		IV		Heteroskedasticity Identification	
	Coeff	t-stat	Coeff	t-stat	Coeff	t-stat	Coeff	t-stat
PIAT Math								
Coefficients								
BMIZ	**3.04**	2.67 ***	**2.77**	2.67 ***	**70.70**	3.43 ***	**57.42**	2.49 **
BMIZ squared	**-0.14**	-2.42 **	**-0.13**	-2.50 **	**-3.50**	-3.52 ***	**-2.43**	-2.09 **
Marginal Effects								
underweight threshold (-1.6449)	**0.74**	3.56 ***	**0.55**	3.09 ***	**12.28**	3.03 ***	**16.81**	3.97 ***
a-risk threshold (1.0364)	**0.00**	0.02	**-0.16**	0.92	**-6.46**	4.82 ***	**-0.82**	-0.17
overweight threshold (1.6449)	**-0.17**	0.76	**-0.32**	1.41	**-10.72**	4.24 ***	**-3.78**	-1.74 *
R-squared	0.20		0.06		0.17		0.22	
F-test of excluded instruments (p-value)					0.000			
Weak identification statistics								
Cragg-Donald statistic					22.59			
- Stock & Yogo critical value					*(7.03)*			
Anderson-Rubin F-test (p-value)					*0.00*			
Breusch-Pagan test of heteroskedasitcy; $X^2(1)$							238.3	
PIAT Reading Recognition								
Coefficients								
BMIZ	**2.15**	1.72 *	**0.67**	0.70	**25.15**	1.75 *	**49.80**	2.35 **
BMIZ squared	**-0.10**	-1.66 *	**-0.03**	-0.65	**-1.28**	-1.82 *	**-2.52**	-2.38 **
Marginal Effects								
underweight threshold (-1.6449)	**0.40**	1.80 *	**0.14**	0.84	**3.75**	1.42	**7.76**	2.07 **
a-risk threshold (1.0364)	**-0.16**	0.85	**-0.03**	0.20	**-3.11**	2.53 **	**-5.73**	-2.11 **
overweight threshold (1.6449)	**-0.29**	1.14	**-0.07**	0.33	**-4.67**	2.27 **	**-8.79**	-2.29 **
R-squared	0.20		0.06		0.17		0.20	
F-test of excluded instruments (p-value)					0.000			
Weak identification statistics								
Cragg-Donald statistic					21.47			
- Stock & Yogo critical value					*(7.03)*			
Anderson-Rubin F-test (p-value)					0.006		2.1	
Breusch-Pagan test of heteroskedasitcy; X2(1)								
Number of observations	20,856		20,856		20,856		20,856	

Note: Mother's BMI and BMI-squared in 1981 used as instruments

Table 6: Models of Determinants of Cognitive Development
- Effects of Nutritional Status (Overweight Gap)

NLSY79 Children of Age 6-13

	OLS		Individual Fixed Effects		IV		Heteroskedasticity Identification	
	Coeff	t-stat	Coeff	t-stat	Coeff	t-stat	Coeff	t-stat
PIAT Math								
Coefficients								
BMIZ	**0.38**	4.39 ***	**0.13**	1.14	**1.83**	1.71 *	**10.68**	4.22 ***
Overweight gap (BMIZ - 1.648)	**-1.68**	-2.87 ***	**0.06**	0.09	**-31.98**	-3.70 ***	**-42.35**	-3.01 ***
Simulation - BMIZ increase by 0.1 standard deviations								
Effect on all scores	**0.02**				**-0.23**		**0.51**	
Effect on scores of overweight kids	**-0.12**				**-2.92**		**-3.03**	
R-squared	0.22		0.08		0.10		0.23	
F-test of excluded instruments (p-value)					0.000			
Weak identification statistics								
Cragg-Donald statistic					44.45			
- Stock & Yogo critical value					*(7.03)*			
Anderson-Rubin F-test (p-value)					0.000			
Breusch-Pagan test of heteroskedasitic; $X^2(1)$							237.5	
PIAT Reading Recognition								
Coefficients								
BMIZ	**0.13**	1.42	**-0.05**	-0.45	**1.30**	0.00	**2.85**	1.15
Overweight gap (BMIZ - 1.648)	**-1.29**	-2.03 **	**0.04**	0.06	**-55.33**	-5.25 ***	**-106.2**	-6.12 ***
Simulation - BMIZ increase by 0.1 standard deviations								
Effect on all scores	**-0.03**				**-0.34**		**-1.09**	
Effect on scores of overweight kids	**-0.11**				**-4.92**		**-9.84**	
R-squared	0.20		0.06		0.15		0.20	
F-test of excluded instruments (p-value)					0.000			
Weak identification statistics								
Cragg-Donald statistic					42.24			
- Stock & Yogo critical value					*(7.03)*			
Anderson-Rubin F-test (p-value)					0.000			
Breusch-Pagan test of heteroskedasitic; X2(1)							1.7	
Number of observations	20,856		20,856		20,856		20,856	

Note: Mother's BMI and BMI-squared in 1981 used as instruments

Table 7: First-Stage Models

NLSY79 Children of Age 6-13	BMIZ		BMIZ-squared		Overweight Gap		Underweight Dummy		Overweight Dummy	
	Coeff	t-stat	Coeff	t-stat	Coeff	t-stat	Coeff	t-stat	Coeff	t-stat
Instruments:										
Mother's BMI in 1981	0.179	11.36 ***	3.549	11.16 ***	0.002	0.73	-0.020	-5.95 ***	0.016	3.44 ***
Mother's Squared BMI in 1981	-0.0021	-6.82 ***	-0.0400	-6.45 ***	0.0001	3.15 ***	0.0003	4.62 ***	0.0000	0.44
Dummy: Child is female	0.05	3.10 ***	1.01	3.03 ***	0.00	-0.29	-0.01	-1.89 *	0.00	0.11
Dummy: Child is 7 years old	0.04	1.34	0.77	1.20	-0.01	-1.33	-0.01	-1.20	0.00	-0.11
Dummy: Child is 8 years old	0.12	3.73 ***	2.20	3.47 ***	-0.01	-1.34	-0.02	-2.66 ***	0.01	0.63
Dummy: Child is 9 years old	0.12	3.63 ***	2.20	3.37 ***	-0.01	-1.44	-0.02	-3.48 ***	0.01	0.59
Dummy: Child is 10 years old	0.17	5.34 ***	3.25	4.92 ***	-0.01	-2.82 ***	-0.04	-5.02 ***	0.00	0.15
Dummy: Child is 11 years old	0.19	5.54 ***	3.39	5.01 ***	-0.01	-2.80 ***	-0.04	-5.90 ***	0.00	-0.42
Dummy: Child is 12 years old	0.17	4.80 ***	2.97	4.16 ***	-0.03	-5.52 ***	-0.04	-5.69 ***	-0.03	-2.43 **
Dummy: Child is 13 years old	0.16	4.23 ***	2.61	3.52 ***	-0.03	-4.91 ***	-0.05	-6.27 ***	-0.03	-2.86 ***
Birthweight (ounces)	0.01	13.20 ***	0.13	13.02 ***	0.00	5.38 ***	0.00	-8.11 ***	0.00	6.07 ***
Birthweight missing	-0.12	-2.86 ***	-2.42	-2.78 ***	-0.01	-1.32	0.02	2.44 **	-0.02	-1.55
Child born early (no. of weeks)	0.01	1.36	0.17	1.58	0.00	3.65 ***	0.00	0.53	0.01	3.56 ***
Dummy: Child was breastfed	-0.04	-2.16 **	-0.83	-2.23 **	-0.01	-2.02 **	0.01	1.55	-0.01	-2.06 **
Dummy: Hispanic	0.06	2.55 **	1.36	2.79 ***	0.01	2.66 ***	0.01	1.23	0.03	3.82 ***
Dummy: African American	0.16	6.80 ***	3.31	7.04 ***	0.03	8.17 ***	-0.01	-1.63	0.05	6.89 ***
Dummy: Child is 2nd Born	-0.05	-2.63 ***	-1.12	-2.80 ***	0.00	-1.44	0.00	0.40	0.00	-0.83
Dummy: Child is 3rd Born	-0.07	-2.72 ***	-1.56	-2.83 ***	0.00	-0.93	0.00	-0.27	-0.01	-1.69 *
Dummy: Child is 4th Born	-0.11	-2.62 ***	-2.38	-2.74 ***	-0.02	-2.93 ***	0.01	0.72	-0.03	-2.05 **
Dummy: Child is 5th Born	-0.23	-3.31 ***	-5.09	-3.62 ***	-0.04	-3.86 ***	0.00	-0.14	-0.06	-2.84 ***
Dummy: Child is 6th or higher	-0.28	-2.77 ***	-5.83	-2.82 ***	-0.04	-2.29 **	0.03	1.55	-0.06	-1.95 *
No. of HH members age 0-2	-0.04	-2.06 **	-0.67	-1.87 *	0.00	0.51	0.01	2.35 **	0.00	-0.07
No. of HH members age 3-5	-0.08	-5.34 ***	-1.75	-5.57 ***	-0.01	-4.25 ***	0.00	1.05	-0.02	-4.01 ***
No. of HH members age 6-11	-0.09	-8.51 ***	-1.85	-8.80 ***	-0.01	-8.57 ***	0.01	2.49 **	-0.03	-8.97 ***
Mother's age (years)	0.00	-0.39	-0.04	-0.51	0.00	-0.67	0.00	-1.36	0.00	-1.12
Mother's AFQT score in 1989	0.00	-1.62	-0.02	-1.92 *	0.00	-3.21 ***	0.00	-0.70	0.00	-2.73 ***
Mother's AFQT score missing	0.02	0.31	0.47	0.43	0.01	1.34	0.00	0.06	0.02	1.35
Mother's highest grade - some HS	-0.23	-5.64 ***	-4.72	-5.68 ***	-0.03	-4.68 ***	0.02	2.53 **	-0.05	-4.01 ***
Mother's highest grade - completed HS	-0.19	-4.49 ***	-3.76	-4.51 ***	-0.03	-4.60 ***	0.02	2.77 ***	-0.04	-3.41 ***
Mother's highest grade - college+	-0.16	-3.66 ***	-3.39	-3.74 ***	-0.03	-4.48 ***	0.01	1.54	-0.05	-3.52 ***
Mother's wage (predicted)	0.00	-2.59 ***	-0.01	-2.56 **	0.00	-1.94 *	0.00	1.72 *	0.00	-2.62 ***
Share of poor households in county	-0.23	-3.76 ***	-4.56	-3.70 ***	-0.01	-0.69	0.02	1.66 *	-0.05	-2.60 ***
Dummy: Urban area	-0.01	-0.35	-0.13	-0.36	0.00	0.07	0.00	0.52	0.00	0.32
Dummy: 1988	0.14	3.51 ***	3.06	3.71 ***	0.02	2.65 ***	0.00	0.10	0.04	3.54 ***
Dummy: 1990	0.15	3.44 ***	3.37	3.86 ***	0.03	4.04 ***	0.02	2.47 **	0.06	4.83 ***
Dummy: 1992	0.31	6.73 ***	6.60	7.19 ***	0.04	6.17 ***	0.01	0.59	0.09	7.01 ***
Dummy: 1996	0.10	2.16 **	2.80	2.91 ***	0.04	6.09 ***	0.05	5.05 ***	0.08	6.10 ***
Dummy: 1998	0.24	4.56 ***	5.51	5.20 ***	0.06	7.26 ***	0.03	2.37 **	0.11	7.40 ***
Dummy: 1998	0.29	5.00 ***	6.61	5.58 ***	0.06	7.29 ***	0.02	1.77 *	0.12	6.82 ***
Dummy: 2000	0.51	7.86 ***	11.20	8.50 ***	0.09	9.12 ***	0.01	0.75	0.17	8.77 ***
Dummy: 2002	0.58	8.11 ***	12.55	8.74 ***	0.10	9.22 ***	0.01	0.50	0.20	9.66 ***
Constant	6.7	28.3 ***	35.5	7.5 ***	-0.1	-2.0 **	0.5	9.3 ***	-0.3	-4.1 ***
R-squared	0.10		0.11		0.07		0.09		0.07	
Number of observations	20,856		20,856		20,856		20,856		20,856	

Table 8: Models of Determinants of Cognitive Development
- *Other Determinants*

NLSY79 Children of Age 6-13	PIAT Math		PIAT Reading Recognition	
	Coeff	t-stat	Coeff	t-stat
Dummy: Child is female	-0.16	-0.64	2.93	10.08 ***
Dummy: Child is 7 years old	-0.01	-0.04	0.41	1.34
Dummy: Child is 8 years old	0.08	0.29	1.26	4.45 ***
Dummy: Child is 9 years old	0.28	0.84	0.14	0.39
Dummy: Child is 10 years old	-0.26	-0.79	-0.48	-1.38
Dummy: Child is 11 years old	-0.45	-1.21	-1.88	-4.86 ***
Dummy: Child is 12 years old	-1.64	-4.12 ***	-2.40	-5.27 ***
Dummy: Child is 13 years old	-2.43	-5.35 ***	-2.75	-5.35 ***
Birthweight (ounces)	0.02	2.04 **	0.02	2.04 **
Birthweight missing	-0.89	-1.48	-1.12	-1.64
Child born early (no. of weeks)	-0.11	-1.39	-0.18	-1.94 *
Dummy: Child was breastfed	0.84	2.58 ***	0.58	1.53
Dummy: Hispanic	-1.93	-4.27 ***	0.51	0.96
Dummy: African American	-3.21	-7.45 ***	-1.04	-2.03 **
Dummy: Child is 2nd Born	-1.16	-4.30 ***	-2.48	-8.08 ***
Dummy: Child is 3rd Born	-1.89	-4.67 ***	-3.75	-7.90 ***
Dummy: Child is 4th Born	-2.57	-3.66 ***	-5.16	-6.57 ***
Dummy: Child is 5th Born	-2.99	-2.76 ***	-5.20	-3.85 ***
Dummy: Child is 6th or higher	-7.63	-4.53 ***	-8.77	-4.41 ***
No. of HH members age 0-2	-0.40	-1.65 *	-0.55	-2.14 **
No. of HH members age 3-5	-0.61	-3.10 ***	-0.60	-2.93 ***
No. of HH members age 6-11	-0.51	-3.14 ***	-0.68	-3.85 ***
Mother's age (years)	0.00	0.03	0.12	1.39
Mother's AFQT score in 1989	0.14	16.21 ***	0.14	14.34 ***
Mother's AFQT score - missing	-2.07	-2.38 **	-1.60	-1.53
Mother's highest grade - some HS	1.54	2.09 **	1.22	1.37
Mother's highest grade - completed HS	3.15	4.30 ***	3.27	3.67 ***
Mother's highest grade - college+	4.03	5.00 ***	4.05	4.15 ***
Mother's wage (predicted)	0.01	2.30 **	0.01	3.20 ***
Share of poor households in county	-1.08	-1.27	-0.38	-0.39
Dummy: Urban area	-0.05	-0.17	-0.23	-0.70
Dummy: 1988	-0.97	-2.73 ***	-1.55	-4.24 ***
Dummy: 1990	-0.84	-1.80 *	-1.95	-3.79 ***
Dummy: 1992	-0.52	-0.94	-1.55	-2.44 **
Dummy: 1996	-0.50	-0.76	-2.20	-2.91 ***
Dummy: 1998	0.80	1.04	-1.47	-1.66 *
Dummy: 1998	0.33	0.37	-1.47	-1.42
Dummy: 2000	1.40	1.36	-0.83	-0.69
Dummy: 2002	2.36	2.09 **	0.33	0.25
Constant	77.0	12.6 ***	81.3	12.2 ***
R-squared	0.22		0.20	
Number of observations	20,856		20,856	

Figure 1: Distribution of BMI-for-Age
for Children of Age 6-13 in the NLSY79 Sample

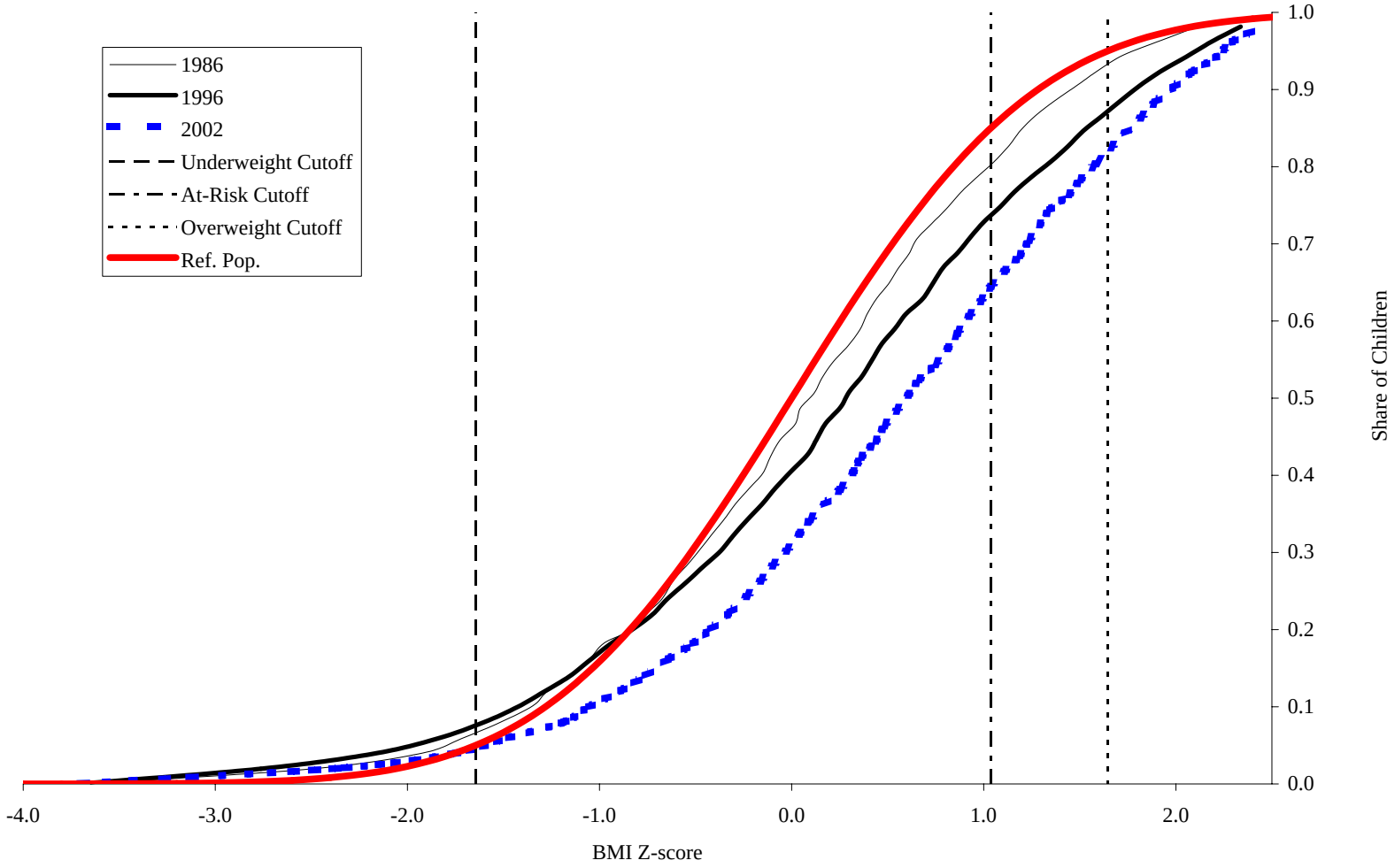

Figure 2: Evolution of PIAT Math Scores
by Nutritional Status among NLSY79 Children

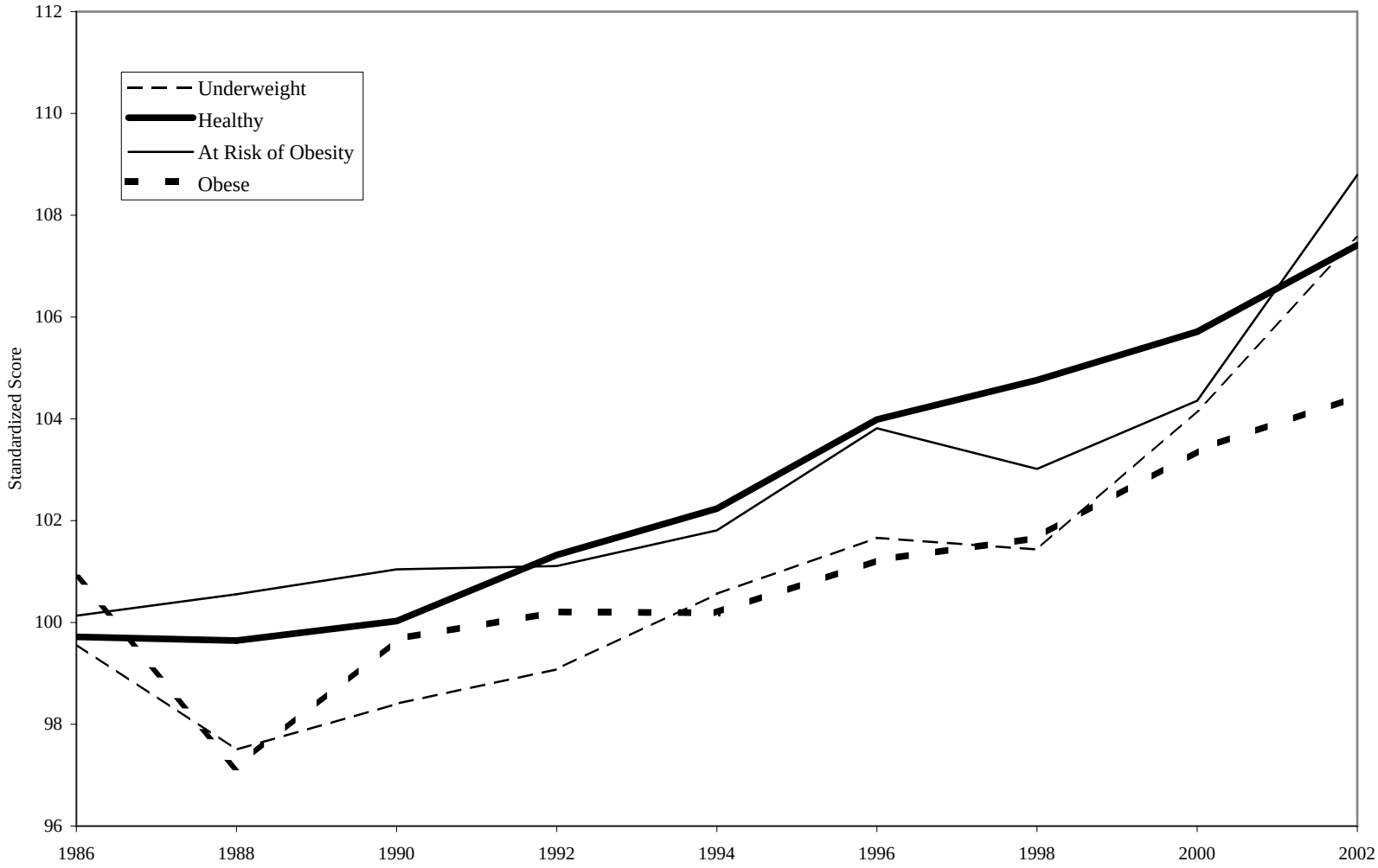

Figure 3: Evolution of PIAT Reading Recognition Score by Nutritional Status among NLSY79 Children