Measures of the Intergenerational Transmission of Body Mass Index Between Mothers and Their Children in the United States, 1981 – 2004

Abstract: This research provides estimates of the intergenerational persistence of Body Mass Index (BMI) between women and their children when both are at similar stages of the lifecycle. Using data from the National Longitudinal Survey of Youth 1979 (NLSY79) and the Young Adults of the NLSY79, associations between the weight status of women and their children are measured when both generations are between the ages of 16 and 24. In the entire sample, the measured intergenerational correlation of BMI is significantly different from zero and equal to 0.35. This result differs by gender with a BMI correlation between female children and their mothers of 0.38, compared to a significantly lower BMI correlation of 0.32 between mothers and their sons. Measures of this relationship across the distribution of BMI using quantile regression and quadrant dependence techniques indicate that the intergenerational persistence of BMI is strongest at higher levels of BMI. Strong dependence across generations is found when categorical outcomes of obesity and overweight are implemented. These results provide evidence of the strong persistence of weight problems across generations which may affect economic mobility within families.

JEL Codes: I10, J62

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Both the genetic traits and household environments shared by parents and children influence the intergenerational transmission of health capital, which may influence economic success for both generations. The goal of this paper is to provide estimates of the intergenerational persistence of one form of health capital, weight status. The influence of obesity on income, wealth and other measures of socioeconomic status has been documented in recent research (e.g., Cawley (2004) and Zagorsky (2005)). Thus, the transmission of weight problems between generations may provide a mechanism to explain a portion of the relatively high degree of persistence in economic status found in Solon (1992) and Zimmerman (1992).[1]

Various dimensions of health status have been identified as potentially important mechanisms in the intergenerational transmission of economic status between generations.[2] The measure of health capital used in this paper, Body Mass Index (BMI),[3] has substantial variation across the population and combines the anthropometric measures of height and weight, whose intergenerational correlations have been previously studied.[4] Of greatest interest for the current research is the persistence across generations of elevated BMI levels associated with obesity that are most likely to influence economic success.

Rapid expansion in rates of obesity in the United States and other developed countries has intensified the pursuit for explanations based on changes in food production technology and other aspects of the household environment such as video games and the internet. The

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[1] Bowles & Gintis (2002) review the broad topic of intergenerational transmission of economic status and Grawe and Mulligan (2002) provide a theory of economic interpretations of such correlations.
[2] See Currie (2009) for a review of the pathways through which health may affect intergenerational mobility.
[3] BMI is calculated as (weight in kilograms)/(height in meters^2). Adults with a BMI of 30 and above are typically classified as obese, while those with BMI levels greater than or equal to 25 are classified as overweight.
[4] See Galton's 1889 *Natural Inheritance* for the genesis of this literature.

intergenerational relationships estimated in this paper reflect social, cultural, genetic[5] and environmental components, but attempts to identify the strength of BMI persistence across the distribution of BMI will receive the greatest attention.[6] When parents make food consumption choices (or provide the feasible food consumption set) for children for meals eaten at home, this common environment generates a correlation between generations when both eat unhealthy (or healthy) food together. Since there exist substantial disparities in rates of obesity across various gradients of socioeconomic status and between ethnic groups,[7] intergenerational BMI correlations are estimated separately for subgroups in certain specifications.

There exist numerous economic theories and empirical studies attempting to explain the enormous growth in rates of obesity in the United States, including changes in the relative price of unhealthy foods (caloric intake) and implicit costs of exercise (energy expenditure).[8] While the goal of the present research is not to explain growth in rates of obesity, the data used in this paper span a period of rapid growth in obesity rates. Instead, emphasis is placed on understanding the degree of dependence in BMI and obesity between generations within families during this period of rapid weight growth. The women in the National Longitudinal Survey of Youth 1979 (NLSY79) were entering adulthood during a period of substantial growth in rates of adult obesity while their offspring in this sample were in late adolescence toward the end of the period of analysis in previous studies of obesity growth.[9] The shift in the distribution of BMI

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[5] One pathway may be via rates of time preference that affect obesity (as in Komlos, Smith and Bogin (2007)) when such preferences are transmitted across generations.

[6] For a review of genetic studies of obesity, see Maes et. al. (1997) and Bouchard et. al. (2003).

[7] Odgen (2008) demonstrates significant differences in rates of adolescent obesity by ethnic groups. Baum and Ruhm (2008) provide evidence of disparities in levels and growth rates of obesity across SES levels.

[8] See, for example, Cutler et. al. (2003); Chou et. al. (2004); Lakdawalla and Philipson (2009) and Komlos et. al. (2009).

[9] Burkhauser, Cawley and Schmeiser (2009) provide evidence that growth in rates of obesity are sensitive to the measure of obesity used. Measures of skinfold thickness indicate that substantial growth in rates of obesity in United States occurred 10 to 20 years earlier than indicated by more commonly used BMI measures.

between the NLSY79 mothers and their offspring detailed in Section 3 (Figure 1) reflects this substantial growth in rates of obesity.

The longitudinal data provided in the NLSY79 allow for multiple observations of height and weight for both generations during late adolescence and early adulthood so that a portion of the potential bias resulting from transitory fluctuations in BMI is mitigated. The data also permit intergenerational associations to be measured when both generations are at similar points in the lifecycle so that the estimated relationships are not confounded by influences that affect both generations contemporaneously. The intergenerational persistence in BMI is first measured using traditional estimates of correlation coefficients that allow for comparison to previous studies of this topic (which typically use single observations of BMI or obesity status measured in the same year for both generations). Then, non-central measures of the association of BMI and obesity between generations are estimated that allow for the dependence of the relationship to vary across the distribution of weight outcomes.

Estimating the relationships of interest separately by the gender of the offspring, I find that the intergenerational correlation of BMI between mothers and daughters is significantly higher than between mothers and sons, with a correlation of approximately 0.38 (relative to 0.32 for sons to mothers). Using measures of this relationship estimated via quantile regression and quadrant dependence techniques, which allow for the magnitude of intergenerational BMI associations to vary across the distribution of the offspring's BMI, the strength of this correlation appears to be largest at higher levels of BMI. This indicates that the intergenerational persistence of BMI is strongest for children born to mothers with the highest levels of BMI. Since the negative economic consequences of weight status occur at relatively higher levels of BMI, a higher rate of transmission of such weight problems among obese mothers could limit the

economic mobility of their offspring. Estimates from probit models for obese and overweight outcomes add to this evidence of a strong dependence in potentially harmful (both economically and physiologically) weight outcomes across generations.

I. Previous Research on the Intergenerational Transmission of Obesity and its Economic Consequences

The influential role of obesity on measures of economic outcomes such as income, wealth and educational attainment has received increasing attention in recent studies. Research in this field has often found that the economic consequences of obesity vary by ethnicity and gender.[10] A common result among these studies is that females are much more likely than males to suffer negative economic consequences due to elevated weight.

The causal influence of obesity on income is estimated by Cawley (2004) who uses an instrumental variable exploiting within-generation correlations of BMI in the NLSY79. Controlling for the potential endogeneity of weight status in determining income using siblings' BMI as an instrument, he finds that white females in the NLSY79 who weigh two standard deviations above mean BMI earn 9 to 18% less than their average weight counterparts, while no significant differences were found for black or Hispanic females (or males of any ethnicity). Numerous other studies have found additional evidence of the negative consequence of obesity on wages for females, including Baum and Ford (2004), Conley and Glauber (2005) and Han, Norton and Stearns (2009). Brunello and D'Hombres (2007) find negative effects of obesity on wages for both males and females in Europe, especially in the southern part of Europe where obesity is relatively rare. Morris (2006) finds consistent evidence of a negative effect of obesity

[10] See Averett and Korenman (1999) for a comparison of the influence of obesity on economic outcomes for blacks and whites. Case and Menedez (2009) consider explanations of higher rates of obesity among females in low-income countries.

on occupational attainment (measured as the average wage rate associated with a given occupation) for women in England. Paraponaris et. al. (2005) find that obese individuals are less likely to be employed in France and experience longer unemployment spells. Other studies of the influence of obesity on wages find larger effects for women in several European countries (Atella et. al. (2008)), Denmark (Greve (2008)), and Finland (Johansson et. al. (2009)). A large and significant negative relationship between wealth and obesity is found for white females in Zagorsky (2005) who uses data from the NLSY79, and finds smaller effects for black females and white males. Crosnoe (2007) finds a significant reduction in the likelihood of college attendance for obese females with the strongest influence of weight problems on education attainment in schools with the lowest rates of obesity. Murasko (2009) identifies influential socioeconomic factors on the development of obesity during childhood while Cawley and Spiess (2008) find that obese children of both genders have lower verbal skill development at ages as young as 2 to 3 years old. Given the likely negative economic consequences of elevated BMI levels (and other measures of poor health), the persistence of such health problems within families may explain reduced prosperity in multiple generations. Obviously, the transmission of health outcomes between generations is a complicated process governed by a myriad of factors including genetics, culture, family values and consumption choices. One of the first discussions in the economics literature of such correlations in health status is provided by Ahlburg (1998). He reviews studies measuring intergenerational relationships in outcomes such as lifespan and certain diseases and reports estimates of intergenerational correlations for lifespan in the range of 0.15 to 0.3.

The transmission of obesity from parents to children is studied for families within a single health insurance organization in the state of Washington in Whitaker et. al. (1997). Using

observations of BMI during childhood and adolescence combined with data on parental BMI levels, they find that children who are obese early in life and have at least one obese parent are three times more likely to become obese adults than non-obese children in households where neither parent is obese. While this effect dissipates slightly as children grow older, they find a very strong correlation between obesity in adolescence and becoming an obese adult with parental obesity raising the likelihood of obesity of offspring in early adulthood.

Classen and Hokayem (2005) measure the influence of maternal obesity on the likelihood of child and adolescent obesity with controls for several influential socioeconomic and demographic characteristics included. Using data from the NLSY79, they find that children of extremely obese mothers (with BMI greater than 40) are 50% more likely to be obese than their counterparts with mothers having BMI levels in the recommended range of 18.5 to 25.

Martin (2008) measures the intergenerational relationship of BMI using data from the National Longitudinal Survey of Adolescent Health (Add Health) which includes measures of BMI for adolescent children and reports of parental obesity status. She finds that children whose parents are both reported to be obese have BMI levels one standard deviation above the sample mean. The primary limitation of this study is that parental weight status is limited to a simple indicator for obesity and likely measured with substantial reporting error since the question asks one parental respondent to report whether the child's mother and father are obese, but no measures of actual parental height and weight are collected. Furthermore, only the contemporaneous relationship of obesity status across generations can be measured, rather than correlations generated when both generations are at similar stages of life as in the current study.

Anderson, Butcher and Schanzenbach (2007) use data from repeated cross sections of the National Health and Nutrition Examination Survey (NHANES) to measure the contemporaneous

correlation of BMI between women and their children in a given survey year. They find that the intergenerational BMI elasticity between women and their children has increased over time (using observations from 1971 to 2004), but does not vary significantly between families of different income levels. They also find similar intergenerational BMI elasticities for both fathers and mothers to their children. Overall, they find an intergenerational BMI elasticity between women and their children in the most recent NHANES of roughly 0.2 which they attribute to the interaction of common environments and genes between parents and children, with the role of common environments becoming increasingly influential over time.

Relative to previous studies, the current paper has several advantages. The longitudinal structure of the NLSY data allows for averaging over multiple observations of BMI to avoid potential measurement error resulting from observing height and weight at only a single point in time. Furthermore, observations of BMI for women in the NLSY79 and their children are available when both generations are at similar points in the lifecycle (late adolescence and early adulthood) instead of contemporaneous observations of BMI for both generations in a single year as in previous studies. The implementation of quantile regression and quadrant dependence estimates provide measures that allow the dependence of BMI between generations to vary across the entire distribution of weight outcomes, rather than relying solely on measures of central tendency such as correlation coefficients. A limitation of the NLSY data relative to the NHANES data is that height and weight is self-reported for a majority of observations in the NLSY.[11] If the bias resulting from self-reported weight levels is constant across generations

[11] Cawley (2004) finds small effects on estimated coefficients of interest when correcting for self-reporting bias in the NLSY data, but Cawley and Burkhauser (2008) find significant underreporting of weight among those with elevated BMI levels in the NHANES data. Average BMI levels calculated from self-reported height and weight are 0.34 to 0.98 points lower than BMI values calculated from measured height and weight for demographic groups other than African American and Mexican American males where no significant differences are found.

within families, then this would not introduce substantial bias in the coefficients of interest in this paper, but could be of concern in the case of large changes in BMI across generations.[12]

II. Description of Data

To estimate the degree of correlation in BMI between generations, I use data from the National Longitudinal Survey of Youth 1979 (NLSY79) and the Children and Young Adults of the NLSY79 (YA NLSY79). The NLSY79 began in 1979 with interviews of a nationally-representative sample of people born between 1957 and 1964 (so that all respondents were between ages 14 and 22 at the beginning of the survey). This sample was interviewed annually until 1986 and biennially since 1988. Height and weight data for women in the NLSY79 were first collected in the 1981 survey. Beginning in 1986, data on the children of the women in the NLSY79 sample were collected. In 1994, children aged 14 or older of women in the NLSY79 were surveyed in the YA NLSY79.

The structure of these surveys provides the highly desirable feature of being able to measure BMI at similar stages of life for mothers and their children. Previous studies of intergenerational mobility in economics have identified two critical shortcomings of data used to measure such relationships: potential measurement error from using only a single observation of the outcome of interest (assuming there exists temporal variance in the measure of mobility studied) and observing the outcome at different stages of the lifecycle (such as in cross sectional observations of earnings for two generations at a single point in time).[13] For example, an observation of income for a father at age 50 might be paired with an observation on his son's

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[12] This would result in the measured correlation overstating the true correlation (since an obese son of a woman with a recommended BMI level would be more likely to understate his weight while the mother is less likely to have substantial measurement error in a self-report if she is of recommended weight).

[13] Problems arising from measurement of income at different points in the lifecycle for measuring intergenerational correlations are discussed in Haider and Solon (2006).

earnings in the same year at age 25. While measurements of the age of each generation are often incorporated to attempt to adjust for such lifecycle differences, it is evident that knowledge of the son's earnings when he is age 50 would provide much more accurate estimates of the intergenerational correlation of income. Using the NLSY79 and YA NLSY79 data, I calculate the weight status of mothers and their children when both generations are between the ages of 16 and 24. This generates an estimate of the intergenerational persistence of BMI across generations at a crucial stage for determining the likelihood of developing weight problems later on in life.

A second critique of studies of intergenerational mobility focuses on whether the observed data are representative of the true level of the variable of interest. The transitory nature of weight and height during late adolescence could result in significant deviations from a more permanent measurement when data are only collected for a given year. Obviously, weight is a variable that is likely to fluctuate over time and multiple observations are desirable for generating an accurate measure of weight status. The longitudinal structure of the NLSY data allows for multiple observations of BMI for both generations to generate an estimate of its intergenerational dependence.[14]

Since the NLSY79 cohort contains individuals who were first interviewed at age 14 to 22 in 1979 and height and weight data are only available beginning with the 1981 survey, the age range for averaging BMI observations extends up to age 24. Although the period from age 16 to 24 is likely to feature significant changes in BMI (potentially as a result of individuals making food choices without supervision from parents for the first time), averaging BMI values over this

[14] The average number of observations of BMI per young adult in the NLSY is 2.5. Approximately one-quarter of the YA sample includes only a single observation of BMI between ages 16 and 24, while more than 20 percent have four or more observations of BMI in this age range.

range should accurately reflect weight status during this phase of development and reduce potential bias from only a single observation of BMI.[15] For the determination of whether individuals are obese, overweight or underweight, the cutoffs for adults of BMI over 30 indicating obesity, BMI above 25 and below 30 as indicating overweight and BMI below 18.5 indicating underweight are used.[16]

Among the 12,686 people initially surveyed in the NLSY79, 6,283 were female. Of these women, approximately two-thirds have had children who have been included in at least one of the YA surveys. The selection criterion for inclusion in this analysis requires that the child have reached at least age 16 by the 2004 YA NLSY79 survey. This leaves 4,748 children born to 2,560 women in the NLSY79 sample for analysis. Given this criterion, the included sample contains an oversampling of younger mothers and the demographics displayed in Table 1 make apparent that the racial composition of the sample is not nationally representative. Thus, results for several of the specifications will be presented separately by racial and ethnic categories.

Due to the structuring of the data for this analysis, there is no evident method to incorporate the available sample weights from the NLSY79 and YA NLSY79 surveys since height and weight measurements are collected for both generations in multiple survey years. Sample weights are provided for each NLSY survey year to generate a representative cross-section for a given survey year. However, since this study combines observations on mothers interviewed in the beginning of the NLSY79 panel (who were assigned sample weights for the first survey in 1979) and averages BMI over multiple survey years (each of which has unique

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[15] BMI values for women who were pregnant at the time of the survey are set to missing, but the women may still be included in the sample if they were ever interviewed between ages 16 and 24 while not pregnant.

[16] The 2000 CDC Growth Charts for BMI have 85th percentile cutoffs of 24.2 and 24.7 and 95th percentile cutoffs of 27.6 and 28.9 for the youngest 16 year old males and females, respectively. These percentiles are generally used to classify adolescents as "at risk of overweight" and overweight, but often referred to as overweight (above 85th percentile) and obese (above 95th percentile). The 85th and 95th percentile cutoffs exceed 25 and 30 respectively for females by the end of their 17th year and for males early in their 19th year.

sampling weights) with observations on their children who are also assigned sample weights in each survey year, this structure of data from multiple NLSY surveys does not lend itself to using the cross-section NLSY sample weights. The results reported in this paper are unweighted, but an appendix includes results generated using custom longitudinal sampling weights available for the YA NLSY data that provide representative weights for multiple survey years of the survey. The limitation of these custom weights for this application is that height and weight observations for the offspring are not all taken from the same set of survey years. Thus, the weighted results in the appendix use the custom sample weights for the YA NLSY survey from 1986 to 2004. Results (Tables A1 and A2) are not sensitive to the oversampling of black and Hispanic individuals in the unweighted data, but most results are presented separately by ethnicity given this potential concern.

A limitation of the health data available in the NLSY79 is the lack of measures of actual body fatness (adiposity) which limits the analysis to using height and weight in the calculation of BMI which may be a poor proxy for adiposity. While some studies include measures of skinfold thickness and waist circumference, only self-reported height and weight are available in the NLSY data.[17]

III. Measures of Intergenerational BMI Correlations

This section provides estimates of intergenerational elasticities and correlations of BMI for the NLSY79 sample described in the previous section. In order to estimate the correlation of BMI between generations, this section specifies an empirical model of obesity transmission. While there are numerous studies of the genetic epidemiology of obesity and relationships between generations (e.g. Bouchard et. al. (2003) and Garn et. al. (1989)), there have been

[17] See Cawley and Burkhauser (2008) for a discussion of the limitations of BMI as a measure of actual body fatness.

relatively few attempts to measure this correlation in a large data set with broad geographic diversity when both generations are at similar stages of physical development.

A. Intergenerational Elasticities and Correlations of BMI

Following a method for measuring intergenerational correlations developed in Goldberger (1989), the general form for the regressions estimated in this section is:

$$BMI_{ijg+1} = \beta_o + \beta_1 BMI_{jg} + e_{ijg+1}$$

where BMI_{ijg+1} is the natural logarithm of average BMI between ages 16 and 24 of child *i* in family *j* of generation *g+1*. Similarly, BMI_{jg} is the natural logarithm of average BMI between ages 16 and 24 of the mother (belonging to generation *g*) in family *j*. The stochastic disturbance term e_{ijg+1} for the regression has family and individual-specific components. The standard errors reported in this paper are adjusted (using the cluster command in Stata) to account for the presence of multiple children from a single mother in the data. With this specification, β_1 represents the elasticity of a child's BMI with respect to his or her mother's BMI, with an elasticity of zero indicating no persistence in weight across generations. The intergenerational correlation of BMI is then given by

$$\rho_{BMI} = \beta_1 \frac{\sigma_g}{\sigma_{g+1}}$$

where σ_g and σ_{g+1} are the standard deviations of BMI_g and BMI_{g+1}, respectively. In order to mitigate possible measurement error from temporal variation in BMI, I average all available observations for mothers and their children when both are between ages 16 and 24.

A kernel density approximation of the distribution of average BMI for both generations between ages 16 and 24 indicates an evident shift toward higher BMI levels among children of the women in the NLSY79 (Figure 1).

INSERT FIGURE 1 ABOUT HERE

Summary statistics of relevant variables in the NLSY sample (Table 1) show that average BMI increased by more than 7% between generations, while rates of overweight and obesity have both increased by more than 50% across generations in the NLSY and YA NLSY samples.

INSERT TABLE 1 ABOUT HERE

B. Estimates of the Intergenerational Correlation of BMI

Estimates of the intergenerational elasticities and correlations of BMI between mothers and their children for the entire sample and separately by gender and ethnicity indicate a strong degree of persistence across generations (Table 2a). For the full sample, the estimated intergenerational elasticity of BMI is significantly different from zero and equal to 0.42 which implies a correlation of 0.35.[18] The elasticity of BMI between mothers and their daughters is nearly 0.5 which implies a correlation of BMI across generations of 0.38 for females. Males have a significantly lower correlation of BMI to their mothers of 0.32. The estimated correlations do not vary significantly between ethnic groups. Significant differences between genders in intergenerational BMI elasticity persist only for the black sub-sample. Results using the custom sample weights for the NLSY YA data indicate similar levels of correlations and significant differences by gender for the full sample (Table A1).

INSERT TABLE 2a ABOUT HERE

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[18] The correlation is lower than the elasticity in this case since the standard deviation of the maternal BMI is less than that for their children's BMI.

Estimates of the persistence of BMI across generations within families of different SES measures indicate higher levels of intergenerational correlations among higher SES families (Table 2b). Using income measured as the average family income per household member for the offspring during the period of life from age 2 to 18 (available in the NLSY79 interviews with the mothers), children are organized into quartiles based on their family's relative income within the NLSY79 sample.[19] Intergenerational BMI correlations range from 0.31 in the lowest income quartile to 0.36 in the highest, but there are not statistically significant differences in elasticities between income quartiles.

Another measure of SES disparities considers the duration of time during childhood that the offspring's family was in poverty. The poverty variable is calculated as the percentage of time the child's family was classified as being in poverty (reported for mothers in the NLSY surveys) when the child was age 2 to 18.[20] The correlation of BMI across generations is significantly higher for children who grew up in families who were never in poverty relative to children whose families were classified as having income below the Federal Poverty Line at least once (1 – 25% of the time in poverty). Comparing families that were ever classified as being in poverty during the offspring's upbringing relative to families that were never in poverty reveals a significantly lower intergenerational BMI correlation for families who were ever impoverished, but the magnitude of the correlation increases as the duration of poverty rises.

Results for differences in BMI correlations by maternal education indicate a higher persistence of BMI at higher education levels, with the correlation significantly larger in households in which the mother has completed college relative to households in which the

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[19] The oversampling of relatively younger mothers is evident with the overrepresentation of families in the lowest income quartile among the NLSY79 sample.

[20] The NLSY provides indicator variables for whether the household income for each family is below the Federal Poverty Income Guideline, controlling for household size, for each survey year.

mother did not complete high school. However, the largest disparity in magnitudes of intergenerational correlations is between the two highest levels of education (college graduates relative to those who attended some college). The relationship between intergenerational BMI correlations and these measures of SES are not monotonic in any of the three cases, but there exists some evidence of stronger intergenerational BMI persistence in higher SES families. This is in contrast to the results in Anderson, Butcher and Schanzenbach (2007) who do not find significant differences between SES groups in BMI correlations between women and their children in the NHANES data.

IV. Non-Central Measures of the Intergenerational Persistence of BMI

The previous section provides estimates of intergenerational elasticities (and associated correlations) of BMI using least-squares regressions. Such estimates allow for inference for changes in BMI levels between generations around the population mean of the maternal BMI distribution. However, since relatively high BMI levels substantially above the mean (indicative of obesity) are most likely to influence economic outcomes, results in this section measure the strength of intergenerational BMI transmission across the entire distribution of observed BMI levels. The consequences of weight status for economic outcomes are most relevant for BMI levels above 30 indicating obesity, and thus, understanding the intergenerational transmission of such undesirable health outcomes is of greatest interest.

A. Quantile Regression Estimates of BMI Correlations

Quantile regressions estimate parameters by minimizing sums of absolute deviations (rather than squared residuals as in classical linear regression) for a specified division of the

dependent variable into quantiles.[21] Such estimates provide measures of the influence of a covariate (maternal BMI) at multiple quantiles of the distribution of the dependent variable (in this case, the BMI of a woman's offspring).[22] The particular quantiles used in quantile regressions can be chosen based on areas in the distribution of greatest interest, number of available observations or a variety of other criterion. This flexibility allows for potentially non-linear relationships to be more readily recognized than in an ordinary least squares regression. For the application here, we are interested in whether the association to maternal BMI is constant across the distribution of the offspring's BMI.[23]

Parameters in a quantile regression model of intergenerational BMI transmission are estimated for the θ*th* quantile ($0 < \theta < 1$) by solving for the elasticity β_{1_θ} by defining $Diff_i = BMI_{ijg+1} - (\beta_o + \beta_1 BMI_{jg})$ for *n* observations and solving

$$\min_{\beta} \frac{1}{n} \sum_{i=1}^{n} \left\{ 1_{Diff_i \geq 0} \theta Diff_i + 1_{Diff_i < 0} (1-\theta) \left| Diff_i \right| \right\}$$

with the indicator function $1_{Diff>0}$ defined to equal one for individuals having positive values of $Diff_i$, and zero otherwise.

INSERT FIGURE 2 ABOUT HERE

INSERT TABLE 3 ABOUT HERE

This produces a range of estimates for the intergenerational elasticity of BMI between women and their children across the distribution of BMI_{g+1} as θ varies. Figure 2 displays estimates of the elasticity of BMI between generations for quantile regressions allowing θ to vary

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[21] Koenker and Hallock (2001).

[22] An example of quantile regression is a median regression which takes the median of the distribution of the dependent variable as its single quantile of interest.

[23] Stifel and Averett (2009) use quantile regressions to study the influence of a variety of correlates on childhood obesity across the distribution of BMI.

from .05 to .95 in uniform increments of .05 with the quantile cutoffs based on the distribution of the offspring's BMI in the YA NLSY79 data.[24] The estimated elasticities around the median of the distribution are similar to those estimated using ordinary least squares (from the elasticity column in Table 2a). The strength of the intergenerational relationship declines at quantiles below the median and increases significantly at higher quantiles in the BMI distribution. These results show a clear trend of an increased elasticity of BMI transmission at higher levels of the offspring's BMI. Table 3 provides results for selected quantile levels. The estimated elasticities around the 90th percentile (BMI levels just above the cutoff for obesity) vary from 0.53 for sons to 0.62 for daughters, with the elasticity estimate for the full sample more than twice as large at the 90th percentile as at the 10th percentile (where BMI levels are just above the cutoff indicating underweight). Thus, the estimated intergenerational dependence in BMI is strongest among offspring at the upper end of the distribution of BMI.[25] Such a result indicates that the degree of similarity in weight outcomes between generations is highest among those children who are obese or overweight in late adolescence.

B. Quadrant Dependence in Intergenerational BMI Transmission

While correlation coefficients, such as those presented in Section 3, provide a parsimonious measure of the direction and degree of dependence between random variables, it is possible that the strength of this relationship may vary substantially across BMI distributions as was found in the quantile regression results presented above. Summaries of associations based on measures of central tendency may conceal (possibly non-linear) conditional relationships in

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[24] Note that quantiles in this procedure are determined by the empirical distribution of BMI observed in the NLSY data, rather than from an external reference such as the CDC BMI growth charts.

[25] As indicated in Table 3, the 75th percentile of child's BMI in the YA NLSY surveys is above the threshold for overweight (BMI greater than 25), while the 90th percentile is above the cutoff for obesity classification (BMI greater than 30).

ranges of the distributions of most interest. This section describes and provides results for techniques that allow for measuring the degree of dependence between random variables at multiple points across the distributions of BMI in both generations. These measures, known as *quadrant dependence*, were developed in Lehmann (1966) and are useful for describing the comovement of two (or more) random variables. Allowing the strength of the association of BMI across generations to vary in a more flexible fashion will provide insights on areas of the distributions of interest where persistence in weight is strongest.

Formally, a pair of random variables, X and Y, with a distribution $F(X,Y)$ are said to exhibit *positive quadrant dependence* (PQD) if

$$\Pr(X \leq x, Y \leq y) \geq \Pr(X \leq x)\Pr(Y \leq y), \forall x, y$$

with strict PQD holding if the inequality is strict for at least one (x,y). Negative quadrant dependence (NQD) is defined analogously with the inequality reversed. The family of distribution functions for which PQD and NQD hold are defined as F_1 and G_1, respectively. Karlin (1983) further develops these measures by providing a technique allowing for comparisons of the degree of quadrant dependence between pairs of random variables. In order to implement measures of quadrant dependence empirically, indicator functions are employed to approximate the space of increasing functions defined by F_1. Following the notation of Karlin (1983), we define

$$\phi_b(x) = \begin{cases} 1 \ if \ x \geq b \\ 0 \ if \ x < b \end{cases} \quad \text{and} \quad \psi_b(y) = \begin{cases} 1 \ if \ y \geq b \\ 0 \ if \ y < b \end{cases}$$

where b varies within the support of x and y to generate a series of cutoffs. Correlations between the indicator functions $\phi_b(x)$ and $\psi_b(y)$ are then calculated for selected values of b in the support of x and y to measure the dependence between x and y across different segments of

their joint distribution. In the results presented here, *b* takes on the values of the 20th, 40th, 60th and 80th percentiles of the distributions of BMI for each generation. The measured correlations are then arranged into a 4 x 4 "association array" whose entries are the correlations between $\phi_i(x)$ and $\psi_j(y)$ for $i, j \in \{20,40,60,80\}$. With $r_{ij} = r(\phi_i(x), \psi_j(y))$ representing the correlation between $\phi_i(x)$ and $\psi_j(y)$, arrays of the dependence between random variables are organized from $r_{20,20}$ in the bottom left corner to $r_{80,80}$ in the upper right corner with *i* increasing from the bottom to the top of the array and *j* increasing from left to right in the array.

Variables are said to exhibit *strong* PQD if all of the entries in the array are strictly positive. For comparison of the strength of dependence in two populations A and B, Karlin argues that if all of the entries in an association array for population A are strictly greater than the entries in the array for population B then outcomes are more associated in population A than B.

INSERT TABLES 4 AND 5 ABOUT HERE

Comparisons of the degree and direction of quadrant dependence in BMI between generations by gender and ethnic groups indicate strong positive quadrant dependence in all cases (Tables 4 and 5). The magnitude of dependence generally increases toward the upper right in each table indicating stronger dependence at higher BMI levels. Comparing the degree of PQD for daughters and sons in Table 4, BMI is more associated between women and their daughters than women and their sons.[26] No evident differences exist when comparing the quadrant dependence of BMI between ethnic groups in Table 5.

[26] Each entry in the association array for daughters is larger than the corresponding entry in the association array for sons.

V. Intergenerational Obesity and Overweight Transmission

This section provides results for the empirical probabilities of transitions between weight status categories across generations. While the previous analysis provided correlations and measures of the dependence of BMI levels across generations, this section considers the distribution of categorical weight outcomes for young adults conditional on their mother's weight status when she was at the same stage of life. Individuals with average BMI levels above 30 are classified as obese and those with BMI between 25 and 30 are categorized as overweight.

Distributions of offspring weight status conditional on maternal weight status indicate a high degree of persistence in weight problems across generations (Table 6). These results indicate that 70% of children born to women who were obese in early adulthood became obese or overweight themselves by early adulthood which is more than double the rate for children born to women who were in the recommended range of BMI at this stage of life. However, in accord with the expansion in rates of obesity and overweight during the 25 years covered by these data, more than 30% of children born to mothers who were of recommended BMI in early adulthood were obese or overweight when they were at a similar stage of development. Results using the custom NLSY sample weights indicate similar patterns in the conditional distributions of weight outcomes across generations with slightly lower rates of weight problems when the oversampling of black and Hispanic mothers is accounted for (Table A2).

INSERT TABLE 6 ABOUT HERE

Estimates of probit models of offspring obesity (in which the dependent variable equals 1 if the BMI of the offspring is greater than 30) and obese or overweight (where the dependent variable equals one if the offspring's BMI is greater than 25) show large marginal effects of maternal weight status on the weight problems of offspring (Tables 7 and 8). Note that the

dependent variable indicating an overweight or obese offspring in Table 8 includes all offspring with BMI above 25, while the mother's overweight variable used as a regressor includes only those with BMI values between 25 and 30. The calculated marginal effects measure changes in the probability of being obese or overweight for children whose mothers had BMI levels outside of the recommended range. Results are provided for the entire sample, separately by gender and by ethnic categories for each gender given the oversample of certain demographic groups in this sample.

INSERT TABLES 7 AND 8 ABOUT HERE

Not surprisingly, having a mother who was obese between ages 16 and 24 significantly increases the likelihood that her child will also be overweight or obese at a similar stage of life in all specifications (Tables 7 and 8). The magnitude of the increase in the likelihood of obesity for a child born to a mother who was obese in early adulthood (relative to one with a recommended BMI level) ranges from 17% for white females to 38% for black females (Table 7). The estimated influence of having an obese mother is similar across the three demographic categories for sons. Having a mother who was overweight (with a BMI between 25 and 30) significantly increases the likelihood of becoming obese for both white and black females and males, but does not have a statistically significant effect for the likelihood of obesity among Hispanics.

Expanding the classification of weight problems to include both obese and overweight (BMI greater than 25) offspring, the estimated probit results indicate that children of obese mothers are 38% more likely to be overweight or obese (Table 8). Conversely, mothers with BMI levels below 18.5 (classified as underweight) are significantly less likely to have obese or overweight children than women with BMIs in the recommended range between 18.5 and 25. Being born to an obese or overweight mother significantly increases the likelihood of becoming

obese or overweight by early adulthood for all six demographic groups considered, with the magnitude of the effects similar across all three racial and ethnic categories for each gender. The measured reductions in the likelihood of developing weight problems among the offspring of a mother who was underweight are large and statistically significant among Hispanic males and females.

VI. Conclusions and Directions for Further Research

This paper provides several measures of the intergenerational persistence of weight problems that have been argued to causally influence economic success. If elevated parental BMI levels reduce familial resources (for instance, via a reduction in wages for obese workers or an increased likelihood of disability), then economic success for the subsequent generation may be limited by an increased likelihood of weight problems due both to genetic predisposition as well as to resource constraints that bias caloric consumption towards less healthy foods. This process of intergenerational health capital transmission may then serve to explain a portion of the relatively low levels of economic mobility found in studies over the last two decades (beginning with Solon (1992) and Zimmerman (1992)).

The estimated degree of correlation in BMI across generations in the NLSY data used in this study is roughly 0.35 and in line with previous epidemiological studies of the heritability of obesity. A significantly higher correlation of BMI between mothers and their daughters (0.38) than with their sons (0.32) was found. While there exists the possibility that bias in the self-reports of weight in the NLSY data leads to an overstated correlation, the intergenerational dependence is large across the entire range of the BMI distribution. The transmission of obesity appears to be most persistent at the highest levels of obesity. Quantile regressions indicate that toward the upper end of the distribution of BMI, the elasticity of offspring BMI with respect to

mother's BMI is above 0.5. Measures of quadrant dependence provide additional evidence that the strength of the relationship in BMI across generations is increasing at higher points in the distribution of BMI.

The data employed in these measurements also reflect the dramatic increase in obesity in the United States population during the period studied. Roughly 25% of the mothers in the NLSY79 were overweight or obese between ages 16 to 24, while 37% of their offspring were classified as overweight or obese by the same age range and rates of obesity have more than doubled between these two generations. Additionally, 70% of the offspring born to women who were obese were themselves at least overweight by early adulthood. Children born to women who were obese in early adulthood are more than 30% more likely to themselves be obese, relative to children born to women with a recommended BMI level.

The research presented in this paper provides estimates of the dependence of weight outcomes across two generations when both generations are at similar stages of the lifecycle. Given previous estimates of a significant causal effect of obesity on income and wages for females as well as correlations between obesity and accumulation of wealth and education, the substantial intergenerational persistence in BMI, especially at its highest levels, may provide a pathway to explain some portion of reduced levels of economic mobility. Future studies using the NLSY and other longitudinal data will be able to measure intergenerational relationships in economic mobility measured by income, wealth and education controlling for the strong persistence in weight outcomes found in this paper.

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Appendix Tables

INSERT TABLE A1 ABOUT HERE

INSERT TABLE A2 ABOUT HERE

Tables

Table 1 - Summary Statistics for NLSY Sample

	NLSY79 Mothers (n = 2,560)		YA NLSY79 Children (n = 4,748)	
	Mean	*Std. Dev.*	*Mean*	*Std. Dev.*
Avg. BMI, age 16-24	23.0	3.9	24.7	5.0
Percent Overweight *BMI>25*	24.3%		37.2%	
Percent Obese *BMI>30*	5.7%		13.1%	
White	42.0%		39.5%	
Black	36.0%		37.2%	
Hispanic	22.0%		23.3%	
Male			50.6%	

Table 2a - Intergenerational BMI Elasticities and Correlations by Ethnicity and Gender, 1981 - 2004

Relationship	Sample Size	Elasticity β_1	Std. Error	R^2	p-value of difference by gender	Correlation in BMI ρ_{BMI}
Full Sample						
Mother-Child	4,748	0.420	0.019	0.122		0.350
Mother-Daughter	2,348	0.486	0.030	0.144	} 0.001	0.379
Mother-Son	2,400	0.356	0.025	0.102		0.319
Whites						
Mother-Child	1,875	0.401	0.033	0.106		0.326
Mother-Daughter	923	0.433	0.052	0.114	} 0.346	0.338
Mother-Son	952	0.370	0.042	0.101		0.318
Blacks						
Mother-Child	1,769	0.403	0.030	0.120		0.346
Mother-Daughter	909	0.455	0.045	0.129	} 0.035	0.359
Mother-Son	860	0.332	0.038	0.103		0.321
Hispanics						
Mother-Child	1,104	0.406	0.041	0.110		0.332
Mother-Daughter	516	0.465	0.064	0.138	} 0.208	0.371
Mother-Son	588	0.358	0.055	0.090		0.300

Table 2b - Intergenerational BMI Elasticities and Correlations by SES measures

	Sample Size	Elasticity β_1	Std. Error	R^2	p-value of F-test for all coeff. equal	p-value of F-test for higest-lowest diff.	Correlation in BMI ρ_{BMI}
Income Level (Child aged 2-18)							
<25th percentile	1,399	0.36	0.032				0.314
25-50th percentile	1,280	0.38	0.033	0.130	0.183	0.105	0.324
50-75th percentile	1,140	0.46	0.045				0.363
>75th percentile	810	0.47	0.061				0.359
Mother's Education Level							
Less than high school	713	0.35	0.044				0.320
High School	2,371	0.44	0.025	0.126	0.123	0.042**	0.368
Some college	1,136	0.39	0.048				0.299
College graduate	409	0.53	0.079				0.404
Poverty Level (while Child age 2-18)							
75-100% of time in poverty	883	0.37	0.039				0.334
25-75% of time in poverty	1,045	0.39	0.039	0.129	0.071*	0.034**	0.319
1-25% of time in poverty	647	0.35	0.051				0.287
Never in poverty	2,054	0.47	0.033				0.374
vs. Ever in Poverty	2,575	0.38	0.025		0.021**	0.021**	0.326

Notes: Income quartiles are not uniformly distributed due to quartiles being calculated for entire YA NLSY79 sample.
* indicates statistically significant difference at 10% level, ** indicates statistically significant difference at 5% level

Table 3 - Quantile Regression Results for Intergenerational Elasticity of BMI

Dependent variable is natural log of child's average BMI from age 16-24 and elasticity reported is coefficient on natural log of mother's average BMI from 16-24, n = 4,748 observations

	Quantile					
Full Sample	***10***	***25***	***50***	***75***	***90***	OLS
Elasticity	0.27	0.33	0.43	0.53	0.58	0.42
Std. Error	(0.036)	(0.027)	(0.024)	(0.026)	(0.038)	(0.019)
Females	***10***	***25***	***50***	***75***	***90***	OLS
Elasticity	0.32	0.41	0.50	0.62	0.62	0.49
Std. Error	(0.045)	(0.044)	(0.036)	(0.046)	(0.060)	(0.030)
Males	***10***	***25***	***50***	***75***	***90***	OLS
Elasticity	0.20	0.27	0.38	0.43	0.53	0.36
Std. Error	(0.050)	(0.027)	(0.022)	(0.031)	(0.061)	(0.025)

	BMI Value at Quantile Cutoff					
	10	***25***	***50***	***75***	***90***	Mean BMI
Full Sample	19.6	21.2	23.6	26.9	31.3	24.7
Females	19.2	20.9	23.3	27.3	32.0	24.7
Males	19.9	21.5	23.7	26.6	30.6	24.7

Note: Standard errors for quantile regressions are generated via bootstrap replications.

Table 4 - Measures of Quadrant Dependence in BMI Transmission

Full Sample
(Corr = 0.35)
(n = 4,748)

Mother's BMI	*Child's BMI Percentile*			
Percentile	20	40	60	80
80	0.135	0.205	0.232	0.231
60	0.160	0.226	0.235	0.222
40	0.166	0.214	0.215	0.190
20	0.160	0.194	0.178	0.124

Daughters
(Corr = 0.38)
(n = 2,348)

Mother's BMI	*Daughter's BMI Percentile*			
Percentile	20	40	60	80
80	0.171	0.241	0.260	0.238
60	0.213	0.260	0.261	0.232
40	0.216	0.234	0.236	0.222
20	0.201	0.201	0.192	0.153

Sons
(Corr = 0.32)
(n = 2,400)

Mother's BMI	*Son's BMI Percentile*			
Percentile	20	40	60	80
80	0.097	0.171	0.205	0.222
60	0.105	0.193	0.209	0.211
40	0.116	0.197	0.195	0.155
20	0.124	0.193	0.165	0.094

Table 5 - Quadrant Dependence Measures of BMI Transmission by Race

Whites

(Corr = 0.33)
(n = 1,875)

Mother's BMI	*Child's BMI Percentile*			
Percentile	20	40	60	80
80	0.128	0.211	0.226	0.227
60	0.167	0.237	0.234	0.228
40	0.174	0.214	0.217	0.178
20	0.156	0.182	0.161	0.102

Blacks

(Corr = 0.35)
(n = 1,769)

Mother's BMI	*Child's BMI Percentile*			
Percentile	20	40	60	80
80	0.132	0.193	0.217	0.231
60	0.129	0.191	0.207	0.215
40	0.121	0.176	0.164	0.189
20	0.112	0.169	0.164	0.148

Hispanics

(Corr = 0.33)
(n = 1,104)

Mother's BMI	*Child's BMI Percentile*			
Percentile	20	40	60	80
80	0.115	0.171	0.221	0.202
60	0.146	0.202	0.225	0.193
40	0.162	0.207	0.231	0.176
20	0.196	0.213	0.189	0.097

Table 6 - Distributions of Intergenerational Weight Status Transitions

Full Sample

			Child's Weight Status				
	BMI		<18.5	18.5-25	25-30	>30	Mother's Distribution
		Category	Underweight	Recommended	Overweight	Obese	
Mother's	<18.5	Underweight	11.6%	71.9%	12.6%	3.9%	6.0%
Weight	18.5-25	Recommended	4.4%	64.2%	22.1%	9.4%	69.6%
Status	25-30	Overweight	1.9%	42.9%	33.3%	21.9%	18.7%
	>30	Obese	2.2%	28.4%	29.9%	39.5%	5.7%
	Child's Distribution		4.2%	58.6%	24.1%	13.1%	N=4,748

Daughters

			Daughter's Weight Status				
	BMI		<18.5	18.5-25	25-30	>30	Mother's Distribution
		Category	Underweight	Recommended	Overweight	Obese	
Mother's	<18.5	Underweight	17.1%	67.9%	11.4%	3.6%	6.0%
Weight	18.5-25	Recommended	6.4%	63.8%	19.5%	10.3%	68.4%
Status	25-30	Overweight	2.2%	41.5%	31.4%	25.0%	19.4%
	>30	Obese	2.8%	28.3%	25.5%	43.5%	6.2%
	Daughter's Distribution		6.0%	57.5%	21.7%	14.8%	N=2,348

Sons

			Son's Weight Status				
	BMI		<18.5	18.5-25	25-30	>30	Mother's Distribution
		Category	Underweight	Recommended	Overweight	Obese	
Mother's	<18.5	Underweight	6.2%	75.9%	13.8%	4.1%	6.0%
Weight	18.5-25	Recommended	2.4%	64.6%	24.5%	8.5%	70.7%
Status	25-30	Overweight	1.6%	44.4%	35.4%	18.5%	18.0%
	>30	Obese	1.6%	28.6%	34.9%	34.9%	5.3%
	Son's Distribution		2.5%	59.8%	26.3%	11.5%	N=2,400

Note: Weight Status for both generations are determined based on average BMI levels between ages 16 and 24. Data are from NLSY79 and Young Adult offspring of women in NLSY79.

Table 7 - Marginal Effects of Maternal Weight Status for Probit Estimates of Offspring's Obesity (BMI > 30)
Change in likelihood of offspring's obesity relative to mother in recommended weight range (18.5 < BMI < 25)

	Full Sample	All Females	All Males
Maternal Weight Status, age 16-24			
Obese	31.7%	34.9%	27.7%
BMI>30	(12.31)**	(9.16)**	(7.79)**
Overweight	12.9%	15.2%	10.3%
25<BMI<30	(8.61)**	(6.86)**	(5.54)**
Underweight	-7.0%	-8.7%	-5.4%
BMI<18.5	(3.28)**	(2.71)**	(1.90)
Observations	4,748	2,348	2,400
Percent Obese	13.1%	14.8%	11.5%
(Fraction of Dependent Variable = 1)			
		Females	
	White	Black	Hispanic
Maternal Weight Status, age 16-24			
Obese	16.9%	37.8%	32.4%
BMI>30	(2.78)**	(6.91)**	(3.76)**
Overweight	21.7%	12.5%	7.2%
25<BMI<30	(6.08)**	(3.29)**	(1.85)
Underweight	-3.0%	-17.1%	-6.2%
BMI<18.5	(0.86)	(2.13)*	(1.00)
Observations	923	909	516
Percent Obese	9.8%	21.8%	11.4%
(Fraction of Dependent Variable = 1)			
		Males	
	White	Black	Hispanic
Maternal Weight Status, age 16-24			
Obese	26.9%	28.6%	30.3%
BMI>30	(3.87)**	(5.51)**	(4.33)**
Overweight	10.2%	13.2%	6.8%
25<BMI<30	(3.33)**	(4.34)**	(1.85)
Underweight	-7.6%	0.6%	-7.2%
BMI<18.5	(2.01)*	(0.11)	(0.97)
Observations	952	860	588
Percent Obese	10.9%	10.9%	13.1%
(Fraction of Dependent Variable = 1)			

Robust z-statistics corrected for multiple children from same mother in parentheses.
* significant at 5%; ** significant at 1%

Table 8 - Marginal Effects of Maternal Weight Status for Probit Estimates of Likelihood of Obese/Overweight Child (BMI > 25)
Change in likelihood of overweight or obese offspring relative to mother in recommended weight range (18.5 < BMI < 25)

	Full Sample	All Females	All Males
Maternal Weight Status, age 16-24			
Obese	37.7%	38.9%	36.6%
BMI>30	(11.56)**	(8.80)**	(7.25)**
Overweight	23.7%	26.6%	20.8%
25<BMI<30	(11.60)**	(9.04)**	(7.47)**
Underweight	-16.8%	-17.0%	-16.5%
BMI<18.5	(4.92)**	(3.53)**	(3.45)**
Observations	4,748	2,348	2,400
Percent Obese or Overweight (BMI > 25) *(Fraction of Dependent Variable = 1)*	37.2%	36.6%	37.8%
		Females	
	White	Black	Hispanic
Maternal Weight Status, age 16-24			
Obese	33.9%	35.6%	32.5%
BMI>30	(3.96)**	(6.23)**	(3.19)**
Overweight	28.3%	24.0%	19.3%
25<BMI<30	(5.69)**	(5.35)**	(3.26)**
Underweight	-9.2%	-16.1%	-27.6%
BMI<18.5	(1.42)	(1.96)*	(2.73)**
Observations	923	909	516
Percent Obese or Overweight (BMI > 25) *(Fraction of Dependent Variable = 1)*	26.0%	47.7%	35.9%
		Males	
	White	Black	Hispanic
Maternal Weight Status, age 16-24			
Obese	28.5%	38.0%	38.9%
BMI>30	(2.92)**	(5.62)**	(3.23)**
Overweight	24.3%	19.1%	17.4%
25<BMI<30	(4.96)**	(4.20)**	(3.47)**
Underweight	-13.1%	-14.3%	-26.5%
BMI<18.5	(2.03)*	(1.64)	(2.30)*
Observations	952	860	588
Percent Obese or Overweight (BMI > 25) *(Fraction of Dependent Variable = 1)*	33.4%	40.0%	41.8%

Robust z-statistics corrected for multiple children from same mother in parentheses.
* significant at 5%; ** significant at 1%

Table A1 - Weighted Intergenerational BMI Elasticities and Correlations by Ethnicity and Gender, 1981 - 2004

Relationship	Elasticity β_1	Std. Error	R^2	p-value of difference by gender	Correlation in BMI ρ_{BMI}
Full Sample					
Mother-Child	0.414	0.022	0.117		0.342
Mother-Daughter	0.479	0.034	0.137	} 0.005	0.370
Mother-Son	0.356	0.028	0.100		0.316
Whites					
Mother-Child	0.403	0.034	0.106		0.326
Mother-Daughter	0.444	0.054	0.117	} 0.259	0.343
Mother-Son	0.367	0.042	0.099		0.314
Blacks					
Mother-Child	0.392	0.030	0.111		0.333
Mother-Daughter	0.437	0.045	0.116	} 0.064	0.340
Mother-Son	0.332	0.037	0.102		0.320
Hispanics					
Mother-Child	0.388	0.042	0.105		0.324
Mother-Daughter	0.461	0.068	0.143	} 0.146	0.379
Mother-Son	0.324	0.058	0.077		0.277

Note: Results use longitudinal custom sample weights from NLSY YA data for 1986-2004.

Table A2 - Weighted Distributions of Intergenerational Weight Status Transitions

Full Sample

			Child's Weight Status				
	BMI		<18.5	18.5-25	25-30	>30	Mother's Distribution
		Category	Underweight	Recommended	Overweight	Obese	
Mother's	<18.5	Underweight	12.7%	70.8%	12.4%	4.1%	6.9%
Weight	18.5-25	Recommended	4.7%	66.4%	20.4%	8.6%	70.8%
Status	25-30	Overweight	2.3%	43.9%	32.0%	21.9%	17.5%
	>30	Obese	2.4%	32.7%	29.1%	35.9%	4.8%
	Child's Distribution		4.7%	61.1%	22.3%	11.9%	

Daughters

			Daughter's Weight Status				
	BMI		<18.5	18.5-25	25-30	>30	Mother's Distribution
		Category	Underweight	Recommended	Overweight	Obese	
Mother's	<18.5	Underweight	19.2%	66.6%	9.7%	4.5%	6.8%
Weight	18.5-25	Recommended	7.3%	66.4%	17.6%	8.7%	70.1%
Status	25-30	Overweight	2.7%	43.2%	28.8%	25.3%	18.0%
	>30	Obese	3.6%	32.7%	26.7%	37.1%	5.1%
	Daughter's Distribution		7.1%	60.5%	19.5%	12.9%	

Sons

			Son's Weight Status				
	BMI		<18.5	18.5-25	25-30	>30	Mother's Distribution
		Category	Underweight	Recommended	Overweight	Obese	
Mother's	<18.5	Underweight	6.6%	74.7%	14.9%	3.8%	7.0%
Weight	18.5-25	Recommended	2.2%	66.3%	23.0%	8.5%	71.5%
Status	25-30	Overweight	1.8%	44.6%	35.2%	18.5%	17.0%
	>30	Obese	4.5%	32.7%	31.7%	34.5%	4.5%
	Son's Distribution		2.4%	61.7%	24.9%	11.0%	

Note: Weight Status for both generations are determined based on average BMI levels between ages 16 and 24. Data are weighted using longitudinal custom sample weights for NLSY YA from 1986 to 2004.