Weight Gain in Adolescents and Their Peers

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Despite the urgent public health implications, relatively little is yet known about the effect of peers on adolescent weight gain. We describe trends and features of adolescent BMI in a nationally representative dataset and document correlations in weight gain among peers. We find strong correlations between own body mass index (BMI) and peers' BMI's. Though the correlations are especially strong in the upper ends of the BMI distribution, the relationship is smooth and holds over almost the entire range of adolescent BMI. Furthermore, the results are robust to the inclusion of school fixed effects and basic controls for other confounding factors such as race, sex, and age. Some recent research in this area considers whether or not adolescent weight gain is caused by peers. We discuss the econometric issues in plausibly estimating such effects while accounting for growth spurts and difficulties in defining adolescent obesity. While our work identifies correlations between adolescent BMI and peers' BMI, it is not intended to and cannot fully address the existence of endogenous peer effects.

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I. Introduction

Since the 1960's, obesity rates among children in the United States have risen dramatically. The Centers for Disease Control and Prevention (CDC) reports that while fewer than 5% of children ages six through eleven were considered obese in 1963, over 15% are considered obese today. Among adolescents, similar trends hold; obesity rates for this group were just under 5% in 1966 and are well over 15% today. Rates of overweight have increased for all groups of children, for both boys and girls and across all ages and races. This increase is especially pronounced among black girls (Komlos et al, 2009). In addition, the rate at which children gain weight as they grow older has increased over time (Anderson et al, 2003).

Researchers are particularly interested in pinpointing the causes of this trend because of the steep costs associated with it. Obesity puts children at risk for a myriad of physical diseases such as cardiovascular disease, type 2 diabetes, hypertension, dyslipidemia, sleep apnea, menstrual abnormalities, osteoarthritis, and lower life expectancy as well as emotional problems such as depression, social marginalization, and low self-esteem (Must and Strauss, 1999). Also, obese children often grow up to be obese adults (Serdula, 1993). Economic estimates of the cost of the overall obesity epidemic in the United States currently range in the billions of dollars[1] and will likely continue to rise. Furthermore, there appears to be a public willingness to deal with the problem. For example, Cawley (2008) estimates public willingness-to-pay for a 50% reduction in childhood obesity equal to $690.6 million in New York State.

Researchers have identified low levels of exercise and high calorie intake as prime causes of childhood obesity. Gordon-Larsen et al (2002) show a correlation between low frequency of exercise and obesity in adolescents. Other studies show that higher calorie intake also leads to higher rates of obesity. Whitmore (2008) estimates that school lunches contain 40 to 120 more

[1] See for example, http://www.cdc.gov/nccdphp/dnpa/obesity/economic_consequences.htm.

calories than lunches packed at home. She claims this difference results in a two to four percentile point increase in the incidence of obesity among children who consume school lunches.

Some researchers also point to the changing composition of calories consumed by adolescents. For example, adolescents consume more fat (Donnelly et al, 2008; Donahoo et al, 2008) and more 'empty' calories such as sodas (Troiano et al, 2000). Less obvious are the causes behind the growing propensity of children to eat more and exercise less. Researchers point to television viewing habits (Gordon-Larsen et al, 2002; Anderson and Butcher, 2006), the increased advertising and availability of fast food (Pereira et al, 2005; Robinson et al, 2007), and the increased availability of calorie-dense foods at school (Anderson and Butcher, 2006) as prime culprits. Gordon-Larsen et al (2002) note these behaviors vary by sex and ethnicity, perhaps shedding additional light on factors that may contribute to the propensity to eat more and exercise less. In addition, economists show the relationship between rising obesity rates and mother's labor supply (Anderson et al, 2003), problems in school finance and accompanying soft drink and snack vending contracts (Anderson et al, 2003), and improvements in technology that lower the time price of food (Lakdawalla and Philipson, 2002; Cutler et al, 2003; Komlos et al, 2004). Researchers find no single culprit for increasing weight gain, but rather many complementary changes which lead to higher calorie intake and lower levels of exercise (Anderson and Butcher, 2006).

A recent study in the New England Journal of Medicine (NEJM) explores another possible factor in rising obesity rates, the effect of peers on weight gain (Christakis and Fowler, 2007). Researchers use data from the Framingham Heart Study to show that overweight and obese adults in Framingham, Massachusetts tend to cluster together socially. Using this evidence, the authors argue for the existence of endogenous peer effects in the spread of obesity. The existence (or not) of peer effects in rising obesity rates is important for policy. If gaining weight causes one's friends to gain weight, policy interventions intended to reduce obesity rates affect targeted subjects as well

as their friends and consequently have multiplier effects. Three additional papers explore this relationship. Cohen-Cole and Fletcher (2008) refute the existence of peer effects in weight gain among adolescents while Trogdon, Nonnemaker, and Pais (2008) and Renna, Grafova, and Thakur (2008) find significant peer effects particularly among girls and among adolescents with high body mass indexes[2] (BMI). A fourth paper, Fowler and Christakis (2008) attempts to replicate Cohen-Cole and Fletcher's results using identical data, but arrives at different conclusions.

In this paper, we document some trends in adolescent BMI and document correlations in weight gain among peers. The main contributions of the paper are as follows. First, we the discuss the analyses in Christakis and Fowler (2007), Cohen-Cole and Fletcher (2008), and Trogdon, Nonnemaker, and Pais (2008) and compare their results to our own. We provide interpretations of the seemingly disparate results across the three papers. We discuss the difficulties in empirically identifying causal peer effects in weight gain and focus on a careful and thorough set of descriptive results documenting the distribution and other features of adolescent BMI and their relationship to peer BMI.

We show that changes in adolescent BMI are highly susceptible to adolescent growth spurts. As a result, we believe it is inaccurate to treat this data using adult definitions of overweight. Instead, we use definitions of overweight created especially for adolescents by the Centers for Disease Control and Prevention (CDC) and are careful to account for these biological factors in our description of the correlations we analyze. While our work identifies correlations between adolescent BMI and peers' BMI, it is not intended and cannot address the existence of causal peer effects.

In our summary of the existing literature, we discuss four main points. First, regressions in levels are apt to be biased by omitted variables but regressions in differences are apt to be biased by

[2] BMI is a commonly used measure of weight in the obesity literature. It is an index that measures weight in kilograms normalized by height in meters.

measurement errors. Second, most results vanish when individual fixed effects are used because this eliminates much of the variation available to estimate the effects of interest. Third, IV results that use omitted contextual effects are not robust and because they are in levels are susceptible to omitted variables bias. Fourth, use of dynamic regressions is also an imperfect solution to the problems inherent in identifying the casual effect of peers on obesity.

The remainder of the paper is organized as follows: We describe our data in section II, including a discussion of our construction of the peer group variables. In section III, we discuss our methods and in section IV we present our empirical findings. Section V concludes.

II. Data

We use data from the National Longitudinal Adolescent Health Survey (Add Health). The Add Health survey was conducted by the Carolina Population Center and is available for a nationally representative sample of students who were in seventh through twelfth grades in 1994.[3] Wave I, which was fielded in 1994-1995, consists of an In-School questionnaire that was filled out by 90,118 students in 145 schools in 80 communities. A subset of 20,745 students was then chosen for an in-depth In-Home survey. The In-Home survey contains information on self-reported body weight and height.

Wave II, which was fielded in 1996, includes an In-Home questionnaire that was completed by 14,738 students, a subset of the original 20,745 Wave I pupils.[4] The full dataset consists of 14,738 students who were surveyed In-Home in both Wave I and Wave II. We include in our sample those students who have full information on height and weight and who have at least one

[3] For more information on accessing the data, please see http://www.cpc.unc.edu/projects/addhealth/data

[4] The 145 schools in the Wave I survey consist of pairs of sister schools. That is, if a particular high school was included in the survey, the corresponding feeder junior high or middle school was also included. If a school spanning seventh through twelfth grades was chosen for the survey, no sister school was included.

friend with full information on height and weight. This reduces our sample to 4,617 students in Wave I and 2,970 students in Wave II. Table 1 presents some basic summary statistics for our sample and for the full sample.

Body Mass Index

We construct a Body Mass Index (BMI) variable according to the formula: $\text{BMI} = \frac{\text{weight in lbs}}{\text{height in inches}^2} * 703$.[5] A weakness of the survey is that the height and weight measures are self-reported. As a result, we expect biases to differ by sex; for example, we might expect boys to systematically overstate their heights while girls may systematically understate their weights. We are unable to confidently determine the exact direction or magnitude of these biases. However, we report our results by sex in order to facilitate comparisons of the two groups.

For children and adolescents, the medical literature defines "overweight" as being above the 95th percentile in the weight distribution by sex and age. Similarly, "at risk of overweight" is defined as being above the 85th percentile in the weight distribution by sex and age. Following this convention, we use BMI measures to construct two additional variables: "overweight" which we define to be above the 85th percentile of BMI for the appropriate age-sex group and "underweight" which corresponds to being below the 10th percentile of BMI for the appropriate age-sex group. The BMI cutoffs are derived from age and sex specific distributions; we use the distributions given in the 2000 CDC Growth Charts (see Appendix A). These alternative cut-off points account for the growth spurts children experience in adolescence.

Figures 1 displays the distribution of BMI for males and females in our sample. While BMI tends to be concentrated in the low twenties, there are substantial numbers of adolescents with

[5] BMI is calculated as weight in kilograms normalized by height in meters. Because height and weight in the Add Health survey are recorded in inches and pounds, we use a conversion factor of 703.

BMI's above 25; the densities for both girls and boys exhibit fat upper tails. This is especially striking since BMI percentile cutoff points for whether or not an individual is considered overweight or obese increase with age for adolescents (see Appendix A).[6] Average BMI in the overall sample is 22.59 in Wave I of the survey and 22.95 in Wave II (Table 2).

Self-Reported Health Status

The measure of self-reported health status in the survey is a categorical variable. It takes on the value 1 if the student rates his own health as 'excellent', 2 if 'very good', 3 if 'good', 4 if 'fair', 5 if 'poor'. Despite the problems associated with self-reporting and with the use of categorical, qualitative measures of health, there is some evidence in the literature that self-reported health status is a reliable measure of actual health status (Deaton and Paxson, 1998). We examine the relationship between self-reported health status and BMI. We plot three kernel regressions of self-reported health status on BMI: one for the full sample, another for boys and for girls (Figure 2). For boys, the relationship is upward sloping and quite smooth. For girls, the relationship is u-shaped. This is consistent with the presence of anorexic girls in the data who report poor health which improves over a narrow range of weight gain. In general, BMI and self-reported health status move together so that as BMI increases, the health of the individual worsens.

We create a binary indicator variable for self-reported bad health. Probit regression indicate correlations between self-reported bad health and BMI (Table 3). We find that these are strongly positive, significant, remarkably stable and robust to inclusion of controls for sex, race, and socioeconomic status as measured by parental education. Though we conduct the remaining

[6] For example, a 15 year old boy with a BMI of 23 is considered overweight, but a 21 year old man with the same BMI is not. Thus, the high number of adolescents with BMI's in the upper tail of the distribution is even more worrisome than it would be in a sample of adults. Using adult definitions of overweight and obese in this setting will cause us to significantly underestimate the rates of overweight and obese adolescents in our sample.

analysis for measures of overweight and BMI, these demonstrated correlations between adolescent BMI and self-reported health status suggest that our analysis has implications for not just weight but overall health.

Adolescent Growth Spurts

Because of the rapid changes in height and weight accompanying adolescent growth spurts, the task of identifying causal peer effects in weight gain is more complicated than in adults. We observe a strictly down-ward sloping relationship between change in BMI and change in height between Wave I and Wave II survey years (Figure 3). In other words, those adolescents who experience the largest drops in BMI are also those who grow the most in height. Similarly, those who gain in BMI are those who experience little or no change in height. The larger issue is that analyses of correlations between own and peer weight fluctuations in adolescents are confounded by growth spurts.

Peer Networks

The Add Health survey is well-suited to our purposes because of the extensive data on friendship networks. In each of the surveys, students are asked to nominate five female friends and five male friends. In almost all cases, students report fewer than five male and five female friends indicating that they are not constrained in their choice of friends in their network by the ten-friend limit. The average number of friends nominated by individuals in our sample is 2.10 (with a standard deviation of 1.76). These friend nominations include both friends in the same school as well as friends from outside of school. Because we do not have information on friends outside of the respondent's school, we are unable to include them in our measure of average peer group BMI. However, the vast majority of friend nominations (approximately 85%) are to other students in the

same school. There are a sizeable number of nominations to friends that are not found on the school rosters. This may be due to nicknames not matching official names, students who are new to the school, or errors in the school records.

Our construction of peer groups uses information from the Add Health friendship network to derive average levels of BMI across self-nominated friends. This definition only includes friends directly nominated by the respondent and is limited to at most five male and five female friends.

III. Methods

We begin with standard fixed-effects estimation equations from the peer effects literature[7]:

$$y_{ist} = \bar{y}_{ist}\beta + x_{is}\lambda + \delta_s + \varepsilon_{ist}$$

$$y_{ist} = \bar{y}_{ist}\beta + x_{is}\lambda + \alpha_i + \varepsilon_{ist}$$

where y_{ist} is either BMI or some function of it for pupil *i* in school *s* at time *t*, $\bar{y}_{ist}$ is the average of y_{ist} in the peer network, x_{is} is a vector of controls, α_i is an individual fixed effect and δ_s is a school dummy. The parameter β is a measure of endogenous peer effects. Of course, as has been discussed by Manski (1995) and others, OLS estimation of this model will not identify β correctly for two primary reasons. First, in the presence of endogenous effects, average obesity in the network is an equilibrium outcome and as a result, the residual in the equation is correlated with $\bar{y}_{ist}$. Second, because pupils tend to choose friends with similar characteristics that are also correlated with behavior, there are apt to be omitted variables in the residual that are correlated with both own and peer obesity. Manksi (1995) refers to this source of bias as "correlated effects."[8] Inclusion of

[7] With two time periods, the individual fixed effects results are equivalent to results using first differencing. Also, note that the inclusion of individual fixed effects automatically controls for school fixed effects, though not vice versa.

[8] There are other sources of bias as well. One source is that own behavior is positively correlated with the background characteristics of the peer group. Manski (1995) refers to these as "contextual effects." Another is that either the

the vector x_{is} accounts for observed characteristics that vary over time. The individual or school fixed effects account for factors that vary across individuals or across schools (respectively) but that do not vary across time. However, we are unable to account for unobserved factors that vary across individuals or schools and that also vary over time. The fixed effects should therefore mitigate, but not necessarily eliminate, bias in the estimates.[9]

To address these concerns, the standard approach is to look for a source of variation in peer behavior and then argue that this variation is exogenous. For example, one commonly used technique is to assume that certain background characteristics of the peer group do not enter the structural model and to use these variables as instruments (Gavrira and Raphael 2001; Trogdon et al, 2008; Renna et al, 2008). When examining obesity, however, these types of instruments are problematic because the relationship between obesity and background characteristics is confounded by biology. It is more difficult than usual to defend the needed exclusion restrictions. Furthermore, while a specification that instruments the second stage in first differences addresses many of the problems with omitted biological factors, it comes at the high cost of increased measurement error and even weaker instruments. Therefore, we forego instrumental variables methods and use school fixed effects techniques in levels rather than differences.

Arcidiacono and Nicholson (2005) use a similar approach. Unfortunately this method does not adequately solve the endogeneity problem. Because we are unable to solve the endogeneity problem, we are unable to provide unbiased estimates of the *causal* effect of peers on adolescents'

behavior within the group is poorly measured or that the definition of the peer group is a poor approximation of the true peer group. See Halliday and Kwak (2008) for a more thorough discussion of this source of bias.

[9] This comes at a cost, however. The use of fixed effects, especially individual effects, reduces much of the variation available for use in estimating the effects of interest.

weight gain or loss.[10] In the absence of bias due to measurement error, this inability to solve the endogeneity problem leads to upper bounds on the true endogenous effect. In addition, if the use of fixed effects only mitigates but does not fully account for correlated effects, the estimates will be further biased upwards.

Without credible estimates of a causal peer effect, we focus instead on the correlations in BMI between adolescents and their self-nominated peers. The fixed effects method allows us to consider these correlations while controlling for a variety of potentially confounding factors. In the following section, we examine these correlations at various percentiles of the BMI distribution; for overweight and underweight adolescents; and for boys and girls.

IV. Results

We estimate models for three different dependent variables: level of BMI, a binary indicator for whether overweight or not, and a binary indicator for whether underweight or not (Table 4). In all three cases, we include controls for gender, grade, race, and health status as well as dummies for grade and a school or individual fixed effect. Sample sizes for the regressions are smaller than the full sample because there is a substantial amount of missing information in the Add Health data. The school fixed effects regressions include control variables for race and health status and are thus substantially smaller than the individual fixed effects regressions which do not separately control for these factors. Reassuringly, the sample sizes are comparable to those in Cohen-Cole and Fletcher (2008) who also use the Add Health data.

Overall, overweight adolescents do tend to associate with one another. We report school fixed effects results for BMI and results for the propensity to be overweight (Table 4). Both sets of results show significant correlations between the average rates of overweight among friends and an

[10] Recent papers by Bramoulle et al (forthcoming) and Calvo-Armengol (forthcoming) attempt to use the network information in the Add Health data to identify these effects.

adolescent's own weight. On average, an increase of one point in the average friend BMI is associated with a 0.19 point increase in an adolescent's BMI. For a 5'9" 150 pound boy with 5'9" friends each weighing 150 pounds, this means an increase of seven pounds in average friends' weight is associated with an increase of about two pounds in own weight. Similarly, an increase of 10 percentage points in friends' propensity to be overweight is associated with a 1.1 percentage point increase in own propensity to be overweight.[11]

Christakis and Fowler (2007) conduct similar analyses of peer effects for adults and find similar results. They conclude that fat adults *cause* each other to be fat. However, we caution against interpreting the results in this way. Neither our results nor those of the NEJM study can differentiate between a causal effect of peers on weight gain and a story in which overweight adolescents choose overweight friends because they are socially ostracized by their slimmer peers. Both scenarios are equally consistent with the results.

We nonetheless see a strong tendency of overweight students to associate with overweight peers. This effect is robust to using different definitions of weight as the dependent variable and also holds for male and female sub-samples. We show kernel regression estimates of own BMI on average peer group BMI (Figure 4). The figures show a strong positive correlation between own and peer BMI for both girls and boys. Furthermore, the estimates for girls are stronger than for boys. This indicates that either overweight girls are more likely to choose overweight peers than boys are (either because they are ostracized by thinner girls or for other reasons), or girls are more likely than boys to become overweight as a result of having overweight friends. The correlations between own BMI and average peer BMI are consistent with either explanation (Table 4). We see these correlations disappear for underweight peers indicating that underweight adolescents do not

[11] Another way to specify these regressions is to look at *changes* in BMI and estimate the effect of peers on these changes. Such methods, while reducing the omitted variables problems of regressions in levels, fail to account for measurement error which is exacerbated by the first-differencing and yields biased and imprecise estimates.

associate with primarily underweight friends (Table 4). The contagion effects of eating disorders such as anorexia (if they do indeed exist) appear to occur through channels other than friendship networks.

Estimates of peer correlations using individual fixed effects are not significant (Table 4). In general, individual fixed effects more effectively eliminate omitted correlated effects in the residuals than do school fixed effects. However, we also eliminate much of the variation in BMI by including individual fixed effects. Addressing omitted variables bias in fixed-effects regressions come with many costs including: loss of observations, reducing variation in the right-hand side variables and exacerbating the attenuation bias of white noise measurement error (Deaton, 1995). For these reasons, we differ from Cohen-Cole and Fletcher (2008) and find the individual fixed effects results inconclusive.[12]

Next we estimate the models with school fixed effects separately by gender. Girls' tendency to be overweight is strongly associated with that of their female friends while boys' tendency is less so (point estimates are 0.08 versus 0.13 for girls) (Table 4). This pattern of results does not hold for the tendency to be underweight. These results are consistent with the kernel regressions in Figure 4.

We examine further the determinants of changes in weight (Table 5). The estimates measure correlations between change in own BMI and the level of peer BMI, change in own height, and baseline level of own BMI while controlling for race, school, gender, age, health status, and parental background information. In row 1, we see that again girls exhibit the largest correlations between

[12] There is an illuminating example from the development literature on how fixed-effects regression often find insignificant or zero effects, particularly when the variables in the estimation are measured with errors. Subramanian and Deaton (1996) estimate the income elasticity of a calorie. In the paper they argue that previous attempts that yielded an elasticity of zero do so precisely because they used fixed effects methods which tend to be inefficient. Moreover, Deaton (1995) provides evidence on how such procedures will exacerbate the attenuation bias due to the presence of measurement errors.

own and peer BMI measures (Table 5). More specifically, the girls who gain the most weight are also those who have friends that are overweight. This relationship is weaker in boys.

There is significant variation in change in BMI across our study years (Figure 5). A large portion of this variation is explained by changes in height and not changes in weight (Figure 3; Table 5, row 2). Note that omission of this height variable from the regression results in overestimates of the peer effect (Table 5). Because within-school clustering of students by height is likely the result of selection and not causal peer effects, the significant correlations between own height and peer height indicate that taller students (especially male ones) tend to select taller peers (Table 6). Thus, it is likely that students also tend to select peers by BMI.

We also examine the correlations between own and peer BMI at various cutoff points in the BMI distribution (Table 7). We see that correlations follow a smooth pattern and that there are no anomalous discontinuities in these correlations at any particular points in the weight distribution. There is a strong association of own BMI and the peer group's propensity to exceed the 90th percentile of the overall weight distribution. This correlation continues to be significant at the 85th percentile cutoff and the 75th percentile cutoff, though the size of the correlation diminishes monotonically (Table 7). These results also hold using a slightly different outcome variable (a dummy variable for whether the individual is overweight or not) (Table 8). The repeated results provide a useful robustness check.

V. Conclusions

We conclude that there is a tendency for students with high levels of BMI to cluster within peer networks. The correlations are strong between both males and females in our sample but are much stronger between overweight females (0.13) than overweight males (0.08).[13]

Due to problematic instruments in instrumental variables estimates, we rely on fixed effects models to control for various correlated and contextual effects. When school fixed effects are included in the models, we do find positive and significant estimates such as those noted above, but concerns about omitted correlated effects and the reflection problem suggest that these may be biased estimates of the true causal effect, if any exists. In the absence of measurement error, these estimates are upper bounds of the true causal effect and so we cannot rule out either positive or zero estimates. When individual fixed effects are included, point estimates drop to zero and the standard errors become large. However, this is due to the lack of efficiency of this estimator in this setting and cannot necessarily be interpreted as evidence of a zero causal peer effect.

We also find significant clustering of peers by height. These correlations are strongly significant with average point estimates of 0.48 between own and peer height in school fixed effects regressions and 0.05 in individual fixed effects regressions. The effect is particularly strong among males than females with point estimates of 0.08 versus 0.02. Because within-school clustering of students by height is likely the result of selection and not causal peer effects, the significant correlations between own height and peer height indicate that taller students (especially male ones) tend to select taller peers. We interpret this to be evidence that there is likely to be significant selection of peers by BMI. Despite the importance of selection as an explanation for the documented correlations between own and peer BMI, we are unable to adequately account for

[13] The impact of correctly defining peer groups on these estimates is discussed in a separate working paper, Halliday and Kwak (2008).

omitted correlated effects and the endogeneity problem. Thus, we cannot definitively rule out positive causal peer effects.

Given the inability to satisfactorily solve the omitted correlated effects and the endogeneity problem, the results suggest one of two possibilities: first, overweight adolescents sort themselves into networks with other overweight friends or second, overweight adolescents influence their friends to also become overweight. The significant correlations between own and peer height indicate the first possibility is significant. Our study documents these correlations in a nationally representative sample of adolescents. However, neither our study nor the three that precede ours can differentiate between these two explanations.

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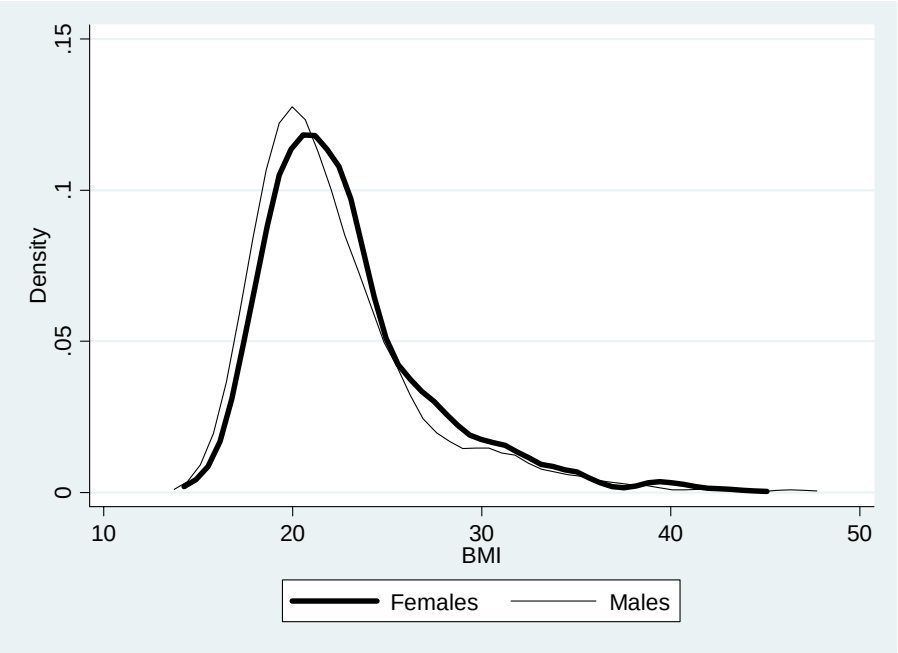
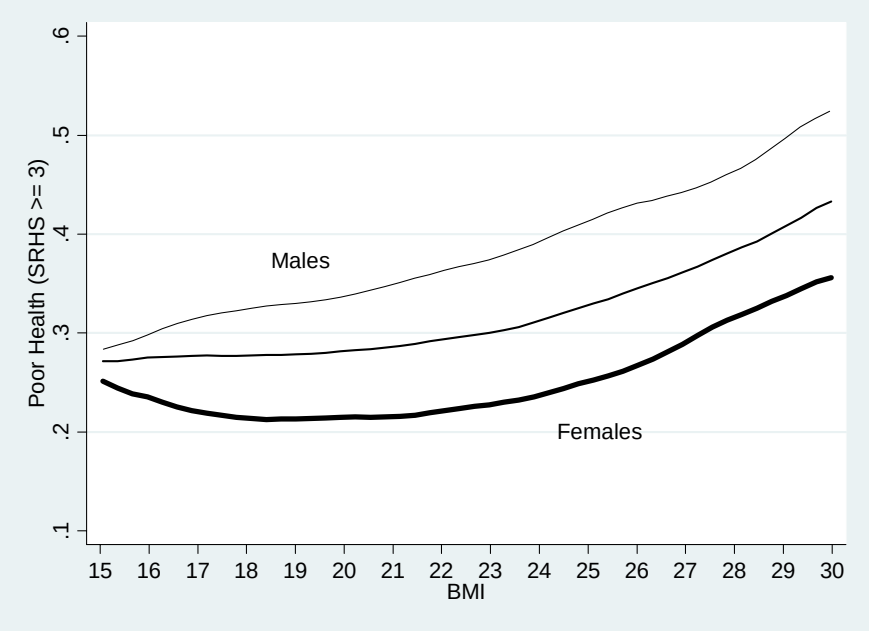
Figure 1. Kernel Density of BMI, Adolescents in Grades 7-12



Source: National Longitudinal Adolescent Health Survey (Add Health), Waves I and II

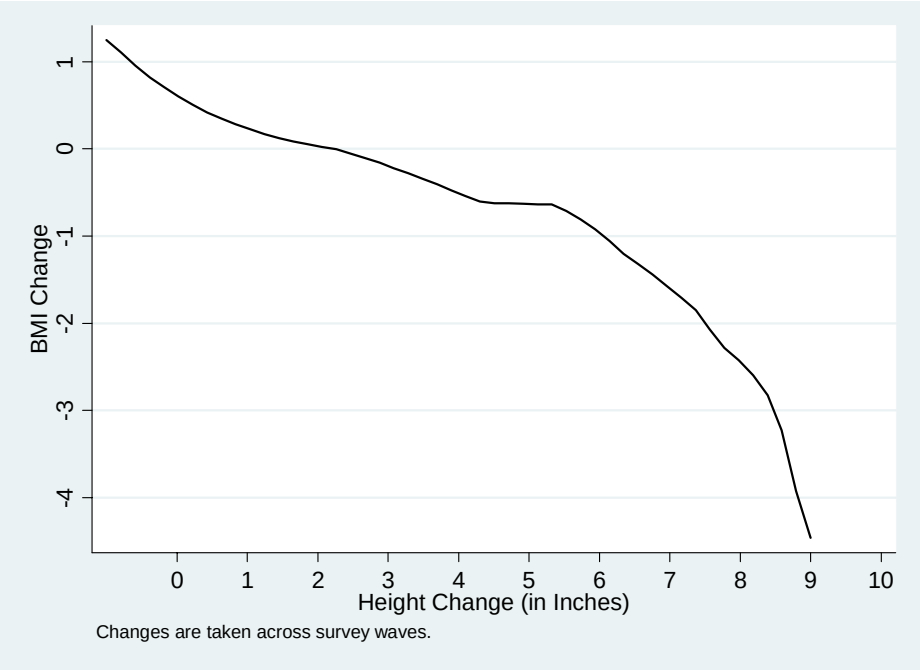
Figure 2. Kernel Regression of Self-Reported Health Status (SRHS) on BMI, Adolescents in Grades 7-12



Source: National Longitudinal Adolescent Health Survey (Add Health), Waves I and II

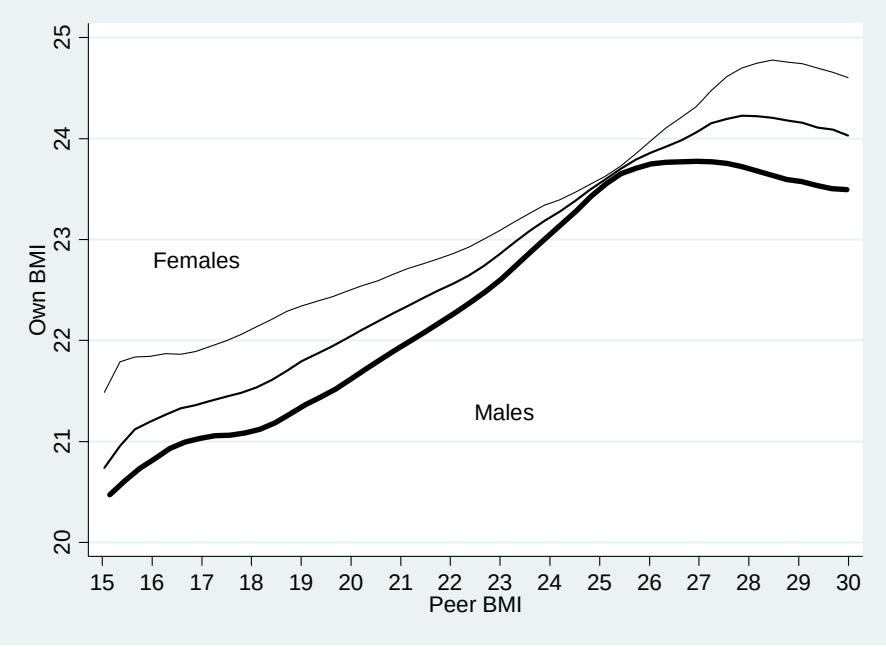
Note: SRHS is a categorical variable corresponding to the pupil's assessment of their own health (1=excellent; 2 = very good; 3 = good; 4 = fair; 5 = poor).

Figure 3. Kernel Regression of Changes in BMI on Changes in Height, Adolescents in Grades 7-12



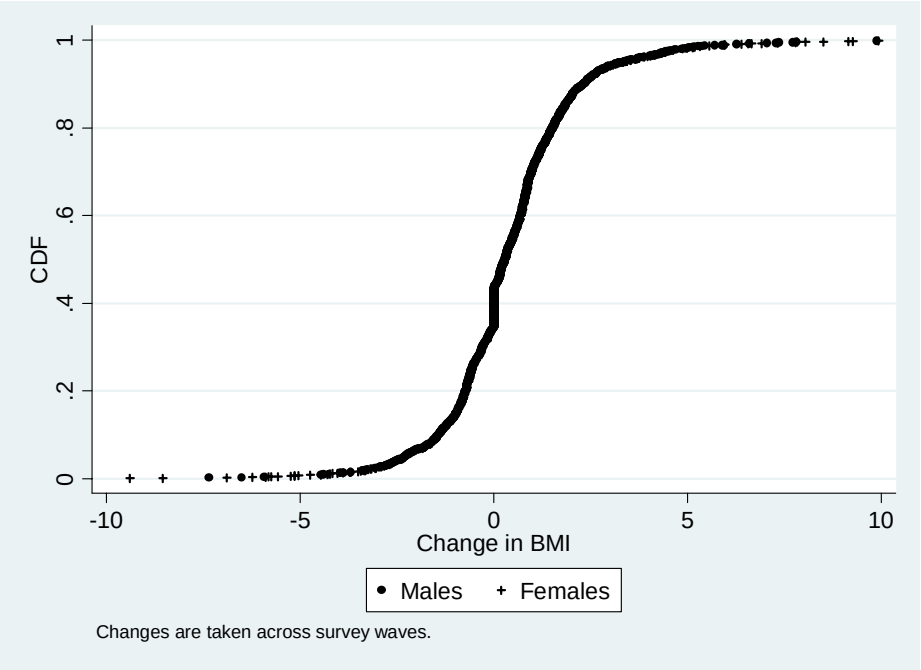
Source: National Longitudinal Adolescent Health Survey (Add Health), Waves I and II (1994-95, 1996)

Figure 4. Kernel Regression of Own BMI on Average Peer Group BMI, Adolescents in Grades 7-12



Source: National Longitudinal Adolescent Health Survey (Add Health), Waves I and II (1994-95, 1996)

Figure 5. CDF of Changes in BMI, Adolescents in Grades 7-12



Source: National Longitudinal Adolescent Health Survey (Add Health), Waves I and II (1994-95, 1996)

Table 1. Baseline Characteristics and Sample Sizes

Variable	Our Sub-Sample	Full Sample
Age	15.11	14.99
	(1.63)	(1.72)
Self-Reported Health	2.11	2.09
Status[1]	(0.95)	(0.94)
Male[2]	0.48	0.50
	(.50)	(0.50)
Grade	9.80	9.58
	(1.58)	(1.62)
White[2]	0.55	0.61
	(.50)	(0.49)
Black[2]	0.19	0.19
	(.39)	(0.39)
Asian[2]	0.12	0.07
	(0.33)	(0.26)
Self-Reported Health	2.11	2.09
Status (SRHS)[1]	(0.95)	(0.94)
Mother Has a College	0.34	0.32
Degree	(0.47)	(0.47)
Father Has a College	0.38	0.38
Degree	(0.49)	(0.49)
In-Home Wave 1 Sample Size	4617	
In-Home Wave 2 Sample Size	2970	

Standard deviations are reported in parentheses. Descriptive statistics are calculated at baseline i.e. for wave 1.

[1] SRHS is a categorical variable corresponding to the pupil's assessment of their own health (1=excellent; 2 = very good; 3 = good; 4 = fair; 5 = poor).

[2]Dummy variable equals one if the characteristic is true and zero otherwise.

Table 2. Summary Statistics for BMI

All	Male	Female
	Wave 1	
22.59	22.91	22.30
(4.48)	(4.50)	(4.46)
	Wave 2	
22.95	23.37	22.55
(4.80)	(4.79)	(4.78)

Standard deviations are reported in parentheses.

Table 3. Self-Reported Health Status, BMI, and Socioeconomic Status

	Dependent Variable: Self-Reported Health Status[1]			
	(1)	(2)	(3)	(4)
BMI	0.02	0.02	0.02	0.02
	[10.79]	[10.60]	[10.92]	[8.23]
Male	-	-0.14	-0.14	-0.14
		[10.50]	[-10.61]	[-8.31]
White	-	-	-0.07	-0.05
			[-3.30]	[-1.43]
Black	-	-	-0.09	-0.07
			[-4.28]	[-2.12]
Asian	-	-	0.00	0.05
			[0.23]	[1.77]
Mother's Education[2]	-	-	-	-0.03
				[-0.89]
Father's Education[2]	-	-	-	-0.09
				[-5.15]
R2	0.02	0.04	0.04	0.05
N	4426	4408	4408	2686

Note: This table reports marginal effects of a probit model in which the dependent variable is a binary indicator for SRHS. Robust t-statistics are in brackets. All standard errors adjust for clustering by school. All regressions include a complete set of grade dummies.

[1] SRHS is a categorical variable corresponding to the pupil's assessment of their own health (1=excellent; 2 = very good; 3 = good; 4 = fair; 5 = poor).

[2]These variables are binary variables indicating that the mother (father) has a college degree.

Table 4. Correlations Between Own BMI and Peer BMI

	Own BMI		Own BMI > 85%		Own BMI < 10%	
	(1) School Fixed Effects	(2) Individual Fixed Effects	(3) School Fixed Effects	(4) Individual Fixed Effects	(5) School Fixed Effects	(6) Individual Fixed Effects
All						
Average	0.19	-0.00	0.11	-0.02	-0.01	0.01
Peer BMI	[4.64]	[-0.23]	[4.85]	[-0.86]	[-1.25]	[0.81]
R2	0.14	0.03	0.06	0.00	0.03	0.00
NT	7133	7386	7133	7386	7133	7386
Males						
Average	0.18	-	0.08	-	-0.01	-
Peer BMI	[3.62]		[2.97]		[-0.58]	
R2	0.15	-	0.08	-	0.06	-
NT	3410	-	3410	-	3410	-
Females						
Average	0.19	-	0.13	-	-0.02	-
Peer BMI	[4.18]		[3.59]		[-1.63]	
R2	0.16	-	0.11	-	0.05	-
NT	3723	-	3723	-	3723	-

Robust t-statistics are reported in brackets. All regressions include dummy variables for grade. The regressions with school fixed effects also include controls for gender, race and health status (broken into five dummy variables).

Note: 'Average Peer BMI' is the average BMI of all peers as designated by the respondent.

Table 5. Determinants of Changes in Own BMI

	Change in Own BMI					
	(1)	(2)	(3)	(4)	(5)	(6)
	All		Males		Females	
Average Peer BMI	0.04 [1.73]	0.05 [2.00]	0.02 [0.81]	0.03 [0.89]	0.06 [2.05]	0.07 [2.36]
Change in Own Height	-0.50 [-9.23]		-0.43 [-5.95]		-0.61 [-9.97]	
Own BMI	-0.03 [-2.53]	-0.03 [-2.42]	-0.02 [-1.39]	-0.02 [-1.10]	-0.04 [-1.97]	-0.04 [-2.48]
R2	0.19	0.10	0.21	0.13	0.26	0.16
N	1888	1888	903	903	985	985

All regressions include race, school, gender and age dummies as well as health status (broken into five dummy variables) and parental living arrangements. All standard errors are clustered by school. Robust t-statistics are in brackets.
Note: 'Average Peer BMI' is the average BMI of all peers as designated by the respondent.

Table 6. Correlations Between Own Height and Peer Height

	All		Males		Females	
	(1) School Fixed Effects	(2) Individual Fixed Effects	(3) School Fixed Effects	(4) Individual Fixed Effects	(5) School Fixed Effects	(6) Individual Fixed Effects
Average Peer Height	0.48 [28.40]	0.05 [4.29]	0.46 [20.75]	0.08 [4.89]	0.43 [17.42]	0.02 [1.07]
R2	0.53	0.11	0.42	0.27	0.24	0.02
NT	7133	7349	3410	3555	3723	3794

Robust t-statistics are reported in brackets. All regressions include dummy variables for grade. The regressions with school fixed effects also include controls for gender, race and health status (broken into five dummy variables).
Note: 'Average Peer Height' is the average height in inches of all peers as designated by the respondent.

Table 7. The Impact of the Distribution of BMI in Peer Networks on Own BMI

	Dependent Variable: Own BMI			
	(1)	(2)	(3)	(4)
% of Peer Group >= 90th Percentile	1.25 [4.27]	-	-	-
% of Peer Group >= 85th Percentile	-	1.11 [4.63]	-	-
% of Peer Group >= 75th Percentile	-	-	1.03 [5.12]	-
% of Peer Group <= 10th Percentile	-	-	-	-0.00 [-0.02]
R2	0.12	0.12	0.12	0.12
NT	7133	7133	7133	7133

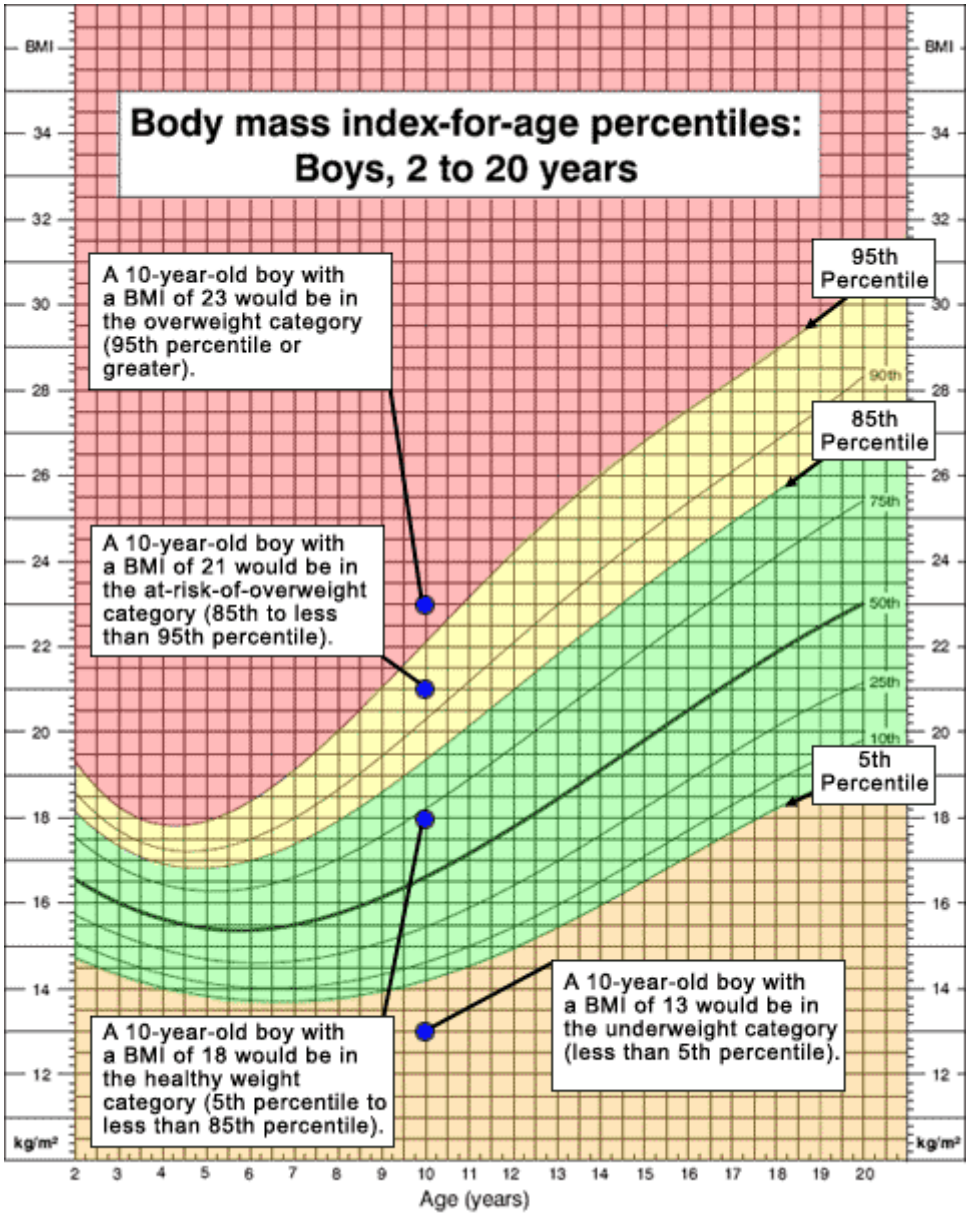
This table reports OLS results of regressions of a dependent variable on the percentages of the individual's peer network who are above or below various thresholds. Robust t-statistics are in brackets. All standard errors adjust for clustering by school. All regressions include controls for gender, race, health status (broken into five dummy variables) as well as a complete set of grade and school dummies.

Table 8. The Impact of the Distribution of BMI in Peer Networks on Own BMI > 85%

	Dependent Variable: Own BMI > 85 %			
	(1)	(2)	(3)	(4)
% of Peer Group >= 90th Percentile	0.11 [4.21]	-	-	-
% of Peer Group >= 85th Percentile	-	0.11 [4.85]	-	-
% of Peer Group >= 75th Percentile	-	-	0.10 [5.20]	-
% of Peer Group <= 10th Percentile	-	-	-	-0.02 [-0.93]
R2	0.08	0.08	0.08	0.07
N	7133	7133	7133	7133

This table reports OLS results of regressions of a dependent variable on the percentages of the individual's peer network who are above or below various thresholds. Robust t-statistics are in brackets. All standard errors adjust for clustering by school. All regressions include controls for gender, race, health status (broken into five dummy variables) as well as a complete set of grade and school dummies.

Appendix. An Example of BMI Distributions for Children and Adolescents



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http://www.cdc.gov/nccdphp/dnpa/bmi/childrens_BMI/about_childrens_BMI.htm