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Exposure to Obesity and Weight Gain Among Adolescents[1]

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Abstract

In a treatment-effect framework using Add Health data, we investigate whether adolescents gain weight when increasingly exposed to obesity in their social networks. We find that weight gain can be a reaction to an increase, but not a decrease, in exposure to obesity that is based on social ties, not geographic proximity. Taking an endogenous growth perspective on the prevalence of obesity, we thus attempt to reveal a mechanism through which obesity may potentially develop into a sweeping epidemic. Our results also suggest an uphill battle against the obesity epidemic, and we recommend that its prevention be a high priority.

1 Introduction

The obesity rate in the United States has been rising dramatically. Over three decades, the number of obese adults has grown by nearly 50% (Chou, Grossman, and Saffer, 2004). The percentage of overweight children (between the ages of 6 and 11) has been escalating for four decades (Liu, Hsiao, Matsumoto, and Chou, 2009; Trogdon, Nonnemaker, and Pais, 2008), and has more than tripled since 1980 (Segal and Gadola, 2008). There also has been a dramatic increase in the percentage of obese adolescents (between the ages of 12 and 19) from less than 5% in the 1960s to above 15% today (Halliday and Kwak, 2009).[1] The obesity rate is also rising in other developed countries (Bleich, Cutler, Murray, and Adams, 2007) and in developing countries (Philipson and Posner, 2008). Worldwide, there are nearly 1.1 billion overweight people, and childhood obesity is still on the rise (Rosin, 2008). The prevalence of obesity has become a global phenomenon (Cutler, Glaeser, and Shapiro, 2003) and has been labeled an epidemic (Goel, 2006).

The consequences of obesity are far-reaching. Obesity may shorten life expectancy and can cause many ailments, such as diabetes and heart disease.[2] The negative impacts of obesity or overweight on an individual's labor market outcome have been examined by many studies (Averett and Korenman, 1996; Baum II and Ford, 2004; Biddle and Hamermesh, 1998; Cawley, 2004; Hamermesh and Biddle, 1994; Morris, 2006). After smoking, obesity has become the second leading cause of preventable deaths (Goel, 2006). As the obesity rate increases in the United States, so do health care expenditures (Michaud, Goldman, and et al, 2009), already ranging in the billions of dollars (Halliday and Kwak, 2009).[3] Because of escalating heath care costs imposed on the entire society, obesity has gone beyond a personal matter. Public interventions aimed at controlling or reducing the obesity rate, such as taxes

[1] According to the Centers for Disease Control and Prevention (CDC), by 2008, the obesity rate was below 20% only in Colorado. In 32 states, the obesity rate had already reached or exceeded 25%. They include 6 states (Alabama, Mississippi, Oklahoma, South Carolina, Tennessee, and West Virginia), whose obesity rate was above 30%. More detail about the prevalence of obesity and overweight is provided at the CDC's websites: http://www.cdc.gov/obesity/index.html and http://www.cdc.gov/obesity/data/trends.html.

[2] More detail is provided at CDC: http://www.cdc.gov/obesity/causes/health.html.

[3] More detail is provided at CDC: http://www.cdc.gov/obesity/causes/economics.html.

on junk-foods (Schroeter, Lusk, and Tyner, 2008; Yaniv, Rosin, and Tobol, 2009) or state level physical education requirements for students (Cawley, Meyerhoefer, and Newhouse, 2007), may be justified (Philipson and Posner, 2008). Such interventions can be imperative for children and adolescents, because the long-term effects on health of childhood obesity are worse than the effects of adult obesity (Olshansky, Passaro, and et al, 2005).

The dramatic increase in the obesity rate in recent decades and its far-reaching consequences have driven many researchers to search for explanations, hoping to find cures or preventions. Several studies take a social perspective because people's health-related behaviors, such as smoking, are interdependent (Harris and López-Valcárcel, 2008), as are other risky behaviors (Clark and Lohéac, 2007; Lundborg, 2006). On the one hand, if people care about their own weight relative to that of others, then "imitative obesity" may exist (Blanchflower, Van Landeghem, and Oswald, 2009). There may be an increase in people's own ideal weight in response to an increased (average) weight of their reference group (Burke and Heiland, 2007). On the other hand, if people have more social contacts who are obese, then their tolerance for obesity may increase; this could make it possible for obesity to "spread" within social networks (Christakis and Fowler, 2007). Thus, as Philipson and Posner (2008) point out—"The social aspects of obesity may have a multiplier effect on the growth of obesity. When obesity is relatively rare, it is considered abnormal and repulsive, and this negative response helps to keep it in check. As obesity begins to rise, the negative image of obesity becomes less intense because obesity is now more common (see also Amnon Levy 2002)."

Even though this social aspect of obesity does not pinpoint the origin of obesity, it does reveal a potential mechanism through which obesity may "feed back" (or "propagate") itself. Because social interactions can be common among adolescents, several studies have begun to investigate the existence of peer effects among adolescents in terms of obesity or overweight status (Cohen-Cole and Fletcher, 2008; Fowler and Christakis, 2008; Halliday and Kwak, 2009; Renna, Grafova, and Thakur, 2008; Trogdon, Nonnemaker, and Pais, 2008).[4]

[4]Conceptual analysis of peer effects, also known as social or non-market interactions, or endogenous effects, has been discussed by Manski (2000). Identification of peer effects is hindered by a series of problems,

If social interactions generate a multiplier effect (Glaeser, Sacerdote, and Scheinkman, 2003) for obesity, then policy interventions targeting social groups may be effective in battling the obesity epidemic: a policy intended to prevent or treat overweight or obesity will have effects not only on the targeted individuals but also on their peers in the same social group.[5]

Our study is aimed at providing empirical evidence on Philipson and Posner (2008)'s statement about the social aspect of obesity. Using data from the National Longitudinal Study of Adolescent Health (Add Health), we measure adolescents' exposure to obesity by the number of their obese friends in each of the first two waves of Add Health. We focus on the effect of an increase in adolescents' exposure to obesity within their social networks on their weight gain. We term this "the feedback effect" because it is the occurrence of some people in a population becoming obese that leads to other people's weight gain (not necessarily to becoming overweight or obese) in the population. The existence of the feedback effect may enable obesity to "proliferate," and the magnitude of the feedback effect may ring alarm bells warning of an obesity "catalyst." Our study aims to offer an explanation for the endogenous growth of obesity, while most studies seek to pinpoint a list of exogenous factors that contribute to the growth of obesity. Specifically, our study finds first that an increase in adolescents' exposure to obesity within their social networks does cause weight gain, suggesting a potential accelerator in the spread of obesity. Furthermore, the impact on weight gain from increased exposure to obesity is found largely among those who are neither obese nor overweight in the beginning. Obesity seems to most effectively "infect" those who are not obese or overweight yet.

such as peer group self-selection, group-level unobserved heterogeneities, the simultaneity problem, and the reflection problem (Manski, 1993). Several econometric models have been proposed with various identification assumptions, such as linear-in-expectations models (Manski, 1993; Graham and Hahn, 2005), linear-in-means models (Lee, 2007), discrete choice models with social interactions (Brock and Durlauf, 2001, 2002, and 2007; Durlauf, 2001), multiple-group models (Cohen-Cole, 2006), and excessive variances contrast models (Graham, 2008). Many empirical studies conclude that large endogenous effects are probably biased upward due to the lack of controlling for self-selection and unobserved individual-level heterogeneities (Evans, Oates, and Schwab, 1992). With randomization, impacts of unobservables and assortive behaviors could be controlled across groups (Zimmerman, 2003). Other identification strategies, for example, are based on sibling fixed effects (Aaronson, 1998) or individual fixed effects (Mas and Moretti, 2009).

[5] Several studies have examined peer effects among adolescents' risky behaviors, such as Alexander, Piazza, Mekos, and Valente (2001), Bauman, Carver, and Gleiter (2001), and Haynie (2002).

Second, we do not find any impact of an opposite "treatment": that is, decreased exposure to obesity within individuals' social networks does not have a statistically significant impact on their weight changes, regardless of their initial body weight status. Therefore, any policy aimed at reversing the spread of obesity would not likely take advantage of the feedback effect from obesity within a social network to weight loss.

Third, we find that it is the perceivable increase in the exposure to obesity within individuals' social networks that causes their weight gain. The more visible the change in peers' non-obesity-to-obesity status, the larger the feedback effect of obesity on weight gain becomes.

Finally, we confirm that it is through social ties, not geographic proximity, that the increased exposure to obesity causes weight gain. We measure Individuals' geographic exposure to obesity is by the total number of obese classmates in their grade and school. Here, we do not find any statistically significant impact on individuals' weight changes, either from an increase or a decrease in the number of same-grade and same-school obese classmates.

Our findings are closely related to the peer effect literature. However, our focus and measurement of peer influence are different. The peer effect literature on body weight has concentrated on gauging the impact on individuals' body weight of their peers' average body weight. Here, in a treatment-effect framework, we ask whether a change (a "treatment") in the number of obese people who are socially close to an individual can cause the individual to gain weight. We find that it is not the average body weight of the socially close people but their becoming obese (a qualitative change in their body weight status) that causes weight gain.

We contribute to the peer effect literature in three ways: first, a qualitative (and perceivable) change from non-obesity to obesity, as opposed to a quantitative (and marginal) change in peers' average body weight, can cause weight gain. Second, the obesity feedback effect is asymmetric: one is likely to gain weight with an increased exposure to obesity, but one is unlikely to lose weight with a decreased exposure to obesity. Third, in evaluating the

peer effect on an individual's body weight, it is important to take into account the mean reversion of the body weight, that is the tendency of individuals' weight to revert to a mean level over time. Our findings reveal an uphill battle in the war against obesity. An increase in the exposure to obesity can cause weight gain within a social network, particularly for those who are not already overweight or obese. However, a decrease in the exposure to obesity does not seem to lead to weight loss within the social network. Thus, policies focused on fighting obesity should make prevention a high priority.

While our study examines the feedback effect from obesity itself (through which obesity has the potential to spread along social ties), a number of studies have focused instead on a list of exogenous factors that directly contribute to obesity. Because the obesity epidemic grows so rapidly, and has emerged recently, it is unlikely that genetic or biological factors—which are relatively stable over time—can solely explain the sharp change occurring in such a short time (Philipson, 2001; Rosin, 2008). Characterizing body weight as an individual's independent choice, many studies investigate changes in certain factors that can affect that choice during the development of the obesity epidemic. For example, if body weight is directly related to calories, then overweight or obesity may be the result of imbalanced intake and expenditure of calories (Bleich, Cutler, Murray, and Adams, 2007; Cutler, Glaeser, and Shapiro, 2003). Factors that can induce excessive calorie intake include the low price of food rich in calories and fast-food restaurants (Chou, Grossman, and Saffer, 2004), as well as low income (Philipson and Posner, 2008; Wilde, McNamara, and Ranney, 1999). Meanwhile, certain factors reduce caloric expenditure, including sedentary work and life style (Lakdawalla and Philipson, 2007), mostly as a result of welfare-improving technological advancement (Lakdawalla, Philipson, and Bhattacharya, 2005). These studies all assume that individuals have sufficient knowledge about the benefits and costs of caloric intake and expenditure when they choose their body weight. Other studies have examined the relationship between individuals' knowledge about the health risk of obesity and their tendency to become obese (Kan and Tsai, 2004), as well as the influence of fast-food restaurant

advertising on childhood obesity (Chou, Rashad, and Grossman, 2008). Some studies have investigated relationships between obesity and smoking (Chou, Grossman, and Saffer, 2004; Chou, Grossman, and Saffer, 2006; Gruber and Frakes, 2006), or between weight control and smoking initiation among adolescents in particular (Cawley, Markowitz, and Tauras, 2004), as well as the impacts of maternal working hours on childhood obesity (Anderson, Butcher, and Levine, 2003; Liu, Hsiao, Matsumoto, and Chou, 2009).

Another group of studies attributes obesity to a self-control problem. People having present-biased preferences or an increased time-discounting rate will eat more than enough for today and then regret overeating tomorrow (Borghans and Golsteyn, 2006; Cutler, Glaeser, and Shapiro, 2003; Dodd, 2008; Smith, Bogin, and Bishai, 2005). If the issue of commitment or (rational) addiction is entrenched in human nature (Becker and Murphy, 1988; Schelling, 1978), then this behavioral perspective seems inadequate in explaining why obesity, if due to a weakness in human nature, has only become an epidemic in the past few decades.

The rest of this paper is organized as follows. Section 2 describes the body weight measure and the data we use for our study. Section 3 lays out a conceptual framework, detailing identification assumptions and econometric specifications. Section 4 presents empirical findings, including a series of coherency checks. Section 5 concludes.

2 Body Weight Measure and Data

We use Body Mass Index (BMI) as a body weight measure.[6] This height-adjusted body weight measure has been widely used as a proxy for total body fat, although it does not measure body fat directly.[7] For children and adolescents between the ages of 2 and 20, body weight status is classified based on the 95th, 85th, and the 5th percentile of the BMI distribution shown in the Growth Charts of the CDC. The CDC's classification of body

[6] BMI = weight (in kilograms)/height (in meters)^2 = 703$\cdot$ weight (in pounds)/height (in inches)^2.

[7] BMI is an easy-to-calculate and consistent measure, but it has several drawbacks (Goel, 2006; Rosin, 2008). For example, it ignores body mass composition. A more muscular person can be misclassified as being obese solely based on BMI. BMI also ignores the differences in abdominal fat among people who with similar BMI.

weight status is: 1) obesity = BMI $\geqslant$ 95th percentile; 2) overweight = 85th percentile $\leqslant$ BMI $<$ 95th percentile; 3) optimal weight = 5th percentile $\leqslant$ BMI $<$ 85th percentile; and 4) underweight = BMI $<$ 5th percentile. Because children and adolescents' body weight can change dramatically during their growth spurt period, which also differs by gender, those percentiles are specific to age and gender and are defined for each age-gender group.[8] In Table 1, we list all of the 95th, 85th, and 5th percentiles of the BMI distribution for children between the ages of 2 and 20, and for both boys and girls.[9] We use these cutoffs for our study population to classify their body weight status.[10]

2.1 Add Health

We use data from Add Health, a school-based longitudinal study of a nationally representative sample of adolescents in grades 7 to 12 in the United States during the 1994-5 school year.[11] This Add Health cohort has been followed into young adulthood with four in-home interviews after 1994-5. We use both Wave I and Wave II from Add Health: Wave I, done in 1994-5, surveyed 90,118 students from 80 high schools and 52 middle schools; a subset of 20,745 Wave I students then was chosen for an in-depth in-home survey. Wave II, done in 1996, sampled 14,738 students from the original group of 20,745 Wave I students.[12] In our sample we include those students who have full information on their own height and weight and the height and weight of their nominated best friends—one male and one female.[13] So,

[8] For details, see http://www.cdc.gov/healthyweight/assessing/bmi/childrens_bmi/about_childrens_bmi.html.

[9] For details, see http://www.cdc.gov/growthcharts/html_charts/bmiagerev.htm.

[10] For those who are at least 21 years old, the CDC's classification of body weight status based on BMI is deterministic regardless of gender: 1) obesity = BMI $\geqslant$ 30; 2) overweight = 25 $\leqslant$ BMI $<$ 30; 3) optimal weight = 18.5 $\leqslant$ BMI $<$ 25; and 4) underweight = BMI $<$ 18.5. In our study population, we use these fixed cutoffs (18.5, 25, and 30) for those who are at least 21 years old.

[11] Detailed and comprehensive information is provided at http://www.cpc.unc.edu/projects/addhealth.

[12] Quoted from "Design Facts at a Glance" (http://www.cpc.unc.edu/projects/addhealth/design/designfacts): "The Wave II in-home interview sample is the same as the Wave I in-home interview sample, with a few exceptions: (1) the majority of 12th-grade respondents were removed from the Wave II sample, as they exceeded the grade eligibility requirement (12th graders who were part of a genetic pair were retained); (2) the Wave I disabled sample was not reinterviewed at Wave II; (3) the Wave II sample contains a small number of adolescents who did not participate in the first wave; and (4) no parent interview was conducted at Wave II."

[13] The best friend nomination can include friends in the same school as well as friends from outside the school for whom the information is not available in the Add Health original data.

the number of best friends nominated by each focal student can be 0, 1, or 2. Because a nominee (the nominator's same-school best friend) may not receive Add Health's in-home interview (either in Wave I or in Wave II), there is a substantial reduction in the sample size for estimation. Ultimately, our sample includes 1,184 students in both Wave I and Wave II. Table 2 reports the summary statistics for the students in both waves. Those summary statistics, such as average BMI, have been checked and found to be very similar to other studies regarding BMI also using the Add Health data, such as Cohen-Cole and Fletcher (2008), Halliday and Kwak (2009), and Trogdon, Nonnemaker, and Pais (2008). For each of the first two waves of the Add Health, we measure the exposure to obesity by the number of best friends who are nominated by the adolescent and who are classified as being obese according to the CDC's criteria.

Unlike the three aforementioned studies using Add Health, our study only considers a focal student's nominated best friends (up to one male and one female). In the Add Health surveys, some students were asked to nominate up to five male and five female friends in order of closeness (in the in-school survey), while other students were asked to only nominate up to one best male and one best female friend (in the in-home survey). To examine how the change in the number of obese friends affects the focal student's weight, we need to fix the value range of this variable—which should be the same across students—to be between -2 and 2. For the causal (treatment) effect of changes in the exposure to obesity on weight gain, our treatment group consists of adolescents who experience an increase in exposure to obesity between the two waves. The control group includes those who experience no change (neither an increase nor a decrease) in exposure to obesity between the two waves.

Adolescents are arguably influenced easily by their peers. The Add Health is thus well suited to investigating the feedback effect of obesity on weight gain within an adolescent's social network.[14] The Add Health survey's longitudinal design makes it possible to deal

[14]In addition to the high percentage of overweight adolescents turning into overweight adults, some simulation study shows that overweight adolescents incur greater health care expenditure than adolescents of normal weight (Monheit, Vistnes, and Rogowski, 2009).

with individual-level unobservables. Moreover, commonly shared factors are more likely to exist among best friends instead of ordinary friends. Because there are only one or two years between the first two waves, such commonly shared factors (probably unobservable to a researcher) among best friends are likely to be time invariant. Thus, the effects of those time-invariant unobservables can be removed through differencing over time. Although the first two waves of Add Health contain only self-reported measures for weight and height, some researchers have found that the correlation coefficient between self-reported and actually measured BMI is high ($r = 0.92$). This comparison was made when measured BMI became available in Wave III (Trogdon, Nonnemaker, and Pais, 2008).

Furthermore, the Add Health survey has information on peers who are socially close to an adolescent (such as a best friends) as well as on peers who are geographically close (such as classmates in the same school and same grade). This feature enables us to determine whether social closeness or geographic proximity is more important in transmitting the obesity feedback effect.

2.2 Descriptive Statistics

Table 2 presents summary statistics for both waves.[15] The average BMI is 22.21 in Wave I, and increases slightly to 22.67 in Wave II. Although our sample is much smaller (because we have not only information about individuals' BMI and their characteristics, but also their best friends'), both statistics are quite similar to those from other studies of adolescents' BMI using the Add Health data. For individual body weight status in Wave I, nearly 25% of the adolescents can be classified as being either overweight or obese according to the CDC cutoffs. This proportion remains about the same in Wave II, which is reasonable because there is only a year or two between the two waves. In Wave I, 32.6% of the adolescents wanted to lose weight, and 37.3% of the adolescents wanted to simply maintain their current weight. This pattern remains almost the same in Wave II: about 31.5% of the adolescents wanted

[15] The set of variables in our study is similar to Trogdon, Nonnemaker, and Pais (2008).

to lose weight, and 34.9% of the adolescents preferred their current weight. Table 2 also reports the averages of each student's best friends' BMI, body weight status, and body image assessment, which show similar patterns. If a focal student's peers' BMI (or body weight status) directly affects his or her own BMI (or body weight status), then excessive variations (due to peers' influence) will appear in the focal students' BMI (or body weight status) distribution (Graham, 2008). Therefore, the variations will be reflected in the standard deviations (or the variances) in the Wave II variables for self BMI and body weight status. However, the standard deviations of a focal student's BMI and body weight status shown in Table 2 remain very similar between the two waves, suggesting a lack of direct evidence about the effect of changes in peers' BMI (or body weight status) on the focal students' BMI (or body weight status). From a different angle, and following Philipson and Posner's (2008) statement on the social aspects of obesity, we focus on the impact on a focal student's weight of a change in his or her exposure to obesity. The existence of such an effect suggests that when obesity becomes more prevalent, it has the potential to soften its own negative image within a social network. Considering three potential states of weight gain caused by an increase, a decrease, or no change in one's exposure to obesity, we conduct the following empirical analysis using a treatment effect evaluation framework.

3 Conceptual Framework

We focus here on the causal effect of an increase in an individual's exposure to obesity (a treatment) on that individual's weight gain. We use BMI as a measure of height-adjusted body weight. We define the treatment effect as the difference in the individual's BMI between two potential states: the treated and the untreated (control) states. In each of the Add Health's two waves, we measure exposure to obesity as the number of adolescents nominated as the individual's best friends who are obese, based on the CDC's cutoffs. The treatment group experiences an increase in the number of obese friends, while the control (untreated)

group experiences no change in the number of obese friends. The increased number of obese friends may occur either with the same friends who became obese between the two waves, or with different friends being nominated in each wave. It would be interesting to decompose the total effect of an increase in the exposure to obesity into two components: one due to same friends, and the other due to different friends. However, because of limited sample size, we cannot conduct that decomposition analysis. For the same reason, we cannot decompose the treatment effect by gender, either. Instead, we focus on the composite effect of an increased exposure to obesity. We use a difference-in-differences (DID) approach to identify the treatment effect.

A linear model of two potential outcomes is:

$$y_{jit} = \beta_0 + \beta_1 G_i + \beta_2 T_i + \beta_d j + \mathbf{x}'_{it}\beta_x + \alpha_i + u_{jit}, \tag{1}$$

where y_{jit} denotes an individual i's potential BMI in state j ($j = 0, 1$) in period t ($t = 1, 2$ for Wave I and II, respectively). Here j, the state indicator, equals 1 for the treatment state and 0 for the control state. We allow the individual i's unobserved heterogeneities u_{jit} to vary over time (t) and to differ by state (j), and to be normalized with zero mean ($\mathbb{E}(u_{jit}) = 0$). Some unobservables (to a researcher), denoted by α_i, including genetic factors, are assumed to be time-invariant within the short period between the two waves. Here G_i, the group indicator, equals 1 for the treatment group and 0 for the control group. T_i, the time indicator, equals 0 for Wave I (the first period, $t = 1$) and 1 for Wave II (the second period, $t = 2$). Other time-varying factors are included in $\mathbf{x}_{it}$, and are assumed to remain unchanged in the two states. The causal effect of an increase in the exposure to obesity on BMI averaged across all individuals is denoted by β_d, which indicates the average treatment effect (ATE $\equiv \mathbb{E}(y_{1it} - y_{0it}) = \beta_d$).

3.1 Identification Assumptions

Given that we have observations for each individual i in both waves, we can difference out the unobservables α_i in equation (1). The observed change in BMI (Δy_i) is a linear combination of the two potential outcomes (based on G_i):[16]

$$\begin{aligned} \Delta y_{0i} &= \beta_2 + \Delta \mathbf{x}_i' \beta_x + u_{0i2} - u_{0i1}, && (2) \\ \Delta y_{1i} &= \beta_2 + \beta_d + \Delta \mathbf{x}_i' \beta_x + u_{1i2} - u_{0i1}, && (3) \\ \Delta y_i &= \Delta y_{0i} + G_i(\Delta y_{1i} - \Delta y_{0i}). && (4) \end{aligned}$$

To simplify notations, we hereafter omit the subscript i. DID gives:

$$\begin{aligned} \text{DID} &= \mathbb{E}(\Delta y | \Delta \mathbf{x}, G = 1) - \mathbb{E}(\Delta y | \Delta \mathbf{x}, G = 0) && (5) \\ &= \beta_d + \mathbb{E}(u_{12} - u_{01} | \Delta \mathbf{x}, G = 1) - \mathbb{E}(u_{02} - u_{01} | \Delta \mathbf{x}, G = 0), \end{aligned}$$

which does not identify the causal effect of the intervention (j) without assumptions about the unobservables (u_{jt}). In the absence of treatment, if the unobservables on average can be assumed to have the same change over time (that is, a "common trend") in both the treatment and the control groups, conditional on the changes in the observables,

$$\mathbb{E}(u_{02} - u_{01} | \Delta \mathbf{x}, G = 1) = \mathbb{E}(u_{02} - u_{01} | \Delta \mathbf{x}, G = 0), \quad (6)$$

then DID identifies the average effect of treatment on the treated (ATT):

$$\text{DID} = \beta_d + \mathbb{E}(u_{12} - u_{02} | \Delta \mathbf{x}, G = 1) = \mathbb{E}(y_1 - y_0 | \mathbf{x}, GT = 1) \equiv \text{ATT}(\mathbf{x}). \quad (7)$$

[16] Because there is no group membership switched between the two waves, we have $\Delta G_i = 0$. By definition, $\Delta T_i = 1$.

A nonzero $\mathbb{E}(u_{12} - u_{02}|\Delta\mathbf{x}, G = 1)$ reveals the treatment effect heterogeneity attributable to the unobservables in the treatment group, which distinguishes ATT from ATE (β_d). Based on equation (4), we have the following linear regression model:

$$\Delta y = \beta_2 + \Delta\mathbf{x}'\beta_x + \beta_{\text{ATT}}G + (u_{02} - u_{01}) + [(u_{12} - u_{02}) - \mathbb{E}(u_{12} - u_{02}|G = 1, \Delta\mathbf{x})]G, \quad (8)$$

where $\beta_{\text{ATT}} = \beta_d + \mathbb{E}(u_{12} - u_{02}|\Delta\mathbf{x}, G = 1)$. The identification of β_{ATT} depends on:

$$Cov\{G, (u_{02} - u_{01}) + [(u_{12} - u_{02}) - \mathbb{E}(u_{12} - u_{02}|\Delta\mathbf{x}, G = 1)]G|\Delta\mathbf{x}\} = 0. \quad (9)$$

This equation (9) essentially requires $Cov(G, u_{02} - u_{01}|\Delta\mathbf{x}) = 0$,[17] which is equivalent to the common trend assumption in equation (6).[18]

Figure 1 shows the histograms with estimated density of BMI based on the two waves of Add Health. The top two panels are for the control group, and the bottom two panels are for the treatment group. The solid vertical line in each panel corresponds to the average BMI. In both groups, there is a mean shift over time. In the control group, the average BMI increases from 22.204 to 22.697 by 0.493 BMI. The treatment group has a more visible mean shift, from 24.131 to 25.266 by 1.135 BMI. In addition to the mean shift, the shapes of both BMI distributions in these two groups remain the same. They are both skewed to the right in the two waves. Figure 2 presents the empirical cumulative distribution functions (CDF) separately for each group over time. In each panel the vertical line corresponds to the average BMI. Figure 2 reveals that the mean shift in BMI in both groups occurs largely as a result of the entire distribution shifting to the right, as opposed to a significant change in a particular part (e.g., tails) of the BMI distribution. The mean and the distribution shift in the control group is consistent with the fact that adolescents gain weight on average in their growth spurt period. It is in our research interest to determine whether, and how much of,

[17] Because $Cov\{G, [(u_{12} - u_{02}) - \mathbb{E}(u_{12} - u_{02}|\Delta\mathbf{x}, G = 1)]G\} = 0$.

[18] Because $Cov(G, u_{02} - u_{01}|\Delta\mathbf{x}) = Var(G|\Delta\mathbf{x})[\mathbb{E}(u_{02} - u_{01}|\Delta\mathbf{x}, G = 1) - \mathbb{E}(u_{02} - u_{01}|\Delta\mathbf{x}, G = 0)]$ and $Var(G|\Delta\mathbf{x}) \neq 0$ unless G is deterministic in $\Delta\mathbf{x}$.

the "excessive" shift in the mean BMI in the treatment group (0.642 BMI) is a direct result of increased exposure to obesity.

3.1.1 Group Mean Comparison

The common trend assumption allows us to use the BMI change over time in the control group as the counterfactual change in the treatment group if it received no treatment. This assumption enables us to separate out any change due to a treatment in the treatment group and to interpret it as the treatment effect. Because this identification assumption cannot be tested directly in the data, we instead compare the means of the Wave I observables between the treatment group and the control group. In the absence of treatment, if the observables in Wave I on average are similar across these two groups, then it may be reasonable to assume that, on average, the unobservables of these two groups likely have similar changes over time.

Table 3 compare of the means of the Wave I observable characteristics of the treatment group and the control group. There are 1,770 adolescents in the control group and 102 in the treatment group. We compare the following sets of characteristics (which are determined prior to the treatment) between the two groups in Wave I: the first set includes an individual's characteristics, including age (or grade), gender, race, birth weight, weekly allowance, whether breast-fed, and whether ever lived with biological parents. The second set includes the individual's family characteristics, including marital status, educational attainment of the primary caregiver (PCG), whether biological parents are obese, and whether living in urban area. The third set includes individuals' own body weight assessment and their weekly exercise frequency. In the first and second sets, most characteristics are very similar on average between the treatment and the control groups, such as birth weight, whether breast-fed, parental educational attainment, and whether living in urban area. However, the treatment group on average has lower family income, more racial diversity, and more obese biological parents. Such differences in these observables should be adjusted for in order to evaluate how much of the difference between the treatment and the control group is solely due to the

treatment. In our later regression analysis, we include variables such as race, family income, and biological parents' obesity status as regressors.

In Figure 3, we use a box-plot of the propensity scores to show how much overlap in observables the two groups share. The propensity score is the probability of being treated conditional on the Wave I observables. We estimated it with a logit model. The boxes represent the distribution of the estimated propensity scores from the 25th to the 75th percentile, with the horizontal line inside the box representing the median. The overlap in the box plots implies the similarities in the observables between the treatment group and the control group prior to the treatment. In Panel A, the following observables from Wave I are included in estimating the propensity score: age, gender, race (White, Black, Asian, or Native), birth weight, whether breast-fed, whether biological mother is obese, whether biological father is obese, PCG's gender and age, whether PCG is married, whether PCG has a college degree, family income in 1994, and whether living in urban area. In Panel B, the same observable characteristics as are used in Panel A are included in estimating the propensity score, except for the following: race (White, Black, Asian, or Native), whether biological mother is obese, whether biological father is obese, and family income in 1994. In Panel B, there is a great amount of overlap in the boxes between the treatment and the control group. This suggests a likely random assignment of the treatment group membership prior to the treatment. In Panel A, the overlap in the boxes shrinks, but a large portion remains when we include those variables that are not similar on the average, as shown in Table 3. Besides at the mean level, the dissimilarity in the distribution of Wave I observables between the treatment and the control groups, to a large extent, may be driven by those variables.

The group mean comparison on the pre-treatment observables in Table 3 and the box plots in Figure 3 at best can suggest a randomly assigned treatment, conditional on the observables. It is possible that the very difference in treatment status is determined precisely by the unobservables and is still a result of self selection. However, DID allows for the self-

selected treatment based to some degree on the unobservables: it only requires the over-time change in the unobservables to be the same, on average, across the two groups. With only cross-sectional data, such selection on the unobservables by the two groups must be removed through exclusion restrictions.

3.1.2 Reverse Causality

In the Add Heath surveys, some students were asked to nominate up to five male and five female friends in order of closeness, while other students were asked to nominate only one best male and one best female friend at most. As discussed earlier, to examine the impact of the change in the number of obese friends on the focal student's weight gain, we need to fix the support of this variable, which should be the same across the students, to be between -2 and 2. Therefore, we consider only best friends. Best friends are also likely to share common factors (probably unobservable to a researcher). Because there is only one or two years between the Add Health's two waves, commonly shared factors (such as interest or hobby) among best friends are likely to be time-invariant (or at least unchanged over a short period). In contrast, commonly shared factors among general friends can be temporal or time-varying (even over a short period). Therefore, using fixed effects at the peer group level seems more appropriate for best friends than for general friends. Because we have individual-level panel data, using individual (disaggregate)-level fixed effects will accommodate any higher (aggregate)-level fixed effects.

Admittedly, there is a trade-off in using best friends instead of general friends. It can be relatively easier to deal with omitted variables through fixed effects among best friends, because the omitted variables are likely to be common factors and to be time invariant over a short period. However, mutual influence on body weight can exist among best friends. For our treatment definition, there are two possible cases. One is the same best friend becoming obese; the other is a different best friend becoming obese. When it is the same best friend that becomes obese between the two waves, we can reasonably rule out reverse causality, because

it seems unlikely that a student's weight gain over a short period of time could make his or her best friend become obese. However, if the student changes best friends between the two waves, and if the new best friends are obese, then reverse causality cannot be fully ruled out. In this case, the effect of an increase in the number of a student's obese friends on his or her own weight gain can either be overestimated or underestimated, depending on the sign of the reverse causality. On the one hand, if a student's weight gain results in doing more exercises with physically fit peers, then we will underestimate the effect of an increased exposure to obesity on weight gain. On the other hand, if a student's weight gain makes him or her identify more with obese peers and befriend them, then we will overestimate the effect of an increased exposure to obesity on weight gain. These two effects in opposite directions may counteract each other on average, and therefore could mitigate the bias in our estimates of average causal effects.

3.1.3 Evidence of Mean Reversion

The treatment effect in the DID setting is the interaction effect of the group and time (GT). One important confounding factor is the mean reversion of body weight (or BMI), which results in the tendency of body weight to revert to its long-term average over time. In a DID setting, mean reversion usually is revealed by the interaction effect of time (T) and the outcome variable in the pre-treatment period. In our case, an individual's weight change can be negatively associated with initial weight in the absence of the treatment. If the treatment is positively associated with initial weight, failure to control for mean reversion will underestimate the treatment effect. In Figures 1 and 2, we notice that students in the treatment group, on average, are heavier (in terms of average BMI) than those in the control group in Wave I. In the absence of treatment, we would expect such a difference to be smaller because of mean reversion. Thus, failing to recognize mean reversion can bias our treatment effect estimate downward.

Figure 4 presents the empirical evidence of mean reversion in the change in BMI. In

Panels A and B, we plot the change in BMI of each student between the two survey periods against the initial BMI in Wave I, for the treatment and the control group respectively. The fitted lines, based on the best linear predictor, are downward sloping, suggesting that the change in BMI is negatively related to the initial BMI. Panels A and B provide the evidence of mean reversion at the individual level. We further find that mean reversion becomes more apparent at an aggregate level. In Panels C and D, we plot changes in the average BMI of each student's best friends between the two waves against the average BMI of the student's best friends in Wave I, for the treatment and the control group respectively. The fitted lines, also based on the best linear predictor, become more downward sloping. Next we use regression analysis to gauge the magnitude of such mean reversion, conditional on a set of observables. We show that the treatment effect can be severely underestimated if we ignore mean reversion, which can offset the treatment effect.

3.2 Econometric Model for Estimation

For estimation, we modify equation (8) to:

$$\Delta y_i = \beta_2 + \Delta \mathbf{x}_i' \beta_{\Delta x} + \mathbf{x}_{1i}' \beta_{x_1} + \beta_{\text{ATT}} G_i + \epsilon_i \ (i = 1, 2, \cdots, N), \tag{10}$$

where $\epsilon \equiv (u_{02} - u_{01}) + [(u_{12} - u_{02}) - \mathbb{E}(u_{12} - u_{02} | \Delta \mathbf{x}, G = 1)]G$. We add Wave I observables, $\mathbf{x}_1$, to equation (8) to capture the interaction effect on the change in BMI between time and the observables in the first period. To examine the robustness of the estimated β_{ATT}, we use five variants of equation (10). In case (1), we only include G and an intercept term controlling for the time effect common to both the treatment and the control group. In case (2), we include the regressors used in case (1) plus the individual i's BMI in Wave I (included into $\mathbf{x}_{1i}$) because of the mean reversion. In case (3), we attempt to capture any peer effects (as examined by recent literature) by including the regressors used in case (2) plus the individual i's best friends' average BMI in Wave I (included into $\mathbf{x}_{1i}$) and the change

in individual i's best friends' average BMI between the two waves (included into $\Delta \mathbf{x}_{1i}$). In case (4), we include the regressors used in case (2) plus the individual i's best friends' average BMI in Wave I and the following covariates (included into $\mathbf{x}_{1i}$): individual i's characteristics (age, gender, race, birth weight, whether breast-fed), whether individual i's mother or father is obese, individual i's primary caregiver's characteristics (gender, age, marital status and education level), household income, and whether living in an urban or rural area. We also include (into $\Delta \mathbf{x}_{1i}$) the change in individual i's average weekly allowance between the two waves. In case (5), we include the regressors used in case (4) plus the change in individual i's best friends' average BMI between the two waves (included into $\Delta \mathbf{x}_{1i}$). Case (5) is the complete specification.

Note that in our DID setting, treatment occurs in the treatment group in the second period and can be denoted by GT. Thus, for the treatment group, β_{ATT} can be interpreted as the effect of an increased exposure to obesity ($G = 1$) on the change in BMI (Δy) as well as the increased exposure to obesity in the second period ($GT = 1$) on BMI (y).

4 Empirical Results

Our overarching goal is to examine one of the consequences of obesity from a social perspective. Therefore, our study considers the empirical evidence supporting Philipson and Posner's (2008) claim that obesity itself can soften its negative image as its prevalence increases, and that "the social aspects of obesity may have a multiplier effect on the growth of obesity," which Levy (2002) also mentions. The existence of such an effect from more people becoming obese on others' weight gain could trigger endogenous growth in the obesity epidemic.

4.1 Impacts of a Change in the Exposure to Obesity

We separate out the change in the exposure to obesity into two cases: an increase and a decrease. We find an asymmetric effect on weight gain as detailed in the next two subsections.

4.1.1 Impacts of Increased Exposure to Obesity

Table 4 reports the estimates of the treatment effect in the five specifications discussed in the last section. In all columns (2) to (5), we confirm the existence of mean reversion in BMI changes over time. This is indicated by the negative and statistically significant relationship between the change in a student's BMI and his or her initial BMI. We also confirm that failing to consider the mean reversion can bias the treatment effect estimate downward, because a reversion to the mean can offset any force that increases the mean. For example, from columns (1) and (2), we find that the treatment effect estimate can increase by nearly 25.6% once we take mean reversion into account. In addition to mean reversion, we also take account of any interaction effects on the weight gain between other Wave I observables and time. In column (5), the complete specification, we find that the treatment effect estimate is statistically significant. It shows that increased exposure to obesity within adolescents' social networks can cause their weight to increase by 0.834 BMI. This can be translated into an approximately 0.2 standard deviation increase based on the summary statistics of Wave I BMI in Table 2.[19] Using the BMI calculation formula and assuming an adolescent with average height of 5.6 feet, we also can translate this 0.834 BMI increase into an approximately 5.36 pound increase between the two survey waves.[20] Note that this is the estimate of the average effect of treatment on the treated.

Next we examine whether such an impact is homogeneous across subpopulations by initial body weight status. Table 5 reveals that it is those who are neither overweight nor obese (i.e., either having optimal weight or being underweight) in Wave I who gain weight

[19] $0.834/4.3012 = 0.1939$.

[20] $(5.6 \cdot 12)^2 \cdot 0.834/703 = 5.357$.

as their exposure to obesity increases between Waves I and II. Estimation results in all odd (and even) numbered columns of Table 5 are based on specification (4) (and 5) of the econometric model. We examine the robustness of the treatment effect estimates using these two specifications for each subpopulation, because such robustness was not found for the overall population in Table 4: the baseline treatment effect estimate remains statistically significant across all specifications except for column (4) in Table 4. In columns (1) and (2) of Table 5, we estimate the model based on equation (10) for those who are not obese in Wave I. The treatment effect estimates are very similar in magnitude to our baseline estimate (0.834 BMI) in Table 4. All of the treatment effect estimates (around 0.9 BMI) in Table 5 remain fairly robust and statistically significant for those who are not obese or who have optimal weight in Wave I. This finding suggests that our baseline estimate (0.834 BMI) comes largely from the not-yet obese population. Our conjecture is confirmed by the results in columns (3) and (4) in Table 5. For those who are already overweight or obese in Wave I, we do not find any significant impact on their weight gain from an increased exposure to obesity. Furthermore, we find that the magnitude of the treatment effect appears to increase as we downgrade the subpopulation's initial body weight status from obesity to underweight. For example, comparing column (1) (or 2) with column (7) (or 8), we find that those who have optimal weight in Wave I on average gain more weight than those who are simply not obese initially—by about 0.11-0.16 BMI—as a result of the increased exposure to obesity. Table 5 suggests that obesity appears able to "infect" those who are initially not yet obese along social ties.

4.1.2 Impacts of Decreased Exposure to Obesity

Conceivably, if a decrease in the exposure to obesity can cause weight loss, then any policy designed to fight the obesity epidemic will be effective. However, our empirical findings do not support this claim. Table 6 repeats the entire exercises in Table 4 by changing only the treatment definition from an increase to a decrease in the number of obese friends between

Add Health's two survey periods. The control group still refers to no change in the number of obese friends between the two waves. Across all five specifications except for column (2) and for the overall population, we find no significant effect of decreased exposure to obesity on weight change. We further examine the subpopulations in Wave I by body weight status. Table 7 repeats the entire exercises in Table 5, except for the different treatment definition. The sample size in Table 5 and Table 7 are similar. Across all specifications in Table 7, we find no statistically significant effect of a decrease in the exposure to obesity on weight change. Note that we detected mean reversion in all the Tables, 4 to 7.

These findings suggest an asymmetric effect. A decreased exposure to obesity within a social network does not cause any statistically significant weight change, regardless of one's initial body weight status. But, an increased exposure to obesity does cause statistically significant weight gain, especially for those who are not yet obese.

4.2 Impacts of a Perceivable Increase in the Exposure to Obesity

In this subsection, we differentiate the increase in exposure to obesity into one that may be perceivable and one that may be unperceivable. If Philipson and Posner's (2008) claim is true—that obesity itself can soften its negative image and cause weight gain as its prevalence increases—then such an effect, possibly coming through the softened negative image of obesity, should occur when the change in obesity prevalence is perceivable. Suppose that on average a student's friends initially have optimal weight. Then this student would experience a more "visible" treatment if the number of his or her obese friends increased. The increase in obesity prevalence might result in a softened negative image of obesity, and subsequently cause the student's weight gain.

Tables 8, 9, and 10 use the same specifications as Table 4, except that the regression analyses are conducted on three subpopulations: the average body weight status of each student's nominated best friends (ranging from 0 to 2) in Wave I is classified by the CDC's criteria as 1) having optimal weight, 2) being either optimal or underweight, or 3) being

either obese or overweight.

Comparing the treatment effect estimates with the full specification in column (5) of both Tables 8 and 9, we find that both estimates are statistically significant and very similar in magnitude (around 0.77 BMI). An increase in the number of obese friends can be perceivable to a student if his or her friends' average body weight status is initially optimal. Based on the estimation results in Tables 8 and 9, we find that when the treatment is perceivable, the effect on weight gain does show up.

Because the clinical threshold for obesity is set by the CDC, a student's increased number of obese friends may be a result of counting the student's friends whose BMI barely exceed the clinical threshold. From the student's perspective, this treatment may not be perceivable at all. If a student gains weight because of a softened negative image of obesity through a perceivable increase in its prevalence, then we would expect no effect on weight gain from an unperceivable change in the prevalence of obesity. Across all the specifications in columns (1) to (5) in Table 10, we find no statistically significant treatment effect estimates, except for the family income effect, for those students whose friends' average body weight status in Wave I is already overweight or obese. Admittedly, the power to detect any treatment effect can be low given the small sample size. Our empirical findings suggest that it may be the perceivable, not the clinical, increase in the exposure to obesity that could potentially soften the negative image of obesity and subsequently cause weight gain.

4.3 Alternative Interpretation

If a self-selected treatment is based on unobservables that do not share a common trend in both the treatment and the control group, then our DID estimates can only be interpreted as a correlation between weight change and change in the exposure to body weight status. Conceivably, this correlation could show up in the relationship between weight change and change in the exposure to the underweight. Here, we examine changes in BMI between the two survey periods in response to: 1) an increased exposure to the underweight and 2) a

decreased exposure to the underweight.

A student's exposure to the underweight is defined by the number of his or her underweight friends whose body weight status is classified as such by the CDC's criteria. The treatment group consists of adolescents who experience an increase (or a decrease) in exposure to the underweight between the two waves. The control group includes those who experience no such change. As in Table 4, Tables 11 and 12 use the five specifications of the econometric model (equation 10) discussed earlier. In both tables, we find no statistically significant effect on weight change from either increased exposure to the underweight (shown in Table 11) or decreased exposure to the underweight (shown in Table 12). This suggests a lack of evidence to support the correlation interpretation.

4.4 Social Ties versus Geographic Proximity

So far, our definition of the exposure to obesity relates to social distance, namely, the number of a student's obese friends who are close to the student socially. We have assembled a set of empirical evidence indicating that some students' becoming obese (between the two waves) can cause weight gain among other students if they are bound by social ties. However, this causal effect may have a different interpretation if based on geographic proximity. Close physical distance is likely to generate commonly shared environmental factors, which cannot be differenced out as fixed effects if they change over time. To check the validity of this alternative interpretation, we redefine exposure to obesity as the number of classmates who are in the same grade and the same school, and who are classified as being obese based on the CDC's criteria. We also examine two cases here: an increase and a decrease in the exposure to obesity between the two waves.

If the effect on weight gain of increased exposure to obesity stems from physical distance as opposed to social distance, then we should find significant effects using the redefined exposure to obesity based on same-grade and same-school classmates who are geographically proximate to the focal student. However, we do not find any supportive empirical evidence.

Tables 13 and 14 report the treatment effect estimates based on the five specifications of the econometric model (equation 10) from an increase and a decrease in the exposure to obesity, respectively. In both cases, and with much larger sample size, we do not find any statistically significant effect of a change in the exposure to obesity from geographically rather than socially close peers on individuals' weight gain.

Our empirical results cross-validate the finding of Christakis and Fowler (2007) and Fowler and Christakis (2008) that weight gain arises from social ties, not common exposure to a local environment.[21] This suggests that any policies to fight obesity prevalence by targeting a geographically clustered group are unlikely to take advantage of "ripple effects" that seem only to exist within a socially close group. In contrast to the geographically clustered group, the socially close group may not be observable to policymakers. This makes it even more difficult to design effective policies for fighting the obesity epidemic.

4.5 Comparing Ours with Existing Studies

Our empirical findings are closely related to the recent literature on peer effects in body weight (as measured by BMI) or body weight status (such as obesity). However, our focus is different. We do not attempt to gauge the impact on individuals' body weight of peers' average body weight. Instead, we focus on whether a change in the number of obese people who are socially close to an individual can cause the individual to gain weight. Based on our empirical findings, we attempt to clarify three points in this paper, which may have been overlooked or underemphasized in the recent peer effect literature. First, it is the *qualitative* (and perceivable) change of peers becoming obese as opposed to the *quantitative* (and marginal) change in peers' average body weight that actually causes weight gain. Second, that effect is *asymmetric*: one is likely to gain weight with increased exposure to obesity, but one is unlikely to lose weight with decreased exposure to obesity. Third, in evaluating the impact

[21] Christakis and Fowler (2007) find that weight gain of immediate neighbors does not affect the probability of self weight gain, while the weight gain of friends, siblings, and spouses increases the chance of self weight gain.

of a treatment on a change in individual's body weight, it is important to take into account mean reversion, that is the tendency of individuals' body weight to revert to the mean over time. With longitudinal data, that type of interaction between time and initial body weight must be controlled for along with individual level unobservables (which can be treated as fixed effects if they are time invariant). In the appendix, we illustrate these three points through two sets of replication exercises: 1) whether to control for school-level fixed effects based on Cohen-Cole and Fletcher's (CCF, 2008) specification, or not to control for them based on Christakis and Fowler's (CF, 2007) specification; and 2) whether to control for individual-level fixed effects or not, following Halliday and Kwak's (HK, 2009) specification.

4.6 Treatment Effect Heterogeneity

We find that an increase in individuals' exposure to obesity causes their weight gain. We identify that effect by isolating an *excessive* change in the treatment group from the control group over two periods of time. In our empirical setting, the treatment group experiences the increase in the exposure to obesity, while the control group experiences no change. Our key identification assumption about common trend allows us to use the change over time in the control group as a counterfactual change in the treatment group in the absence of treatment. However, that assumption does not impose any restrictions on how the unobservables may interact with the treatment when it occurs in the treatment group during the second period. Such an interaction effect can be reflected by the treatment-effect heterogeneity in the treatment group—namely, a nonzero $\mathbb{E}(u_{12} - u_{02}|\Delta\mathbf{x}, G = 1)$ in equation (7). As a result, we only identify an average effect of treatment on the treated (ATT), based on the common trend assumption.

Most of our ATT estimates are around 0.8-0.9 BMI. For an adolescent whose average height is 5.5-5.6 feet and who has more best friends who are becoming obese between the two waves, he or she will be expected to gain five to six more pounds on top of the expected natural weight gain during this growth period. To evaluate whether this treatment can

potentially affect the treatment group more than the control group, or whether the treatment has happened to the most "susceptible" group, we need to identify the average effect of the treatment on the control group (ATC) and compare it with ATT. However, the DID approach does not allow for separately identifying ATT and ATC using the same set of assumptions.

Recall that in equation (5), the ability to identify ATT or ATC depends on which one of the two factual (but unobservable) changes, $\mathbb{E}(u_{12}-u_{01}|\Delta\mathbf{x}, G=1)$ or $\mathbb{E}(u_{02}-u_{01}|\Delta\mathbf{x}, G=0)$, can be used as a counterfactual change. If we use $\mathbb{E}(u_{02}-u_{01}|\Delta\mathbf{x}, G=0)$ as a counterfactual change for the treatment group (which is the previously discussed common trend assumption),

$$\mathbb{E}(u_{02}-u_{01}|\Delta\mathbf{x}, G=1) = \mathbb{E}(u_{02}-u_{01}|\Delta\mathbf{x}, G=0),$$

then we can identify ATT, which is $\beta_d + \mathbb{E}(u_{12}-u_{02}|\Delta\mathbf{x}, G=1)$. Similarly, if we use $\mathbb{E}(u_{12}-u_{01}|\Delta\mathbf{x}, G=1)$ as a counterfactual change for the control group,

$$\mathbb{E}(u_{12}-u_{01}|\Delta\mathbf{x}, G=0) = \mathbb{E}(u_{12}-u_{01}|\Delta\mathbf{x}, G=1), \tag{11}$$

then we can identify ATC, which is $\beta_d + \mathbb{E}(u_{12}-u_{02}|\Delta\mathbf{x}, G=0)$. The difference between ATT and ATC occurs because the unobservables in these two groups can interact differently with the treatment. That difference reveals whether there are any differential "benefits" (or "damages") from the treatment between the two groups, and whether the treatment is adopted optimally by one group based on its differential "benefits" (or "damages") relative to the other group.

The identification assumption for ATC (equation 11) is stronger than the one for ATT (equation 6). The common trend assumption for ATT only restricts how the unobservables change over time under the *same* (untreated) state for both groups. In contrast, the assumption for ATC restricts how the unobservables change over time with *different* states (treated and untreated) for both groups. Here, we stress that it is the same DID estimator that gives us the estimate for either ATT or ATC, depending on whether equation (6) or equation

(11) is used as an identification assumption. Thus, using the DID estimator, we are unable to obtain separate estimates for ATT and ATC under the same identification assumption. This prevents us from evaluating whether there is an optimal treatment adoption by testing whether the difference between the ATT and ATC estimates are statistically significant.

In our empirical setting, the treatment group on average has a higher BMI than the control group prior to the treatment in the first period. We assume that the difference in initial BMI between the two groups caused by the unobservables either remains the same or changes by the same amount on average over the two periods of time. Here, we do not impose any restrictions on how the unobservables in these two groups interact with the treatment. As a result, the treatment-effect heterogeneity attributable to the different interaction effect will reveal whether those in the treatment group (with a higher initial BMI than those in the control group) are more susceptible to the increased exposure and consequently gain more weight than those in the control group would if they were to receive the treatment.

To evaluate the treatment-effect heterogeneity separately for the treated and the control group, we need to estimate the counterfactual distribution of the outcomes for the treatment group in the absence of treatment and the counterfactual distribution of the outcomes for the control group in the presence of treatment, using the changes-in-changes (CIC) approach proposed by Athey and Imbens (2006). The CIC approach enables us to obtain separate estimates for ATT and ATC under a set of different assumptions than DID. Unlike DID, the CIC approach treats the time period and the group asymmetrically. CIC's first key assumption is that, within a time period and in the absence of treatment, the outcome is determined by a *monotonic* mapping from the unobservables, and the monotonic mapping is the same for both groups. CIC's second key assumption is that within each group and in both time periods, the distribution of the unobservables remains the *same*. This assumption can be very restrictive, because it essentially precludes possible interaction effects between the time period and the unobservables within each group. But, it still allows the interaction effect between the treatment (either a factual one or a counterfactual one) and the unobservables

within each group to be different.[22] Our earlier empirical findings on mean reversion in body weight over time bring into question the validity of CIC's second key identification assumption: the negative relationship detected between an initial BMI and the subsequent change in BMI could arise from some time-varying unobservable determinants of the initial BMI. The existence of mean reversion requires that we use the initial BMI as a proxy for the unobservable determinants and include an interaction term which is the initial BMI and the time period in equation (10) under a DID setup. However, the CIC approach omits such an interaction effect.

In Table 15, we report both the DID and CIC estimates. To make them comparable, for both DID and CIC estimations, we do not include covariates, other than the time indicator (T), the group indicator (G), and their interactions (GT and $(1-G)(1-T)$) which indicate actual treatment status. Nor do we include any other interaction terms in the DID estimation. We follow the CIC estimation procedure closely.[23] Under the common trend assumption (equation 6), DID yields an ATT estimate of 0.488 BMI, not too different from the one given by CIC of 0.562 BMI, although obtained under a different set of identification assumptions. We also apply the CIC approach to obtaining the estimated ATC, which is 0.504 BMI, similar to CIC's ATT estimate. Acknowledging that we omit the time-induced interaction effect in both DID and CIC estimations, we find little evidence suggesting an "optimal" treatment adoption based on the differential "benefits" specific to the treatment group. The similarity between the estimates of ATT and ATC using CIC suggests that the adolescents in the treatment group are no more susceptible than those in the control group to an increased exposure to obesity.

[22] Meyer (1995) also points out that a difference-in-differences design can fail to identify the treatment effect in a situation when there is an interaction between the treated group and the time period during which the treatment takes place.

[23] To obtain the standard errors, we use bootstrap. This is also used in the empirical example applying the CIC approach provided in the supplement of Athey and Imbens (2006).

5 Conclusion

In this paper, we have assembled a set of empirical evidence that supports an effect of increased exposure to obesity on weight gain within a social network. Based on Wave I and II of the Add Health surveys, we find that adolescents who experience an increase in the number of obese friends over a one to two-year period subsequently can gain more weight, approximately 0.8-0.9 BMI, relative to a control group for whom there is no change in the number of obese friends over time. That increase in BMI corresponds to approximately five to six pounds for an adolescent with an average height of 5.5-5.6 feet. Specifically, the effect on weight gain of an increased exposure to obesity seems to exist mostly among those who are neither obese nor overweight at the beginning (in Wave I). Furthermore, the effect of an increased exposure to obesity comes largely from a perceivable change in exposure. This suggests that an adolescent may gain weight because of a less intensely negative image of obesity, as discussed by Philipson and Posner (2008). This mechanism takes effect when the increase in the prevalence of obesity is perceivable. Our study also provides evidence to support that the increased exposure to obesity through social ties rather than geographic proximity causes weight gain.

Our findings echo Fowler and Christakis' (2008) conclusion that "people are interconnected, and so their health is interconnected." Moreover, we want to stress two points from our empirical findings that may have been overlooked or underemphasized in the recent peer effect literature. First, it is a qualitative (and perceivable) change from non-obesity to obesity, as opposed to a quantitative (and marginal) change in peers' average body weight, that can cause weight gain. Second, such an effect is asymmetric: it is possible to gain weight with an increased exposure to obesity, but it is not probable that one will lose weight with a decreased exposure to obesity, regardless of initial body weight status. Our study takes an endogenous growth perspective on the prevalence of obesity. We explain whether and how obesity, an individual health outcome at first, has the potential to propagate along social ties and develop into a sweeping epidemic. Our study suggests that it may be an uphill battle

against the obesity epidemic. Policies focused on the prevention of obesity probably should receive a high priority.

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A Peer-Group Level Unobserved Heterogeneity

Christakis and Fowler (2007) use the Framingham Heart Study dataset, which contains a network of 12,067 people who underwent repeated measurements over 32 years, to examine the effects of social influence on obesity. They find that people's obesity status does respond to the obesity status of their peers, including their friends, siblings, spouse, and neighbors. Their regression model is as follows:

$$y_{ijt} = \gamma_0 y_{-ijt} + \gamma_1 y_{-ijt-1} + \mathbf{x}'_{ijt}\beta_0 + \beta_1 y_{ijt-1} + \epsilon_{ijt}, \tag{12}$$

where y_{ijt} and y_{ijt-1} are the obesity status (a binary variable) of an individual i who lives in community j at time t and $t-1$, respectively. Individual i's peers' average obesity status at time t and $t-1$ are y_{-ijt} and y_{-ijt-1}, respectively. The vector of $\mathbf{x}_{ijt}$ includes a set of the individual i's characteristics, such as age, gender, and educational level. The parameter for the peer effect in obesity is γ_0. The researchers use lagged own obesity status (y_{ijt-1}) to control for "genetic endowments" and any other predispositions toward obesity. They also include the peers' lagged average obesity status (y_{-ijt-1}) to correct for "homophily" (i.e., selection bias). They find the estimated γ_0 to be statistically significant, and conclude that the existence of peer effects spreads obesity.

Christakis and Fowler's (2007) study ignites a debate on whether such peer effects really exist after controlling for peer-group level unobserved heterogeneities, which are treated as fixed effects. Cohen-Cole and Fletcher (2008) investigate this obesity peer effect using all three waves of the Add Health survey. They augment CF's (2007) specification (equation 12) by including a set of school-level dummy variables, which are also interacted with time:

$$y_{ijt} = \gamma_0 y_{-ijt} + \gamma_1 y_{-ijt-1} + \mathbf{x}'_{ijt}\beta_0 + \beta_1 y_{ijt-1} + \delta_j + \delta_j T_i + \epsilon_{ijt}, \tag{13}$$

where δ_j and $\delta_j T_i$ represent the school-level fixed effect (δ_j) and the school-time interactive

effect ($\delta_j T_i$), respectively. Cohen-Cole and Fletcher (2008) point out that Christakis and Fowler's (2007) estimates can be biased if these fixed effects are omitted. They show that the inclusion of school-level environmental variables (δ_j), which can be interacted with time trends ($\delta_j T_i$), leads to a large drop in the magnitude of estimated γ_0 in equation (13) as compared with the one estimated in equation (12). In fact, the estimate for γ_0 becomes statistically insignificant after using CCF's (2008) specification (equation 13). Thus, Cohen-Cole and Fletcher (2008) conclude that it is the common environmental factors, or the peer group level unobserved heterogeneity, omitted from CF's (2007) specification (equation 12) that induce the association between own body weight status and that of their peers. In response, Fowler and Christakis (2008) point out that CCF's (2008) peer definition is problematic because they use Wave I nominated best friends and lock them in throughout Wave II and Wave III. That may result in a severe attenuation bias (caused by measurement errors in the peer group composition) in CCF's (2008) findings.[24]

Here, we use the first two waves of the Add Health survey to examine this debate on the obesity peer effect, particularly as discussed by CF (2007) and CCF (2008). We use the best friend nomination in Wave II to update the one in Wave I, and we only use data from Wave I and Wave II only. We report our results in Appendix Table 1. The dependent variables are BMI in columns (1) and (2), and obesity status in columns (3) and (4). For columns (1) and (3), we use CF's (2007) specification (equation 12). For columns (2) and (4), we use CCF's (2008) specification (equation 13).

Using the Add Health's nominated best friends (ranging from 0 to 2) instead of general (possibly) friends (ranging from 0 to 10) yields mixed results for this debate. On the one hand, we find evidence supporting CCF (2008): the effects on individuals' BMI (or obesity status) from the average BMI (or obesity status) of their socially close company—their best friends—do not seem to exist. On the other hand, we find supportive evidence for CF (2007)

[24]Moreover, we find that the friend nomination conducted in Wave III was quite different from the first two waves. Only a small subset of young adults were asked to recognize their friends at the time of the Wave III survey from a list of candidates prepared by Add Health and based on the surveyed adolescents' previous activities in Wave I and Wave II.

and FC (2008): all peer-effect estimates are similar in magnitude regardless of whether we include the school-level fixed effects or not. This suggests that the school-level fixed effect is not the most important confounding factor.

These mixed results on the debate between CF (2007) and FC (2008) are actually consistent with our earlier empirical findings. First, it is not the geographic proximity but rather the social tie that gives rise to peers' influence. Thus, we would expect the school-level fixed effect—a proxy for any common factor shared within a geographically clustered group—to be insignificant. Second, we have previously shown that it is a qualitative (and perceivable) change in peers' becoming obese, not a quantitative (and marginal) change in peers' average body weight, that actually causes weight gain. Third, we know that the obesity feedback effect is asymmetric: one is likely to gain weight with increased exposure to obesity, but it is unlikely to lose weight with decreased exposure to obesity. Overlooking this asymmetry of peer effects could explain in part the insignificant peer-effect estimates in Appendix Table 1. Also, in Appendix Table 1 the self's lagged BMI or obesity status remains statistically significant with a magnitude of less than 1, which suggests the existence of mean reversion in self's BMI over time.

B Individual-Level Unobserved Heterogeneity

Another difficulty in identifying the peer effects in BMI or obesity status arises from individual-level unobserved heterogeneity. If this heterogeneity is time invariant, then a fixed-effect model is appropriate to use with panel data. However, there is a trade-off in adding or dropping the fixed effect: ignoring the fixed effect is likely to bias the peer-effect estimate upward, because an individual's habits or work environment, invariant within a short period, can both cause weight gain and give the individual opportunities to hang out with overweight friends. On the other hand, adding the fixed effect may bias the peer-effect estimate downward, because if there are measurement errors in the peers' average BMI or obesity status,

then the two-period fixed-effect model—which is equivalent to a first-difference model with two periods—can exacerbate the measurement errors and result in a greater attenuation bias.

Halliday and Kwak (HK, 2009) confirm this trade-off using the first two waves of the Add Health survey. They specify their regression model as follows:

$$y_{ijt} = \gamma_0 y_{-ijt} + \mathbf{x}'_{ij}\beta_0 + \alpha_i + \delta_j + \epsilon_{ijt}, \tag{14}$$

where y_{ijt} is BMI (or body weight status) for individual i at school j in time period t. y_{-ijt} is the average of peers' BMI (or body weight status), and $\mathbf{x}_{ijt-1}$ is a vector of control variables. They also consider the nominated friends (ranging from 0 to 10) to be the peer group. Either individual or school-level fixed effect, α_i or δ_j, is included in their estimation model based on equation (14).

Halliday and Kwak (2009) find that the estimated peer effect, indicated by γ_0, becomes statistically insignificant when α_i is included, while the peer-effect estimate is statistically significant when α_i is replaced by δ_j. Using HK's (2009) specification but replacing their peer definition with the nominated best friend (ranging from 0 to 2), we find the results in Appendix Table 2 to be consistent with HK (2009). In Appendix Table 2, the dependent variables are BMI in columns (1) and (2), and obesity status in columns (3) and (4). Columns (1) and (3) only include δ_j in equation (14). Columns (2) and (4) only include α_i in equation (14). Both HK's (2009) study and ours use the CDC's percentile criteria to classify the body weight status of children and adolescents.[25] The important difference between the two studies is the criterion for the definition of a peer, although both are based on friend nomination. As mentioned earlier, in Add Health, some students were allowed to nominate up to five male and five female friends in order of closeness while others were only allowed to nominate one best male friend and one best female friend. Halliday and Kwak (2009) include all of

[25]Because changes in adolescent BMI are highly susceptible to adolescent growth spurts, we can have serious measurement errors in body weight status (resulting in attenuation bias in the peer effect estimates) if the adult definitions are mistakenly used.

the nominated friends (ranging from 0 to 10) in a student's peer group. But some students were allowed to nominate more friends than others, so the peer group is thus defined with different levels of closeness and it differs as to quality. As a result, the average peer BMI based on the different nomination requirements (and therefore, the different degrees of social closeness) that is being compared in the regression actually is not comparable. In contrast, we only use the nominated best friends (up to one male and one female friend) ranging from 0 to 2.

In Appendix Table 1, we find that the lagged self BMI or obesity status is highly statistically significant based on CF's (2007) or CCF's (2008) specification. That appears to favor using individual-level rather than school-level fixed effects (even at the cost of measurement errors). The peer-effect estimates in Appendix Table 2, based on HK's (2009) specification, also could be biased downward because of an overlooked nonlinear effect in best friends' influence. That is the focus of our study: the nonlinear (asymmetric) effect of a change in the exposure to obesity on weight gain. Such a nonlinear effect in best friends' influence only becomes salient once the best friend's BMI crosses the obesity threshold.

Table 1: Percentiles of Body Mass Index Distribution for Children Ages 11-20 Years

Age		95th Percentitle BMI Value		85th Percentitle BMI Value		5th Percentitle BMI Value	
(in years)	(in months)	Male	Female	Male	Female	Male	Female
11	132	23.21358	24.14141	20.19667	20.86984	14.56001	14.40290
12	144	24.22985	25.25564	21.02386	21.74263	14.97745	14.83262
13	156	25.17811	26.29880	21.85104	22.57506	15.45918	15.30749
14	168	26.04662	27.25597	22.66325	23.34689	15.99065	15.80753
15	180	26.83688	28.12369	23.45117	24.04503	16.55481	16.30974
16	192	27.56393	28.90981	24.21087	24.66372	17.13250	16.78787
17	204	28.25676	29.63350	24.94362	25.20482	17.70284	17.21234
18	216	28.95862	30.32554	25.65601	25.67786	18.24349	17.55015
19	228	29.72674	31.02880	26.36054	26.09993	18.73019	17.76515
20	240	30.58964	31.76474	27.04607	26.47872	19.12055	17.82009

Note: Body Mass Index (BMI) is calculated using the following formula: BMI = weight (in kilograms)/height (in meters)^2 or BMI = 703*weight (in pounds)/height (in inches)^2. According to the distributions given in the Growth Charts of the Centers for Disease Control and Prevention (CDC), for children ages 2-20 years, "obese" is defined to be ≥ 95th percentile of BMI for the appropriate age-sex group; "overweight" is defined to be ≥ 85th percentile of BMI and < 95th percentile of BMI for the appropriate age-sex group; "optimal weight" is defined to be ≥ 5th percentile and < 85th percentile of BMI for the appropriate age-sex group; "underweight" is defined to be < 5th percentile of BMI for the appropriate age-sex group. For both male and female adults aged at least 21 years, "obese" is defined to be BMI ≥ 30, "overweight" to be BMI ≥ 25 and BMI < 30, "optimal weight" to be BMI ≥ 18.5 and BMI < 25, "underweight" to be BMI < 18.5. This table is reproduced from CDC. More details are provided in the following website: http://www.cdc.gov/growthcharts/html_charts/bmiagerev.htm.

Table 2: Summary Statistics

Variables	Wave I		Wave II	
	Mean	Standard deviation	Mean	Standard deviation
Self's Body Mass Index (BMI) and body weight status				
Height (in foot)	5.5266	0.3258	5.5833	0.3250
Weight (in pound)	139.9113	33.8552	145.5701	34.5774
BMI	22.2119	4.3012	22.6723	4.4160
Obese (1/0)	0.1106	0.3138	0.1030	0.3041
Overweight (1/0)	0.1394	0.3465	0.1436	0.3508
Optimal weight (1/0)	0.7340	0.4421	0.7297	0.4443
Underweight (1/0)	0.0160	0.1257	0.0236	0.1520
Best friends' (peers') BMI and body weight status				
Height (in foot)	5.5156	0.2876	5.5732	0.2874
Weight (in pound)	135.5629	27.4265	143.0296	30.1698
BMI	21.6012	3.2835	22.3562	3.8238
Obese (1/0)	0.0418	0.1888	0.0832	0.2585
Overweight (1/0)	0.1486	0.3345	0.1204	0.3106
Optimal weight (1/0)	0.7918	0.3818	0.7821	0.3920
Underweight (1/0)	0.0177	0.1238	0.0144	0.1154
Number of nominated peers	1.5144	0.5000	1.4248	0.4945
Number of peers being obese	0.0507	0.2194	0.1047	0.3145
Number of peers being overweight	0.1850	0.4055	0.1402	0.3498
Number of peers with optimal weight	0.9932	0.5771	0.9130	0.5234
Number of peers being underweight	0.0220	0.1466	0.0160	0.1257
Self's times of exercises per week				
None (1/0)	0.1360	0.3429	0.1275	0.3337
1 or 2 (1/0)	0.3235	0.4680	0.3530	0.4781
3 or 4 (1/0)	0.2770	0.4477	0.2753	0.4469
5 or more (1/0)	0.2635	0.4407	0.2441	0.4297
Self's body image assessment				
Very underweight (1/0)	0.0144	0.1190	0.0068	0.0820
Slightly underweight (1/0)	0.1520	0.3592	0.1470	0.3542
About right (1/0)	0.5431	0.4984	0.5574	0.4969
Slightly overweight (1/0)	0.2618	0.4398	0.2618	0.4398
Very overweight (1/0)	0.0287	0.1671	0.0270	0.1622
Self wanting to change weight				
To lose weight (1/0)	0.3260	0.4690	0.3145	0.4645
To gain weight (1/0)	0.1807	0.3850	0.1817	0.3858
To keep the same weight (1/0)	0.3733	0.4839	0.3491	0.4769
To do nothing (1/0)	0.1199	0.3250	0.1547	0.3618
Peers' times of exercises per week				
None (1/0)	0.1334	0.3204	0.1360	0.3265
1 or 2 (1/0)	0.3361	0.4378	0.3332	0.4516
3 or 4 (1/0)	0.2652	0.4109	0.2724	0.4262
5 or more (1/0)	0.2652	0.4109	0.2584	0.4202
Peers' body image assessment				
Very underweight (1/0)	0.0101	0.0937	0.0076	0.0844
Slightly underweight (1/0)	0.1554	0.3377	0.1486	0.3413
About right (1/0)	0.5798	0.4594	0.5819	0.4716
Slightly overweight (1/0)	0.2352	0.3952	0.2306	0.4008
Very overweight (1/0)	0.0194	0.1269	0.0313	0.1653
Peers' wanting to change weight				
To lose weight (1/0)	0.3062	0.4305	0.3074	0.4405
To gain weight (1/0)	0.1871	0.3582	0.1867	0.3743
To keep the same weight (1/0)	0.3919	0.4566	0.3666	0.4615
To do nothing (1/0)	0.1149	0.2985	0.1394	0.3309
Self's characteristics				
Grade	9.3767	1.3930	10.3230	1.3806
Age	15.2475	1.4923	16.1740	1.5147

Male (1/0)	0.4932	0.5002	0.4932	0.5002
White (1/0)	0.7348	0.4416	0.7348	0.4416
Black (1/0)	0.1368	0.3438	0.1368	0.3438
Asian (1/0)	0.0794	0.2705	0.0794	0.2705
Native (1/0)	0.0313	0.1741	0.0313	0.1741
Other (1/0)	0.0608	0.2391	0.0608	0.2391
Allowance (dollar/week)	6.5194	9.5624	7.0929	10.8744
Birth weight (in pound)	7.4199	1.2659	7.4199	1.2659
Breastfed (1/0)	0.4882	0.5001	0.4882	0.5001
Peers' characteristics				
Grade	9.4303	1.4131	10.3432	1.3726
Age	15.3074	1.5008	16.2036	1.4692
Male (1/0)	0.4645	0.4308	0.4797	0.4532
White (1/0)	0.7413	0.4298	0.7401	0.4347
Black (1/0)	0.1327	0.3344	0.1293	0.3332
Asian (1/0)	0.0850	0.2747	0.0862	0.2785
Native (1/0)	0.0300	0.1558	0.0325	0.1683
Other (1/0)	0.0609	0.2292	0.0600	0.2331
Allowance (dollar/week)	6.4456	8.6345	6.9603	9.5565
Birth weight (in pound)	7.3708	1.1618	7.3295	1.2075
Breastfed (1/0)	0.4967	0.4756	0.5075	0.4824
Self's parent's characteristics				
Biological mother obese (1/0)	0.1774	0.3821	0.1774	0.3821
Biological father obese (1/0)	0.1132	0.3169	0.1132	0.3169
Male primary caregiver (PCG) (1/0)	0.0507	0.2194	0.0507	0.2194
PCG's age	41.8066	5.9363	41.8066	5.9363
PCG married (1/0)	0.7736	0.4186	0.7736	0.4186
PCG having a college degree (1/0)	0.2703	0.4443	0.2703	0.4443
Ever lived with biological mother (1/0)	0.7843	0.4154	0.7843	0.4154
Ever lived with biological father (1/0)	0.7540	0.4313	0.7540	0.4313
Family income (in thousand dollar) in 1994	50.1647	51.2786	50.1647	51.2786
Peers' parent's characteristics				
Biological mother obese (1/0)	0.1545	0.3393	0.1446	0.3356
Biological father obese (1/0)	0.0914	0.2660	0.0974	0.2832
PCG married (1/0)	0.6930	0.4358	0.7006	0.4401
PCG having a college degree (1/0)	0.2386	0.4058	0.2420	0.4157
Ever lived with biological mother (1/0)	0.8491	0.3597	0.8812	0.3252
Ever lived with biological father (1/0)	0.7062	0.4506	0.6990	0.4537
Family income (in thousand dollar) in 1994	52.2233	65.2933	52.1573	59.7561
Living in urban or rural area				
Urban (1/0)	0.4054	0.4912	0.4046	0.4910
Number of observations used in estimation	1,184		1,184	

Note: Data are from The National Longitudinal Study of Adolescent Health (Add Health) in-home interviews which took place in Wave I (1994-1995) and Wave II (1996). Parental survey was conducted only in Wave I. In both survey waves, a student (in grade 7-12) was asked to nominate up to one best male friend and one best female friend. The number of nominated best friend is between one and two. The Body Mass Index (BMI) is calculated using the following formula: BMI = 703*weight (in pounds)/height (in inches)^2. The body weight status ("obese", "overweight", "optimal weight" and "underweight") is classified based on the 95th, the 85th and the 5th percentile of the BMI distribution given in the Growth Charts of the Centers for Disease Control and Prevention (CDC), for children ages 2-20 years. For those students aged at least 21 years, "obese" is defined to be BMI $\geq$ 30, "overweight" to be BMI $\geq$ 25 and BMI < 30, "optimal weight" to be BMI $\geq$ 18.5 and BMI < 25, "underweight" to be BMI < 18.5. Variables with a 1/0 value is binary. The survey question about "allowance" is "how much is your allowance each week" ranging from 0 to $95. The survey question about "family income" is "about how much total income before taxes did your family receive in 1994", ranging from 0 to $999,000. The original sample sizes are 20,745 in Wave I and 14,738 in Wave II. The best friend nomination includes both friends in the same school as well as friends from outside school for whom the information is not available in the Add Health original data. Because the nominated best friends in the same school may not be selected to receive either in-home interview, such information regarding the best friends will not be available in the estimation sample. This results in a substantial reduction in the sample size for estimation. In this table, we report the number of observations used in both waves for the difference-in-differences estimation. The information on average BMI in our estimation sample has been checked and found to be very similar to other studies on BMI using Add Health data.

Table 3: Treatment-Control Group Mean Comparison on Individual's Wave I Observables

Variables	Mean		*t* -test	
	Control	Treatment	*t* -ratio	*p* -value
Grade	9.4486	9.3600	0.6427	0.5217
Age	15.3339	15.2059	0.8764	0.3827
Male (1/0)	0.4870	0.4902	-0.0624	0.9504
Whitel (1/0)	0.6912	0.5490	2.8029	0.0060
Black (1/0)	0.1493	0.2353	-1.9970	0.0483
Asian (1/0)	0.1012	0.1471	-1.2739	0.2054
Native (1/0)	0.0305	0.0784	-1.7695	0.0797
Other (1/0)	0.0730	0.0588	0.5839	0.5604
Allowance (dollars/week)	6.3941	6.6337	-0.2585	0.7965
Birth weight (pounds)	7.4160	7.3623	0.3774	0.7068
Breastfed (1/0)	0.4774	0.4146	1.1162	0.2673
Times of exercises per week				
None (1/0)	0.1469	0.0980	1.5881	0.1149
1 or 2 (1/0)	0.3209	0.2647	1.2411	0.2171
3 or 4 (1/0)	0.2599	0.3039	-0.9381	0.3502
5 or more (1/0)	0.2723	0.3333	-1.2689	0.2071
Body image assessment				
Very underweight (1/0)	0.0153	0.0294	-0.8297	0.4085
Slightly underweight (1/0)	0.1452	0.1275	0.5186	0.6051
About right (1/0)	0.5401	0.4314	2.1454	0.0341
Slightly overweight (1/0)	0.2627	0.3431	-1.6622	0.0993
Very overweight (1/0)	0.0367	0.0686	-1.2487	0.2145
Wanting to change weight				
To lose weight (1/0)	0.3322	0.4804	-2.9080	0.0044
To gain weight (1/0)	0.1825	0.1078	2.3180	0.0221
To keep the same weight (1/0)	0.3644	0.3431	0.4376	0.6625
To do nothing (1/0)	0.1209	0.0686	1.9859	0.0493
Biological mother obese (1/0)	0.1658	0.2651	-1.9993	0.0487
Biological father obese (1/0)	0.1054	0.2048	-2.1977	0.0306
Male primary caregiver (PCG) (1/0)	0.0632	0.0471	0.6760	0.5007
PCG's age	41.8310	41.8140	0.0207	0.9835
PCG married (1/0)	0.6802	0.6275	1.0690	0.2874
PCG having a college degree (1/0)	0.2311	0.2451	-0.3139	0.7503
Ever lived with biological mother (1/0)	0.8162	0.7778	0.2548	0.8047
Ever lived with biological father (1/0)	0.7086	0.6129	1.0511	0.3008
Family income (in thousand dollars) in 1994	49.7329	41.3133	2.4479	0.0158
Living in urban area (1/0)	0.4407	0.4216	0.3791	0.7054
Number of observations	1,770	102		

Note: Data are from The National Longitudinal Study of Adolescent Health (Add Health) in-home interviews which took place in Wave I (1994-1995) and Wave II (1996). Parental survey was conducted only in Wave I. In both survey waves, a student (in grade 7-12) was asked to nominate up to one best male friend and one best female friend. The number of nominated best friend is between one and two. The Body Mass Index (BMI) is calculated using the following formula: BMI = 703*weight (in pounds)/height (in inches)^2. The body weight status ("obese", "overweight", "optimal weight" and "underweight") is classified based on the 95th, the 85th and the 5th percentile of the BMI distribution given in the Growth Charts of the Centers for Disease Control and Prevention (CDC), for children ages 2-20 years. For those students aged at least 21 years, "obese" is defined to be BMI $\geq$ 30, "overweight" to be BMI $\geq$ 25 and BMI < 30, "optimal weight" to be BMI $\geq$ 18.5 and BMI < 25, "underweight" to be BMI < 18.5. Variables with a 1/0 value is binary. For each wave, the exposure to obesity is defined by the number of best friends who are nominated by the adolescent and who are classified as being obese. The treatment group is the one that each adolescent in the group experienced an increase in the exposure to obesity between the two survey waves. The control group is the one that each adolescent in the group experienced no change (neither an increase nor a decrease) in the exposure to obesity between the two survey periods. A two-tailed *t* -test with unequal variances is conducted to test whether the means of the individual's observables in Wave I are equal between the treatment and the control group. Both the *t* -ratio and the *p* -value of the *t* -test are reported.

Table 4: Impacts of an Increase in the Exposure to Obesity on Weight Gains

Dependnet Variable: change in self's BMI	(1)	(2)	(3)	(4)	(5)
Increased exposure to obesity (treatment)	0.488*	0.613**	0.748**	0.546	0.834**
	(0.280)	(0.282)	(0.322)	(0.355)	(0.402)
Self's BMI in Wave I		-0.062***	-0.063***	-0.087***	-0.085***
		(0.015)	(0.015)	(0.021)	(0.021)
Peers' average BMI in Wave I			0.010	-0.016	-0.031
			(0.020)	(0.019)	(0.021)
Change in peers' average BMI between waves			-0.020		-0.045*
			(0.020)		(0.027)
Age				0.014	0.017
				(0.044)	(0.044)
Male (1/0)				0.239**	0.255**
				(0.113)	(0.112)
White (1/0)				-0.004	0.004
				(0.198)	(0.199)
Black (1/0)				0.076	0.105
				(0.253)	(0.254)
Asian (1/0)				-0.082	-0.070
				(0.235)	(0.237)
Native (1/0)				-0.295	-0.278
				(0.256)	(0.256)
Birth weight (in pound)				-0.019	-0.016
				(0.047)	(0.047)
Breastfed (1/0)				-0.035	-0.034
				(0.113)	(0.113)
Biological mother obese (1/0)				0.275	0.281
				(0.182)	(0.182)
Biological father obese (1/0)				0.255	0.254
				(0.216)	(0.215)
Male primary caregiver (PCG) (1/0)				0.187	0.208
				(0.196)	(0.196)
PCG's age				0.004	0.003
				(0.010)	(0.010)
PCG married (1/0)				-0.169	-0.175
				(0.144)	(0.144)
PCG having a college degree (1/0)				-0.139	-0.157
				(0.121)	(0.122)
Family income (in thousand dollar) in 1994				-0.001	-0.001
				(0.001)	(0.001)
Living in urban area (1/0)				0.166	0.165
				(0.126)	(0.126)
Change in allowance (dollar/week)				-0.006	-0.006
				(0.005)	(0.005)
Constant	0.452***	1.820***	1.642***	2.410***	2.660***
	(0.045)	(0.313)	(0.480)	(0.844)	(0.870)
Observations	1,816	1,816	1,816	1,184	1,184

Note: Data are from The National Longitudinal Study of Adolescent Health (Add Health) in-home interviews which took place in Wave I (1994-1995) and Wave II (1996). Parental survey was conducted only in Wave I. Definitions of variables are given in the summary statistics table. Variables with a 1/0 value is binary. For each wave, the exposure to obesity is defined by the number of best friends who are nominated by the adolescent and who are classified as being obese. The treatment group is the one that each adolescent in the group experienced an increase in the exposure to obesity between the two survey waves. The control group is the one that each adolescent in the group experienced no change (neither an increase nor a decrease) in the exposure to obesity between the two survey periods. The dependent variable is the change in self's BMI between the two waves. In column (1), we include only the treatment dummy variable and an intercept term. In column (2), we add self's BMI in Wave I. In column (3), we add peers' average BMI in Wave I and the change in peers' average BMI between the two waves. In column (4), we include the regressors in column (2) plus peers' average BMI in Wave I and other Wave I covariates and the change in self's allowance between the two waves. In column (5), we include the regressors in column (4) plus the change in peers' average BMI between the two waves. Heteroskedasticity-robust standard errors are in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

Table 5: Impacts of an Increase in the Exposure to Obesity on Weight Gains by Body Weight Status in Wave I

Dependnet Variable: change in self's BMI	Not Obese		Overweight or Obese		Not Overweight Nor Obese		Optimal Weight		Not Underweight	
Variables	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Increased exposure to obesity (treatment)	0.835**	0.932**	0.232	0.870	0.855*	1.014*	0.947*	1.091**	0.586	0.864**
	(0.401)	(0.416)	(0.502)	(0.810)	(0.517)	(0.533)	(0.539)	(0.550)	(0.366)	(0.410)
Self's BMI in Wave I	-0.090***	-0.090***	-0.094*	-0.085*	-0.106***	-0.105***	-0.100***	-0.099***	-0.086***	-0.084***
	(0.023)	(0.023)	(0.052)	(0.050)	(0.028)	(0.028)	(0.029)	(0.029)	(0.021)	(0.021)
Peers' average BMI in Wave I	-0.013	-0.018	-0.026	-0.067	-0.004	-0.011	-0.002	-0.008	-0.014	-0.028
	(0.018)	(0.018)	(0.045)	(0.065)	(0.018)	(0.018)	(0.018)	(0.019)	(0.019)	(0.022)
Change in peers' average BMI between waves		-0.016		-0.103		-0.024		-0.022		-0.044
		(0.020)		(0.088)		(0.021)		(0.021)		(0.027)
Age	-0.008	-0.007	0.204	0.211*	-0.037	-0.036	-0.043	-0.042	0.014	0.017
	(0.044)	(0.043)	(0.124)	(0.126)	(0.045)	(0.045)	(0.044)	(0.044)	(0.045)	(0.045)
Male (1/0)	0.244**	0.250**	0.193	0.213	0.276***	0.287***	0.298***	0.308***	0.257**	0.273**
	(0.105)	(0.105)	(0.348)	(0.342)	(0.104)	(0.103)	(0.104)	(0.104)	(0.114)	(0.114)
White (1/0)	0.031	0.033	-0.187	-0.278	0.087	0.100	0.047	0.058	-0.042	-0.035
	(0.182)	(0.183)	(0.483)	(0.500)	(0.197)	(0.198)	(0.201)	(0.201)	(0.201)	(0.202)
Black (1/0)	0.184	0.194	0.069	0.097	0.184	0.202	0.129	0.145	0.042	0.069
	(0.250)	(0.250)	(0.565)	(0.576)	(0.268)	(0.270)	(0.272)	(0.273)	(0.255)	(0.256)
Asian (1/0)	-0.016	-0.014	-0.922	-0.853	0.081	0.089	0.059	0.068	-0.106	-0.093
	(0.221)	(0.222)	(0.780)	(0.804)	(0.234)	(0.235)	(0.237)	(0.238)	(0.238)	(0.240)
Native (1/0)	-0.278	-0.276	-0.066	-0.014	-0.284	-0.279	-0.282	-0.277	-0.297	-0.280
	(0.235)	(0.234)	(0.648)	(0.675)	(0.243)	(0.241)	(0.243)	(0.242)	(0.255)	(0.255)
Birth weight (in pound)	-0.048	-0.047	-0.047	-0.055	-0.023	-0.021	-0.027	-0.024	-0.023	-0.019
	(0.047)	(0.047)	(0.119)	(0.118)	(0.050)	(0.050)	(0.050)	(0.050)	(0.048)	(0.047)
Breastfed (1/0)	-0.010	-0.009	0.100	0.090	-0.032	-0.031	-0.031	-0.030	-0.000	0.000
	(0.022)	(0.021)	(0.062)	(0.064)	(0.022)	(0.022)	(0.022)	(0.022)	(0.022)	(0.022)
Biological mother obese (1/0)	0.132	0.136	0.599	0.589	0.104	0.112	0.103	0.111	0.277	0.283
	(0.169)	(0.169)	(0.420)	(0.413)	(0.175)	(0.175)	(0.176)	(0.176)	(0.183)	(0.183)
Biological father obese (1/0)	0.491***	0.492***	0.076	0.057	0.344**	0.347**	0.349**	0.352**	0.259	0.258
	(0.172)	(0.172)	(0.501)	(0.499)	(0.172)	(0.172)	(0.172)	(0.172)	(0.215)	(0.214)
Male primary caregiver (PCG) (1/0)	0.266	0.272	-0.359	-0.324	0.407*	0.416*	0.309	0.320	0.112	0.135
	(0.217)	(0.218)	(0.368)	(0.377)	(0.236)	(0.235)	(0.229)	(0.229)	(0.191)	(0.192)
PCG's age	-0.004	-0.004	0.011	0.005	0.003	0.003	0.003	0.003	0.004	0.003
	(0.009)	(0.009)	(0.024)	(0.023)	(0.009)	(0.009)	(0.009)	(0.009)	(0.010)	(0.010)
PCG married (1/0)	-0.160	-0.163	0.006	-0.060	-0.192	-0.194	-0.245*	-0.247*	-0.212	-0.218
	(0.140)	(0.140)	(0.377)	(0.381)	(0.141)	(0.141)	(0.142)	(0.142)	(0.145)	(0.145)
PCG having a college degree (1/0)	-0.107	-0.115	-0.580	-0.617	-0.077	-0.088	-0.110	-0.119	-0.175	-0.191
	(0.117)	(0.117)	(0.374)	(0.377)	(0.121)	(0.122)	(0.122)	(0.122)	(0.119)	(0.120)
Family income (in thousand dollar) in 1994	-0.001	-0.001	0.006	0.007	-0.001**	-0.001**	-0.001*	-0.001*	-0.001	-0.001
	(0.001)	(0.001)	(0.006)	(0.006)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Living in urban area (1/0)	0.103	0.104	0.344	0.294	0.134	0.138	0.145	0.149	0.171	0.170
	(0.122)	(0.122)	(0.362)	(0.356)	(0.121)	(0.120)	(0.122)	(0.122)	(0.127)	(0.126)
Change in allowance (dollar/week)	-0.001	-0.001	-0.014	-0.015	-0.001	-0.001	-0.002	-0.002	-0.006	-0.007
	(0.005)	(0.005)	(0.011)	(0.011)	(0.006)	(0.006)	(0.006)	(0.006)	(0.005)	(0.005)
Constant	3.266***	3.350***	-0.964	0.093	3.520***	3.611***	3.494***	3.575***	2.409***	2.651***
	(0.820)	(0.841)	(2.242)	(2.480)	(0.786)	(0.798)	(0.807)	(0.818)	(0.852)	(0.879)
Observations	1,053	1,053	296	296	888	888	869	869	1,165	1,165

Note: Data are from The National Longitudinal Study of Adolescent Health (Add Health) in-home interviews which took place in Wave I (1994-1995) and Wave II (1996). Parental survey was conducted only in Wave I. Definitions of variables are given in the summary statistics table. Variables with a 1/0 value is binary. For each wave, the exposure to obesity is defined by the number of best friends who are nominated by the adolescent and who are classified as being obese. The treatment group is the one that each adolescent in the group experienced an increase in the exposure to obesity between the two survey waves. The control group is the one that each adolescent in the group experienced no change (neither an increase nor a decrease) in the exposure to obesity between the two survey periods. The dependent variable is the change in self's BMI between the two waves. Estimates reported in columns (1) and (2) are based on the adolescents who were classified as not being obese in Wave I. Estimates reported in columns (3) and (4) are based on the adolescents who were classified as being either overweight (but not obese) or obese in Wave I. Estimates reported in columns (5) and (6) are based on the adolescents who were classified as being neither overweight (but not obese) nor obese in Wave I. Estimates reported in columns (7) and (8) are based on the adolescents who were classified as having optimal weight in Wave I. Estimates reported in columns (9) and (10) are based on the adolescents who were classified as not being underweight in Wave I. Heteroskedasticity-robust standard errors are in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

Table 6: Impacts of a Decrease in the Exposure to Obesity on Weight Gains

Dependnet Variable: change in self's BMI	(1)	(2)	(3)	(4)	(5)
Decreased exposure to obesity (treatment)	0.297	0.391*	0.275	0.430	0.238
	(0.211)	(0.213)	(0.238)	(0.303)	(0.297)
Self's BMI in Wave I		-0.057***	-0.058***	-0.078***	-0.076***
		(0.015)	(0.015)	(0.021)	(0.021)
Peers' average BMI in Wave I			0.002	-0.018	-0.035*
			(0.019)	(0.018)	(0.021)
Change in peers' average BMI between waves			-0.017		-0.051*
			(0.021)		(0.027)
Age				0.008	0.011
				(0.042)	(0.042)
Male (1/0)				0.210*	0.228**
				(0.112)	(0.110)
White (1/0)				-0.002	0.013
				(0.188)	(0.189)
Black (1/0)				-0.074	-0.038
				(0.233)	(0.234)
Asian (1/0)				-0.146	-0.140
				(0.232)	(0.235)
Native (1/0)				-0.394	-0.380
				(0.265)	(0.260)
Birth weight (in pound)				0.003	0.006
				(0.047)	(0.047)
Breastfed (1/0)				0.012	0.013
				(0.022)	(0.022)
Biological mother obese (1/0)				0.194	0.198
				(0.184)	(0.184)
Biological father obese (1/0)				0.295	0.284
				(0.228)	(0.226)
Male primary caregiver (PCG) (1/0)				0.087	0.100
				(0.192)	(0.192)
PCG's age				0.006	0.005
				(0.010)	(0.009)
PCG married (1/0)				-0.168	-0.175
				(0.140)	(0.140)
PCG having a college degree (1/0)				-0.063	-0.083
				(0.118)	(0.119)
Family income (in thousand dollar) in 1994				-0.001	-0.001
				(0.001)	(0.001)
Living in urban area (1/0)				0.145	0.153
				(0.119)	(0.119)
Change in allowance (dollar/week)				-0.007	-0.008
				(0.005)	(0.005)
Constant	0.452***	1.724***	1.693***	2.094***	2.379***
	(0.045)	(0.318)	(0.480)	(0.810)	(0.829)
Observations	1,827	1,827	1,827	1,184	1,184

Note: Data are from The National Longitudinal Study of Adolescent Health (Add Health) in-home interviews which took place in Wave I (1994-1995) and Wave II (1996). Parental survey was conducted only in Wave I. Definitions of variables are given in the summary statistics table. Variables with a 1/0 value is binary. For each wave, the exposure to obesity is defined by the number of best friends who are nominated by the adolescent and who are classified as being obese. The treatment group is the one that each adolescent in the group experienced a decrease in the exposure to obesity between the two survey waves. The control group is the one that each adolescent in the group experienced no change (neither an increase nor a decrease) in the exposure to obesity between the two survey periods. The dependent variable is the change in self's BMI between the two waves. In column (1), we include only the treatment dummy variable and an intercept term. In column (2), we add self's BMI in Wave I. In column (3), we add peers' average BMI in Wave I and the change in peers' average BMI between the two waves. In column (4), we include the regressors in column (2) plus peers' average BMI in Wave I and other Wave I covariates and the change in self's allowance between the two waves. In column (5), we include the regressors in column (4) plus the change in peers' average BMI between the two waves. Heteroskedasticity-robust standard errors are in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

Table 7: Impacts of a Decrease in the Exposure to Obesity on Weight Gains by Body Weight Status in Wave I

Dependnet Variable: change in self's BMI	Not Obese		Overweight or Obese		Not Overweight Nor Obese		Optimal Weight		Not Underweight	
Variables	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Decreased exposure to obesity (treatment)	0.190	0.146	0.693	0.062	0.274	0.242	0.294	0.262	0.439	0.250
	(0.269)	(0.283)	(0.604)	(0.548)	(0.305)	(0.322)	(0.315)	(0.331)	(0.310)	(0.303)
Self's BMI in Wave I	-0.105***	-0.104***	-0.068	-0.062	-0.129***	-0.129***	-0.125***	-0.124***	-0.076***	-0.075***
	(0.023)	(0.023)	(0.053)	(0.051)	(0.027)	(0.027)	(0.027)	(0.028)	(0.022)	(0.021)
Peers' average BMI in Wave I	-0.018	-0.022	-0.012	-0.085	-0.010	-0.012	-0.006	-0.008	-0.015	-0.033
	(0.016)	(0.017)	(0.043)	(0.063)	(0.016)	(0.017)	(0.016)	(0.017)	(0.018)	(0.021)
Change in peers' average BMI between waves		-0.013		-0.182*		-0.008		-0.009		-0.052*
		(0.018)		(0.093)		(0.018)		(0.019)		(0.028)
Age	0.006	0.007	0.170	0.189	-0.021	-0.021	-0.031	-0.031	0.003	0.006
	(0.040)	(0.040)	(0.126)	(0.129)	(0.039)	(0.039)	(0.039)	(0.039)	(0.043)	(0.043)
Male (1/0)	0.198**	0.203**	0.101	0.123	0.263***	0.267***	0.277***	0.281***	0.223**	0.242**
	(0.101)	(0.101)	(0.346)	(0.334)	(0.100)	(0.100)	(0.101)	(0.101)	(0.113)	(0.111)
White (1/0)	-0.017	-0.014	-0.183	-0.237	0.098	0.102	0.069	0.072	-0.027	-0.016
	(0.168)	(0.169)	(0.476)	(0.483)	(0.183)	(0.184)	(0.189)	(0.189)	(0.192)	(0.194)
Black (1/0)	-0.009	-0.002	0.079	0.243	-0.024	-0.019	-0.064	-0.060	-0.100	-0.069
	(0.224)	(0.225)	(0.561)	(0.581)	(0.240)	(0.240)	(0.245)	(0.245)	(0.237)	(0.238)
Asian (1/0)	-0.060	-0.058	-1.734**	-1.796**	0.106	0.109	0.100	0.102	-0.162	-0.156
	(0.215)	(0.216)	(0.761)	(0.817)	(0.226)	(0.227)	(0.229)	(0.230)	(0.235)	(0.239)
Native (1/0)	-0.357	-0.354	-0.477	-0.348	-0.266	-0.266	-0.270	-0.270	-0.396	-0.382
	(0.241)	(0.239)	(0.724)	(0.708)	(0.235)	(0.234)	(0.234)	(0.234)	(0.264)	(0.260)
Birth weight (in pound)	-0.031	-0.030	0.008	-0.005	-0.006	-0.006	-0.013	-0.013	-0.000	0.003
	(0.047)	(0.047)	(0.121)	(0.119)	(0.048)	(0.048)	(0.049)	(0.049)	(0.047)	(0.047)
Breastfed (1/0)	-0.005	-0.004	0.131**	0.121*	-0.024	-0.023	-0.021	-0.021	0.014	0.015
	(0.021)	(0.021)	(0.063)	(0.064)	(0.021)	(0.021)	(0.021)	(0.021)	(0.022)	(0.022)
Biological mother obese (1/0)	0.210	0.212	0.238	0.225	0.164	0.166	0.168	0.169	0.197	0.201
	(0.165)	(0.166)	(0.440)	(0.426)	(0.171)	(0.171)	(0.171)	(0.172)	(0.185)	(0.184)
Biological father obese (1/0)	0.471***	0.470***	0.160	0.025	0.356**	0.357**	0.362**	0.364**	0.300	0.288
	(0.170)	(0.170)	(0.544)	(0.540)	(0.171)	(0.170)	(0.170)	(0.170)	(0.227)	(0.225)
Male primary caregiver (PCG) (1/0)	0.154	0.157	-0.557	-0.539	0.288	0.290	0.198	0.200	0.017	0.032
	(0.206)	(0.206)	(0.366)	(0.378)	(0.226)	(0.226)	(0.220)	(0.220)	(0.187)	(0.188)
PCG's age	-0.003	-0.003	0.017	0.012	0.002	0.002	0.002	0.002	0.006	0.005
	(0.009)	(0.009)	(0.025)	(0.023)	(0.009)	(0.009)	(0.009)	(0.009)	(0.010)	(0.010)
PCG married (1/0)	-0.154	-0.158	0.080	0.069	-0.187	-0.189	-0.224*	-0.226*	-0.198	-0.205
	(0.133)	(0.133)	(0.400)	(0.402)	(0.132)	(0.132)	(0.134)	(0.134)	(0.142)	(0.142)
PCG having a college degree (1/0)	-0.042	-0.048	-0.449	-0.527	-0.020	-0.023	-0.049	-0.053	-0.090	-0.109
	(0.113)	(0.114)	(0.386)	(0.386)	(0.117)	(0.117)	(0.117)	(0.117)	(0.118)	(0.120)
Family income (in thousand dollar) in 1994	-0.001*	-0.001*	0.005	0.006	-0.001***	-0.001***	-0.001**	-0.001**	-0.001	-0.001
	(0.001)	(0.001)	(0.006)	(0.006)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Living in urban area (1/0)	0.070	0.073	0.361	0.292	0.111	0.114	0.133	0.136	0.162	0.169
	(0.114)	(0.114)	(0.367)	(0.362)	(0.106)	(0.106)	(0.107)	(0.107)	(0.120)	(0.120)
Change in allowance (dollar/week)	-0.002	-0.002	-0.017	-0.018	-0.003	-0.003	-0.003	-0.003	-0.008	-0.008
	(0.005)	(0.005)	(0.012)	(0.012)	(0.006)	(0.006)	(0.006)	(0.006)	(0.005)	(0.005)
Constant	3.369***	3.430***	-2.097	-0.475	3.715***	3.747***	3.778***	3.815***	2.128***	2.428***
	(0.783)	(0.800)	(2.202)	(2.402)	(0.758)	(0.767)	(0.776)	(0.786)	(0.824)	(0.846)
Observations	1,056	1,056	294	294	890	890	871	871	1,165	1,165

Note: Data are from The National Longitudinal Study of Adolescent Health (Add Health) in-home interviews which took place in Wave I (1994-1995) and Wave II (1996). Parental survey was conducted only in Wave I. Definitions of variables are given in the summary statistics table. Variables with a 1/0 value is binary. For each wave, the exposure to obesity is defined by the number of best friends who are nominated by the adolescent and who are classified as being obese. The treatment group is the one that each adolescent in the group experienced a decrease in the exposure to obesity between the two survey waves. The control group is the one that each adolescent in the group experienced no change (neither an increase nor a decrease) in the exposure to obesity between the two survey periods. The dependent variable is the change in self's BMI between the two waves. Estimates reported in columns (1) and (2) are based on the adolescents who were classified as not being obese in Wave I. Estimates reported in columns (3) and (4) are based on the adolescents who were classified as being either overweight (but not obese) or obese in Wave I. Estimates reported in columns (5) and (6) are based on the adolescents who were classified as being neither overweight (but not obese) nor obese in Wave I. Estimates reported in columns (7) and (8) are based on the adolescents who were classified as having optimal weight in Wave I. Estimates reported in columns (9) and (10) are based on the adolescents who were classified as not being underweight in Wave I. Heteroskedasticity-robust standard errors are in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

Table 8: Impacts of a Perceivable Increase in the Exposure to Obesity on Weight Gains:
Average BMI of Each Adolescent's Best Friends in Wave I Classified as Having Optimal Weight

Dependnet Variable: change in self's BMI	(1)	(2)	(3)	(4)	(5)
Increased exposure to obesity (treatment)	0.390	0.544*	0.753**	0.557*	0.769*
	(0.299)	(0.300)	(0.339)	(0.336)	(0.403)
Self's BMI in Wave I		-0.059***	-0.058***	-0.076***	-0.075***
		(0.015)	(0.016)	(0.021)	(0.021)
Peers' average BMI in Wave I			-0.003	-0.015	-0.033
			(0.029)	(0.032)	(0.036)
Change in peers' average BMI between waves			-0.024		-0.029
			(0.026)		(0.034)
Age				0.035	0.039
				(0.048)	(0.047)
Male (1/0)				0.225*	0.233*
				(0.122)	(0.123)
White (1/0)				-0.113	-0.109
				(0.216)	(0.216)
Black (1/0)				-0.098	-0.086
				(0.264)	(0.263)
Asian (1/0)				-0.062	-0.064
				(0.255)	(0.256)
Native (1/0)				-0.248	-0.231
				(0.279)	(0.280)
Birth weight (in pound)				-0.007	-0.005
				(0.049)	(0.049)
Breastfed (1/0)				-0.009	-0.008
				(0.024)	(0.024)
Biological mother obese (1/0)				0.148	0.145
				(0.196)	(0.196)
Biological father obese (1/0)				0.565***	0.561***
				(0.217)	(0.217)
Male primary caregiver (PCG) (1/0)				0.139	0.150
				(0.249)	(0.248)
PCG's age				-0.004	-0.004
				(0.010)	(0.010)
PCG married (1/0)				-0.083	-0.091
				(0.151)	(0.151)
PCG having a college degree (1/0)				-0.136	-0.145
				(0.138)	(0.139)
Family income (in thousand dollar) in 1994				-0.000	-0.000
				(0.001)	(0.001)
Living in urban area (1/0)				0.046	0.048
				(0.136)	(0.136)
Change in allowance (dollar/week)				-0.002	-0.002
				(0.005)	(0.005)
Constant	0.434***	1.722***	1.776***	2.146**	2.398**
	(0.048)	(0.318)	(0.600)	(0.889)	(0.960)
Observations	1,353	1,353	1,353	892	892

Note: Data are from The National Longitudinal Study of Adolescent Health (Add Health) in-home interviews which took place in Wave I (1994-1995) and Wave II (1996). Parental survey was conducted only in Wave I. Definitions of variables are given in the summary statistics table. Variables with a 1/0 value is binary. For each wave, the exposure to obesity is defined by the number of best friends who are nominated by the adolescent and who are classified as being obese. The treatment group is the one that each adolescent in the group experienced an increase in the exposure to obesity between the two survey waves. The control group is the one that each adolescent in the group experienced no change (neither an increase nor a decrease) in the exposure to obesity between the two survey periods. The dependent variable is the change in self's BMI between the two waves. In column (1), we include only the treatment dummy variable and an intercept term. In column (2), we add self's BMI in Wave I. In column (3), we add peers' average BMI in Wave I and the change in peers' average BMI between the two waves. In column (4), we include the regressors in column (2) plus peers' average BMI in Wave I and other Wave I covariates and the change in self's allowance between the two waves. In column (5), we include the regressors in column (4) plus the change in peers' average BMI between the two waves. All estimations are based on the subsample in which the average BMI of each adolescent's best friends in Wave I is classified as having optimal weight. Heteroskedasticity-robust standard errors are in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

Table 9: Impacts of a Perceivable Increase in the Exposure to Obesity on Weight Gains:

Average BMI of Each Adolescent's Best Friends in Wave I Classified as Being Either Optimal Or Underweight

Dependnet Variable: change in self's BMI	(1)	(2)	(3)	(4)	(5)
Increased exposure to obesity (treatment)	0.386	0.540*	0.716**	0.564*	0.771*
	(0.294)	(0.295)	(0.330)	(0.335)	(0.394)
Self's BMI in Wave I		-0.061***	-0.060***	-0.078***	-0.076***
		(0.015)	(0.015)	(0.021)	(0.021)
Peers' average BMI in Wave I			-0.012	-0.030	-0.048
			(0.028)	(0.031)	(0.035)
Change in peers' average BMI between waves			-0.021		-0.028
			(0.024)		(0.031)
Age				0.035	0.040
				(0.047)	(0.046)
Male (1/0)				0.222*	0.229*
				(0.121)	(0.121)
White (1/0)				-0.093	-0.089
				(0.215)	(0.215)
Black (1/0)				-0.116	-0.102
				(0.261)	(0.261)
Asian (1/0)				-0.082	-0.084
				(0.252)	(0.253)
Native (1/0)				-0.284	-0.268
				(0.272)	(0.272)
Birth weight (in pound)				-0.001	0.002
				(0.048)	(0.048)
Breastfed (1/0)				-0.006	-0.006
				(0.024)	(0.023)
Biological mother obese (1/0)				0.123	0.119
				(0.194)	(0.193)
Biological father obese (1/0)				0.540**	0.536**
				(0.215)	(0.215)
Male primary caregiver (PCG) (1/0)				0.144	0.161
				(0.240)	(0.241)
PCG's age				-0.001	-0.001
				(0.010)	(0.010)
PCG married (1/0)				-0.067	-0.074
				(0.149)	(0.149)
PCG having a college degree (1/0)				-0.144	-0.153
				(0.135)	(0.137)
Family income (in thousand dollar) in 1994				-0.001	-0.000
				(0.001)	(0.001)
Living in urban area (1/0)				0.045	0.044
				(0.133)	(0.133)
Change in allowance (dollar/week)				-0.001	-0.001
				(0.005)	(0.005)
Constant	0.446***	1.783***	2.005***	2.323***	2.579***
	(0.047)	(0.312)	(0.574)	(0.860)	(0.930)
Observations	1,392	1,392	1,392	915	915

Note: Data are from The National Longitudinal Study of Adolescent Health (Add Health) in-home interviews which took place in Wave I (1994-1995) and Wave II (1996). Parental survey was conducted only in Wave I. Definitions of variables are given in the summary statistics table. Variables with a 1/0 value is binary. For each wave, the exposure to obesity is defined by the number of best friends who are nominated by the adolescent and who are classified as being obese. The treatment group is the one that each adolescent in the group experienced an increase in the exposure to obesity between the two survey waves. The control group is the one that each adolescent in the group experienced no change (neither an increase nor a decrease) in the exposure to obesity between the two survey periods. The dependent variable is the change in self's BMI between the two waves. In column (1), we include only the treatment dummy variable and an intercept term. In column (2), we add self's BMI in Wave I. In column (3), we add peers' average BMI in Wave I and the change in peers' average BMI between the two waves. In column (4), we include the regressors in column (2) plus peers' average BMI in Wave I and other Wave I covariates and the change in self's allowance between the two waves. In column (5), we include the regressors in column (4) plus the change in peers' average BMI between the two waves. All estimations are based on the subsample in which the average BMI of each adolescent's best friends in Wave I is classified as being either optimal or underweight. Heteroskedasticity-robust standard errors are in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

Table 10: Impacts of a Possibly Unperceivable Increase in the Exposure to Obesity on Weight Gains:
Average BMI of Each Adolescent's Best Friends in Wave I Classified as Being Either Obese Or Overweight

Dependnet Variable: change in self's BMI	(1)	(2)	(3)	(4)	(5)
Increased exposure to obesity (treatment)	0.796	0.812	0.656	0.460	0.560
	(0.696)	(0.704)	(0.728)	(1.016)	(1.040)
Self's BMI in Wave I		-0.025	-0.024	-0.046	-0.048
		(0.034)	(0.038)	(0.055)	(0.057)
Peers' average BMI in Wave I			0.006	-0.051	-0.050
			(0.053)	(0.052)	(0.052)
Change in peers' average BMI between waves			0.030		-0.019
			(0.039)		(0.052)
Age				-0.015	-0.014
				(0.140)	(0.140)
Male (1/0)				0.351	0.359
				(0.375)	(0.377)
White (1/0)				0.689	0.688
				(0.801)	(0.803)
Black (1/0)				0.481	0.493
				(0.989)	(0.992)
Asian (1/0)				0.357	0.370
				(0.957)	(0.965)
Native (1/0)				-0.721	-0.711
				(0.712)	(0.711)
Birth weight (in pound)				0.049	0.046
				(0.172)	(0.173)
Breastfed (1/0)				0.071	0.067
				(0.085)	(0.087)
Biological mother obese (1/0)				0.546	0.538
				(0.507)	(0.505)
Biological father obese (1/0)				-0.076	-0.093
				(0.574)	(0.578)
Male primary caregiver (PCG) (1/0)				0.191	0.204
				(0.528)	(0.525)
PCG's age				0.017	0.017
				(0.028)	(0.028)
PCG married (1/0)				-0.394	-0.394
				(0.477)	(0.478)
PCG having a college degree (1/0)				-0.037	-0.060
				(0.326)	(0.333)
Family income (in thousand dollar) in 1994				-0.010**	-0.010**
				(0.005)	(0.005)
Living in urban area (1/0)				0.658	0.659
				(0.404)	(0.406)
Change in allowance (dollar/week)				-0.018	-0.018
				(0.016)	(0.017)
Constant	0.606***	1.183	1.048	1.336	1.380
	(0.145)	(0.753)	(1.258)	(3.305)	(3.308)
Observations	283	283	283	182	182

Note: Data are from The National Longitudinal Study of Adolescent Health (Add Health) in-home interviews which took place in Wave I (1994-1995) and Wave II (1996). Parental survey was conducted only in Wave I. Definitions of variables are given in the summary statistics table. Variables with a 1/0 value is binary. For each wave, the exposure to obesity is defined by the number of best friends who are nominated by the adolescent and who are classified as being obese. The treatment group is the one that each adolescent in the group experienced an increase in the exposure to obesity between the two survey waves. The control group is the one that each adolescent in the group experienced no change (neither an increase nor a decrease) in the exposure to obesity between the two survey periods. The dependent variable is the change in self's BMI between the two waves. In column (1), we include only the treatment dummy variable and an intercept term. In column (2), we add self's BMI in Wave I. In column (3), we add peers' average BMI in Wave I and the change in peers' average BMI between the two waves. In column (4), we include the regressors in column (2) plus peers' average BMI in Wave I and other Wave I covariates and the change in self's allowance between the two waves. In column (5), we include the regressors in column (4) plus the change in peers' average BMI between the two waves. All estimations are based on the subsample in which the average BMI of each adolescent's best friends in Wave I is classified as being either obese or overweight. Heteroskedasticity-robust standard errors are in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

Table 11: Impacts of an Increase in the Exposure to the Underweight on Weight Gains

Dependnet Variable: change in self's BMI	(1)	(2)	(3)	(4)	(5)
Increased exposure to the underweight (treatment)	0.104	0.065	0.123	0.469	0.388
	(0.327)	(0.336)	(0.346)	(0.438)	(0.452)
Self's BMI in Wave I		-0.049***	-0.053***	-0.076***	-0.076***
		(0.014)	(0.015)	(0.020)	(0.020)
Peers' average BMI in Wave I			0.024	-0.005	-0.012
			(0.018)	(0.018)	(0.021)
Change in peers' average BMI between waves			0.006		-0.016
			(0.019)		(0.025)
Age				-0.006	-0.004
				(0.044)	(0.045)
Male (1/0)				0.201*	0.205*
				(0.113)	(0.112)
White (1/0)				-0.034	-0.031
				(0.187)	(0.187)
Black (1/0)				0.133	0.143
				(0.237)	(0.236)
Asian (1/0)				-0.095	-0.090
				(0.230)	(0.231)
Native (1/0)				-0.376	-0.370
				(0.260)	(0.261)
Birth weight (in pound)				-0.025	-0.024
				(0.047)	(0.047)
Breastfed (1/0)				0.008	0.008
				(0.022)	(0.022)
Biological mother obese (1/0)				0.242	0.247
				(0.178)	(0.179)
Biological father obese (1/0)				0.374*	0.377*
				(0.222)	(0.221)
Male primary caregiver (PCG) (1/0)				0.111	0.110
				(0.199)	(0.199)
PCG's age				0.002	0.001
				(0.010)	(0.010)
PCG married (1/0)				-0.182	-0.188
				(0.143)	(0.143)
PCG having a college degree (1/0)				-0.144	-0.148
				(0.119)	(0.118)
Family income (in thousand dollar) in 1994				-0.001	-0.001
				(0.001)	(0.001)
Living in urban area (1/0)				0.170	0.170
				(0.123)	(0.123)
Change in allowance (dollar/week)				-0.006	-0.006
				(0.005)	(0.005)
Constant	0.484***	1.592***	1.149**	2.428***	2.541***
	(0.045)	(0.307)	(0.453)	(0.855)	(0.845)
Observations	1,885	1,885	1,885	1,223	1,223

Note: Data are from The National Longitudinal Study of Adolescent Health (Add Health) in-home interviews which took place in Wave I (1994-1995) and Wave II (1996). Parental survey was conducted only in Wave I. Definitions of variables are given in the summary statistics table. Variables with a 1/0 value is binary. For each wave, the exposure to the underweight is defined by the number of best friends who are nominated by the adolescent and who are classified as being underweight. The treatment group is the one that each adolescent in the group experienced an increase in the exposure to the underweight between the two survey waves. The control group is the one that each adolescent in the group experienced no change (neither an increase nor a decrease) in the exposure to the underweight between the two survey periods. The dependent variable is the change in self's BMI between the two waves. In column (1), we include only the treatment dummy variable and an intercept term. In column (2), we add self's BMI in Wave I. In column (3), we add peers' average BMI in Wave I and the change in peers' average BMI between the two waves. In column (4), we include the regressors in column (2) plus peers' average BMI in Wave I and other Wave I covariates and the change in self's allowance between the two waves. In column (5), we include the regressors in column (4) plus the change in peers' average BMI between the two waves. Heteroskedasticity-robust standard errors are in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

Table 12: Impacts of a Decrease in the Exposure to the Underweight on Weight Gains

Dependnet Variable: change in self's BMI	(1)	(2)	(3)	(4)	(5)
Decreased exposure to the underweight (treatment)	0.378	0.286	0.405	0.270	0.294
	(0.243)	(0.230)	(0.246)	(0.289)	(0.289)
Self's BMI in Wave I		-0.054***	-0.059***	-0.079***	-0.079***
		(0.014)	(0.015)	(0.020)	(0.019)
Peers' average BMI in Wave I			0.027	-0.004	-0.012
			(0.018)	(0.018)	(0.020)
Change in peers' average BMI between waves			0.003		-0.018
			(0.019)		(0.025)
Age				0.001	0.004
				(0.043)	(0.044)
Male (1/0)				0.186*	0.190*
				(0.112)	(0.112)
White (1/0)				-0.027	-0.025
				(0.187)	(0.187)
Black (1/0)				0.138	0.149
				(0.239)	(0.238)
Asian (1/0)				-0.127	-0.122
				(0.229)	(0.230)
Native (1/0)				-0.397	-0.391
				(0.256)	(0.257)
Birth weight (in pound)				-0.025	-0.024
				(0.047)	(0.047)
Breastfed (1/0)				0.006	0.006
				(0.022)	(0.022)
Biological mother obese (1/0)				0.250	0.254
				(0.177)	(0.177)
Biological father obese (1/0)				0.360	0.364*
				(0.222)	(0.221)
Male primary caregiver (PCG) (1/0)				0.137	0.141
				(0.194)	(0.195)
PCG's age				0.003	0.003
				(0.010)	(0.010)
PCG married (1/0)				-0.146	-0.153
				(0.143)	(0.143)
PCG having a college degree (1/0)				-0.123	-0.128
				(0.118)	(0.118)
Family income (in thousand dollar) in 1994				-0.001	-0.001
				(0.001)	(0.001)
Living in urban area (1/0)				0.188	0.187
				(0.122)	(0.122)
Change in allowance (dollar/week)				-0.005	-0.005
				(0.005)	(0.005)
Constant	0.484***	1.705***	1.218***	2.288***	2.413***
	(0.045)	(0.305)	(0.449)	(0.848)	(0.836)
Observations	1,883	1,883	1,883	1,229	1,229

Note: Data are from The National Longitudinal Study of Adolescent Health (Add Health) in-home interviews which took place in Wave I (1994-1995) and Wave II (1996). Parental survey was conducted only in Wave I. Definitions of variables are given in the summary statistics table. Variables with a 1/0 value is binary. For each wave, the exposure to the underweight is defined by the number of best friends who are nominated by the adolescent and who are classified as being underweight. The treatment group is the one that each adolescent in the group experienced a decrease in the exposure to the underweight between the two survey waves. The control group is the one that each adolescent in the group experienced no change (neither an increase nor a decrease) in the exposure to the underweight between the two survey periods. The dependent variable is the change in self's BMI between the two waves. In column (1), we include only the treatment dummy variable and an intercept term. In column (2), we add self's BMI in Wave I. In column (3), we add peers' average BMI in Wave I and the change in peers' average BMI between the two waves. In column (4), we include the regressors in column (2) plus peers' average BMI in Wave I and other Wave I covariates and the change in self's allowance between the two waves. In column (5), we include the regressors in column (4) plus the change in peers' average BMI between the two waves. Heteroskedasticity-robust standard errors are in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

Table 13: Impacts of an Increase in the Exposure to Obesity on Weight Gains

Alternative Peer Definition: An Adolescent's Same-School and Same-Grade Classmates in Each Wave

Dependnet Variable: change in self's BMI	(1)	(2)	(3)	(4)	(5)
Increased exposure to obesity (treatment)	-0.045	0.039	0.018	0.135	0.114
	(0.091)	(0.090)	(0.093)	(0.134)	(0.126)
Self's BMI in Wave I		-0.067***	-0.066***	-0.078***	-0.078***
		(0.010)	(0.010)	(0.013)	(0.013)
Peers' average BMI in Wave I			-0.010	-0.047	-0.041
			(0.022)	(0.039)	(0.038)
Change in peers' average BMI between waves			0.066**		0.036
			(0.028)		(0.042)
Age				0.008	0.009
				(0.030)	(0.030)
Male (1/0)				0.275***	0.275***
				(0.068)	(0.068)
White (1/0)				0.065	0.066
				(0.124)	(0.124)
Black (1/0)				0.020	0.021
				(0.141)	(0.141)
Asian (1/0)				-0.093	-0.091
				(0.151)	(0.151)
Native (1/0)				0.118	0.111
				(0.217)	(0.217)
Birth weight (in pound)				0.001	0.001
				(0.028)	(0.028)
Breastfed (1/0)				0.010	0.010
				(0.014)	(0.014)
Biological mother obese (1/0)				0.315***	0.315***
				(0.100)	(0.100)
Biological father obese (1/0)				0.160	0.159
				(0.128)	(0.128)
Male primary caregiver (PCG) (1/0)				-0.066	-0.061
				(0.123)	(0.123)
PCG's age				-0.006	-0.005
				(0.007)	(0.007)
PCG married (1/0)				-0.009	-0.004
				(0.083)	(0.084)
PCG having a college degree (1/0)				-0.044	-0.040
				(0.075)	(0.074)
Family income (in thousand dollar) in 1994				-0.001**	-0.001**
				(0.000)	(0.000)
Living in urban area (1/0)				0.018	0.014
				(0.068)	(0.069)
Change in allowance (dollar/week)				-0.000	-0.000
				(0.003)	(0.003)
Constant	0.547***	1.958***	2.113***	2.977***	2.825***
	(0.087)	(0.216)	(0.482)	(0.712)	(0.692)
Observations	6,875	6,875	6,875	4,195	4,195

Note: Data are from The National Longitudinal Study of Adolescent Health (Add Health) in-home interviews which took place in Wave I (1994-1995) and Wave II (1996). Parental survey was conducted only in Wave I. Definitions of variables are given in the summary statistics table. Variables with a 1/0 value is binary. For each wave, the peers are defined by the adolescent's same-school and same-grade classmates. For each wave, the exposure to obesity is defined by the number of same-school and same-grade classmates who are classified as being obese. The treatment group is the one that each adolescent in the group experienced an increase in the exposure to obesity between the two survey waves. The control group is the one that each adolescent in the group experienced no change (neither an increase nor a decrease) in the exposure to obesity between the two survey periods. The dependent variable is the change in self's BMI between the two waves. In column (1), we include only the treatment dummy variable and an intercept term. In column (2), we add self's BMI in Wave I. In column (3), we add peers' average BMI in Wave I and the change in peers' average BMI between the two waves. In column (4), we include the regressors in column (2) plus peers' average BMI in Wave I and other Wave I covariates and the change in self's allowance between the two waves. In column (5), we include the regressors in column (4) plus the change in peers' average BMI between the two waves. Heteroskedasticity-robust standard errors are in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

Table 14: Impacts of a Decrease in the Exposure to Obesity on Weight Gains

Alternative Peer Definition: An Adolescent's Same-School and Same-Grade Classmates in Each Wave

Dependnet Variable: change in self's BMI	(1)	(2)	(3)	(4)	(5)
Decreased exposure to obesity (treatment)	-0.036	0.057	0.081	0.154	0.159
	(0.091)	(0.090)	(0.098)	(0.137)	(0.135)
Self's BMI in Wave I		-0.070***	-0.069***	-0.077***	-0.077***
		(0.009)	(0.009)	(0.011)	(0.011)
Peers' average BMI in Wave I			-0.025	-0.031	-0.045
			(0.023)	(0.033)	(0.033)
Change in peers' average BMI between waves			-0.039		-0.043
			(0.035)		(0.049)
Age				0.007	0.010
				(0.027)	(0.026)
Male (1/0)				0.079	0.078
				(0.062)	(0.062)
White (1/0)				-0.016	-0.019
				(0.115)	(0.115)
Black (1/0)				0.213	0.211
				(0.132)	(0.132)
Asian (1/0)				-0.076	-0.075
				(0.141)	(0.141)
Native (1/0)				0.054	0.053
				(0.186)	(0.186)
Birth weight (in pound)				0.019	0.019
				(0.027)	(0.027)
Breastfed (1/0)				0.005	0.004
				(0.012)	(0.012)
Biological mother obese (1/0)				0.420***	0.420***
				(0.097)	(0.097)
Biological father obese (1/0)				0.151	0.149
				(0.123)	(0.123)
Male primary caregiver (PCG) (1/0)				0.096	0.101
				(0.141)	(0.140)
PCG's age				-0.011**	-0.011**
				(0.006)	(0.006)
PCG married (1/0)				0.032	0.033
				(0.077)	(0.077)
PCG having a college degree (1/0)				-0.026	-0.026
				(0.072)	(0.072)
Family income (in thousand dollar) in 1994				-0.000	-0.000
				(0.000)	(0.000)
Living in urban area (1/0)				0.012	0.015
				(0.065)	(0.065)
Change in allowance (dollar/week)				-0.001	-0.001
				(0.003)	(0.003)
Constant	0.547***	2.023***	2.561***	2.788***	3.046***
	(0.087)	(0.199)	(0.477)	(0.644)	(0.652)
Observations	7,065	7,065	7,065	4,230	4,230

Note: Data are from The National Longitudinal Study of Adolescent Health (Add Health) in-home interviews which took place in Wave I (1994-1995) and Wave II (1996). Parental survey was conducted only in Wave I. Definitions of variables are given in the summary statistics table. Variables with a 1/0 value is binary. For each wave, the peers are defined by the adolescent's same-school and same-grade classmates. For each wave, the exposure to obesity is defined by the number of same-school and same-grade classmates who are classified as being obese. The treatment group is the one that each adolescent in the group experienced a decrease in the exposure to obesity between the two survey waves. The control group is the one that each adolescent in the group experienced no change (neither an increase nor a decrease) in the exposure to obesity between the two survey periods. The dependent variable is the change in self's BMI between the two waves. In column (1), we include only the treatment dummy variable and an intercept term. In column (2), we add self's BMI in Wave I. In column (3), we add peers' average BMI in Wave I and the change in peers' average BMI between the two waves. In column (4), we include the regressors in column (2) plus peers' average BMI in Wave I and other Wave I covariates and the change in self's allowance between the two waves. In column (5), we include the regressors in column (4) plus the change in peers' average BMI between the two waves. Heteroskedasticity-robust standard errors are in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

Table 15: Difference-in-Differences (DID) and Changes-in-Changes (CIC) Estimates

Dependent Variable: BMI	Average treatment effect on the treated		Average treatment effect on the untreated	
	DID estimate	CIC estimate	DID estimate	CIC estimate
Increased exposure to obesity	0.488*	0.562*	N/A	0.504*
	(0.280)	[0.311]	N/A	[0.284]
Number of observations	1,816	1,816	N/A	1,816

Note: Data are from The National Longitudinal Study of Adolescent Health (Add Health) in-home interviews which took place in Wave I (1994-1995, T = 0) and Wave II (1996, T = 1). For each wave, the exposure to obesity is defined by the number of best friends who are nominated by the adolescent and who are classified as being obese. The treatment group (G = 1) is the one that each adolescent in the group experienced an increase in the exposure to obesity between the two survey waves. The control group (G = 0) is the one that each adolescent in the group experienced no change (neither an increase nor a decrease) in the exposure to obesity between the two survey periods. The dependent variable is BMI for all estimations. For the difference-in-differences (DID) estimation, we include the treatment dummy variable (GT), the time period indicator (T), the intercept term, and individual level fixed effects. For the changes-in-changes (CIC) estimation, we follow the procedure proposed by Athey and Imbens (2006). We first use linear regression of BMI on four group-time dummy variables ((1-T)(1-G), T(1-G), (1-T)G, TG), without an intercept term. We then apply the CIC estimator (Athey and Imbens, 2006) to the residuals from the ordinary least squares regression adding back the effects of the dummy variables. Heteroskedasticity-robust standard errors are in parentheses. Block-bootstrapped standard errors in the panel setting based on 1,000 replications are in brackets. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

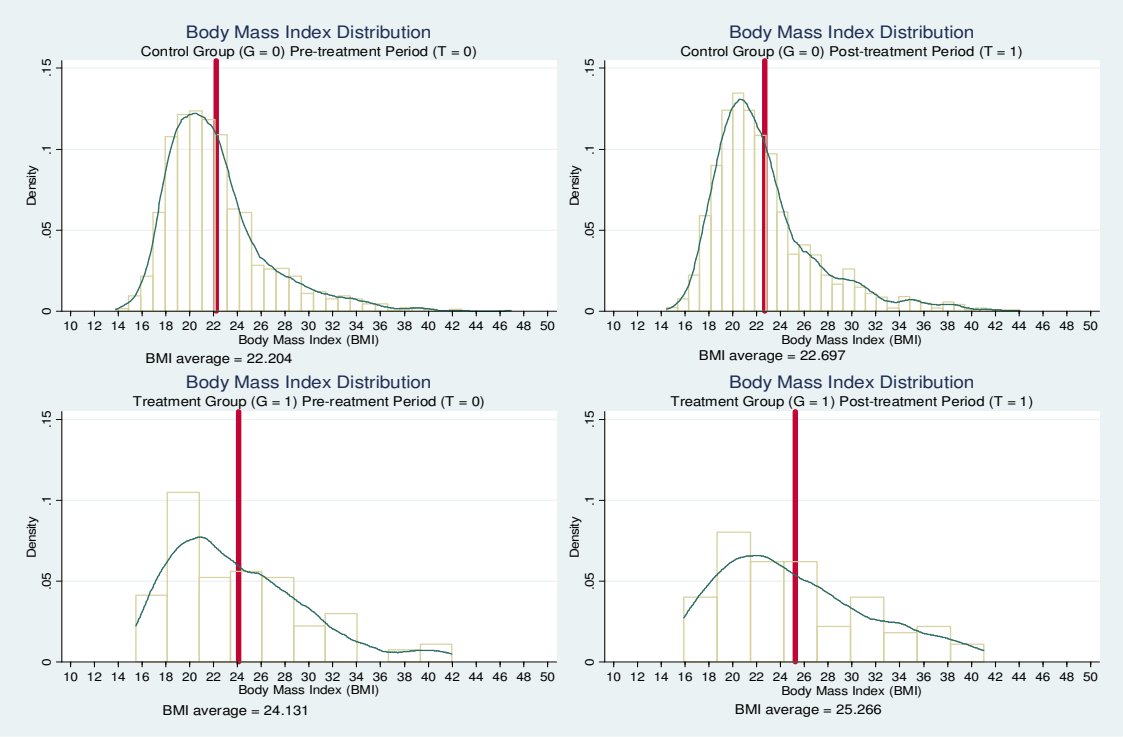

Figure 1: Body Mass Index Distribution (Histogram) by Group and Wave

Note: The smooth curve in each panel is the estimated BMI density function, which is estimated based on the epanechnikov kernel function and the optimal bandwidth. The vertical solid line in each panel corresponds to the average BMI.

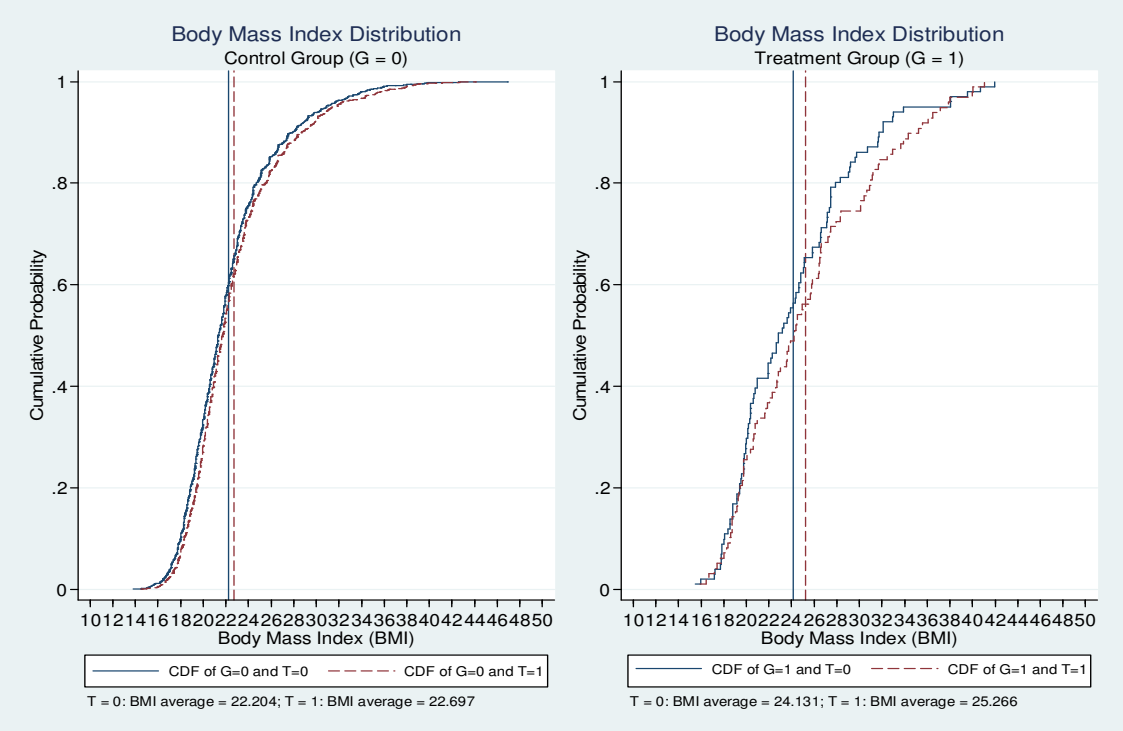

Figure 2: Body Mass Index Distribution (CDF) by Group and Wave

Note: The vertical solid line in each panel correspond to the average BMI in Wave I (T = 0). The vertical dashed line in each panel corresponds to the average BMI in Wave II (T = 1).

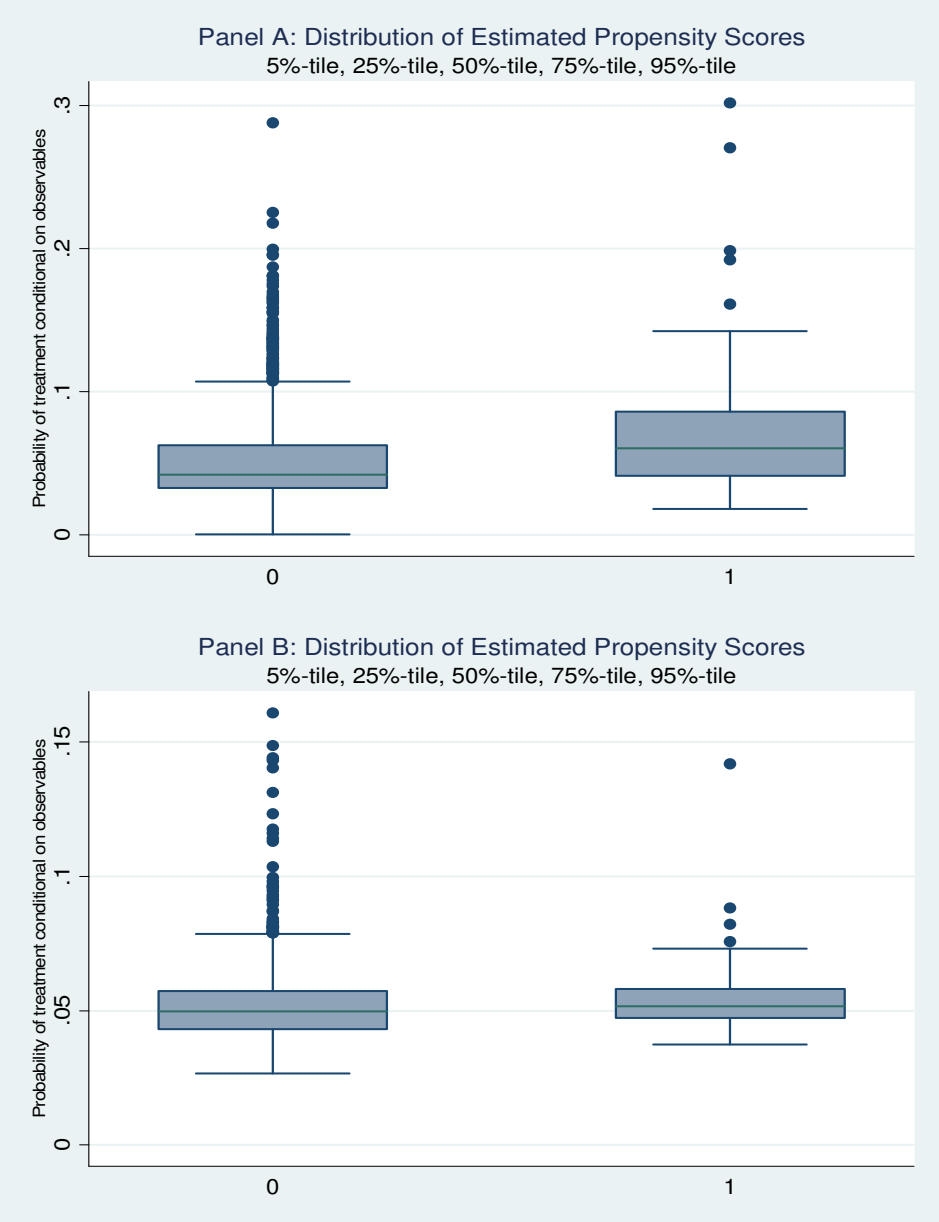

Figure 3: Estimated Propensity Scores Based on Wave I By Treatment Status (in Wave II)

Note: The above box-plot reveals how much overlap of the observable characteristics that the treatment group (labeled "1", in which the treatment occurred between the two survey waves) and the control group (labeled "0", in which the treatment never occurred in either survey wave) share using the estimated propensity scores. The box ends at the quartiles (25%-tile and 75%-tile), extending the "whiskers" to the farthest points that are not outliers (5%-tile and 95%-tile). The statistical median is a horizontal line in the box. The overlap in the box plots implies the similarities of the observable characteristics between the treatment group and the control group in Wave I. In panel A, the following observable characteristics from Add Health Wave I are included in estimating the propensity score (the probability of treatment conditional on observables): age, gender (whether male or not), race (White, Black, Asian, or Native), birth weight, whether breastfed or not, whether biological mother obese, whether biological father obese, primary caregiver (PCG)'s gender (whether male or not), PCG's age, whether PCG married or not, whether PCG having a college degree or not, family income in 1994, whether living in urban area or not. In panel B, the same observable characteristics used in Panel A are included in estimating the propensity score, except for the following variables: race (White, Black, Asian, or Native), whether biological mother obese or not, whether biological father obese or not, and family income in 1994.

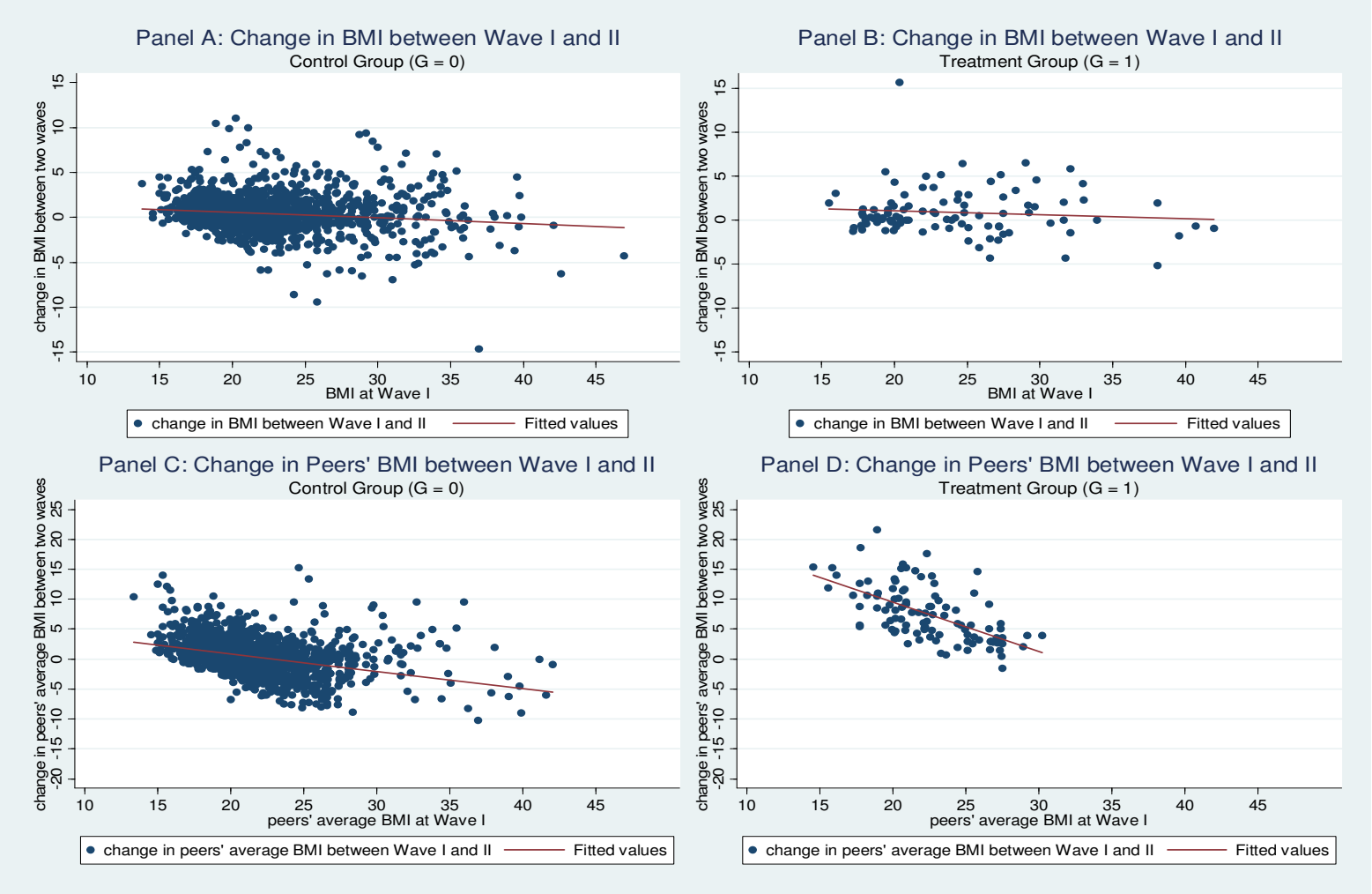

Figure 4: Body Mass Index (BMI) in Wave I and the Change in BMI between Wave I and Wave II

Note: Panel A suggests the mean-reversion pattern of self's Body Mass Index (BMI) for the control group (G = 0). Panel B suggests the mean-reversion pattern of self's BMI for the treatment group (G = 1). In both Panels A and B, the dependent variable is the change in self's BMI between the two survey waves, and the independent variable is self's BMI in Wave I. Panel C suggests the mean-reversion pattern of an adolescent's peers' average BMI for the control group (G = 0). Panel D suggests the mean-reversion pattern of an adolescent's peers' average BMI for the treatment group (G = 1). In both Panels C and D, the dependent variable is the change in an adolescent's peers' average BMI between the two survey waves, and the independent variable is an adolescent's peers' average BMI in Wave I. The fitted lines in all four panels are based on the best linear predictor.

Appendix Table 1: Obesity Peer Effect Estimates Based On Specifications of Previous Studies

Dependent variable:	Body Mass Index		Obesity Status (1/0)	
Variables	(1)	(2)	(3)	(4)
	CF (2007)	CCF (2008)	CF (2007)	CCF (2008)
Peers' average BMI	-0.021	-0.023		
	(0.024)	(0.025)		
Self's BMI in Wave I	0.923***	0.925***		
	(0.019)	(0.020)		
Peers' average BMI in Wave I	0.005	0.002		
	(0.022)	(0.024)		
Peers' average obesity status			0.013	0.009
			(0.041)	(0.042)
Self's obesity status in Wave I			0.713***	0.714***
			(0.037)	(0.038)
Peers' average obesity status in Wave I			0.004	0.009
			(0.028)	(0.031)
Age	0.000	0.050	0.001	0.003
	(0.043)	(0.053)	(0.005)	(0.006)
Male (1/0)	0.184*	0.145	0.019	0.019
	(0.110)	(0.119)	(0.012)	(0.013)
White (1/0)	-0.004	-0.317	0.011	-0.013
	(0.185)	(0.239)	(0.022)	(0.026)
Black (1/0)	0.139	0.170	0.029	0.021
	(0.236)	(0.289)	(0.028)	(0.033)
Asian (1/0)	-0.101	-0.206	0.016	0.019
	(0.224)	(0.251)	(0.026)	(0.027)
Native (1/0)	-0.310	-0.252	-0.015	0.001
	(0.263)	(0.304)	(0.031)	(0.023)
Birth weight (in pound)	-0.018	-0.019	0.006	0.003
	(0.047)	(0.047)	(0.005)	(0.005)
Breastfed (1/0)	-0.077	-0.008	-0.027**	-0.017
	(0.110)	(0.117)	(0.012)	(0.012)
Biological mother obese (1/0)	0.250	0.269	0.032	0.039*
	(0.176)	(0.185)	(0.020)	(0.021)
Biological father obese (1/0)	0.371*	0.396*	0.019	0.021
	(0.216)	(0.220)	(0.025)	(0.026)
Male primary caregiver (PCG) (1/0)	0.124	0.158	-0.025	-0.012
	(0.192)	(0.203)	(0.028)	(0.026)
PCG's age	0.003	0.004	0.000	0.000
	(0.009)	(0.011)	(0.001)	(0.001)
PCG married (1/0)	-0.165	-0.124	-0.017	-0.008
	(0.141)	(0.149)	(0.016)	(0.017)
PCG having a college degree (1/0)	-0.137	-0.034	0.001	0.013
	(0.119)	(0.128)	(0.013)	(0.014)
Family income (in thousand dollar) in 1994	-0.001	-0.000	-0.000	-0.000
	(0.001)	(0.001)	(0.000)	(0.000)
Living in urban area (1/0)	0.176	0.347	0.000	0.014
	(0.121)	(0.265)	(0.014)	(0.021)
Allowance (dollar/week)	-0.002	-0.004	0.000	-0.000
	(0.005)	(0.006)	(0.001)	(0.001)
Constant	2.580***	1.841*	-0.043	-0.050
	(0.828)	(1.060)	(0.085)	(0.097)
Observations	1,253	1,253	1,252	1,252

Note: Data are from The National Longitudinal Study of Adolescent Health (Add Health) in-home interviews which took place in Wave I (1994-1995) and Wave II (1996). Parental survey was conducted only in Wave I. Definitions of variables are given in the summary statistics table. Variables with a 1/0 value is binary. In both survey waves, a student (in grade 7-12) was asked to nominate up to one best male friend and one best female friend. The number of nominated best friend is between one and two. For each wave, the peers are defined by the nominated best friends. In columns (1) and (2), the dependent variable is the Body Mass Index (a continuous variable). In columns (3) and (4), the dependent variable is the obesity status (a binary dummy variable). Estimates reported in columns (1) and (3) are based on the specification used by Christakis and Fowler (CF, 2007). Estimates reported in columns (2) and (4) are based on the specification used by Cohen-Cole and Fletcher (CCF, 2008). Heteroskedasticity-robust standard errors are in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

Appendix Table 2: Obesity Peer Effect Estimates Based On Specifications of Halliday and Kwak (HK, 2009)

Dependent variable:	Body Mass Index		Obesity Status (1/0)	
Variables	Fixed Effect Controlled at		Fixed Effect Controlled at	
	(1)	(2)	(3)	(4)
	School Level	Individual Level	School Level	Individual Level
Peers' average BMI	0.131***			
	(0.018)			
Change in peers' average BMI between waves		-0.014		
		(0.021)		
Peers' average obesity status			0.098***	
			(0.021)	
Change in peers' average obesity status between waves				0.001
				(0.033)
Age	0.422***	-0.044	-0.005	0.004
	(0.050)	(0.041)	(0.004)	(0.005)
Male (1/0)	0.506***	0.148	0.042***	0.007
	(0.122)	(0.112)	(0.009)	(0.014)
White (1/0)	-0.722**	0.085	-0.026	0.030
	(0.284)	(0.180)	(0.021)	(0.025)
Black (1/0)	-0.256	0.151	-0.024	0.043
	(0.310)	(0.233)	(0.023)	(0.031)
Asian (1/0)	-1.290***	0.015	-0.025	0.032
	(0.305)	(0.228)	(0.022)	(0.029)
Native (1/0)	-0.197	-0.452*	0.003	-0.044
	(0.343)	(0.251)	(0.027)	(0.034)
Birth weight (in pound)	0.155***	-0.036	0.007*	0.004
	(0.053)	(0.046)	(0.004)	(0.006)
Breastfed (1/0)	-0.297**	-0.057	-0.015	-0.024*
	(0.123)	(0.112)	(0.009)	(0.013)
Biological mother obese (1/0)	1.901***	0.089	0.116***	-0.000
	(0.196)	(0.172)	(0.015)	(0.022)
Biological father obese (1/0)	2.044***	0.182	0.124***	-0.020
	(0.242)	(0.235)	(0.019)	(0.027)
Male primary caregiver (PCG) (1/0)	0.561*	0.115	0.009	-0.029
	(0.318)	(0.198)	(0.021)	(0.033)
PCG's age	0.008	0.003	0.001	-0.000
	(0.012)	(0.010)	(0.001)	(0.001)
PCG married (1/0)	-0.272*	-0.143	-0.015	-0.012
	(0.156)	(0.142)	(0.012)	(0.017)
PCG having a college degree (1/0)	-0.211	-0.088	-0.006	0.005
	(0.146)	(0.122)	(0.011)	(0.015)
Family income (in thousand dollar) in 1994	-0.003***	-0.000	-0.000**	-0.000
	(0.001)	(0.001)	(0.000)	(0.000)
Living in urban area (1/0)	-0.039	0.218*	0.002	0.010
	(0.274)	(0.123)	(0.020)	(0.015)
Allowance (dollar/week)	0.013**		0.000	
	(0.006)		(0.000)	
Change in allowance (dollar/week)		-0.008		-0.000
		(0.006)		(0.001)
Constant	11.872***	1.256	0.080	-0.100
	(1.070)	(0.821)	(0.074)	(0.097)
Observations	5,069	1,249	5,067	1,248

Note: Data are from The National Longitudinal Study of Adolescent Health (Add Health) in-home interviews which took place in Wave I (1994-1995) and Wave II (1996). Parental survey was conducted only in Wave I. Definitions of variables are given in the summary statistics table. Variables with a 1/0 value is binary. In both survey waves, a student (in grade 7-12) was asked to nominate up to one best male friend and one best female friend. The number of nominated best friend is between one and two. For each wave, the peers are defined by the nominated best friends. In columns (1) and (2), the dependent variable is the Body Mass Index (a continuous variable). In columns (3) and (4), the dependent variable is the obesity status (a binary dummy variable). Estimates reported in columns (1) and (3) are based on the specification used by Halliday and Kwak (HK, 2009), controlling for school-level fixed effect only. Estimates reported in columns (2) and (4) are based on the specification used by HK (2009), controlling for individual-level fixed effect. Heteroskedasticity-robust standard errors are in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

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