The Effect of Bodyweight on Adolescent Sexual Activity*

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Abstract

Recent research suggests that overweight females suffer penalties in the labor and marriage markets, while overweight males do not. This study explores whether gender differences in the effect of body weight exist in what Cawley et al. (2006) labeled "the adolescent sex market." Drawing on data from the National Longitudinal Study of Adolescent Health, we use individual fixed effects and instrumental variables (IV) identification strategies to estimate the relationship between body weight and sexual activity. We find evidence that increases in body weight lower the probability that female adolescents become sexually active. In contrast, there is little evidence of a causal relationship between body weight and sexual activity for male adolescents.

Keywords: obesity, sex, sexual activity

I. Introduction

A number of studies have found that overweight females face substantial obstacles in the labor, education, and marriage markets (Averett and Korenman 1996; Cawley 2004; Sabia 2007), and are more likely to suffer from depression and low self-esteem than their slimmer counterparts (Franklin et al. 2006; Onyike et al. 2003). In contrast, overweight males do not seem to face the same challenges when looking for a job or a spouse, nor do they seem to suffer to the same degree psychologically.¹ This pattern of results is consistent with the hypothesis that society views overweight males and females very differently.

Recently, Cawley et al. (2006) used data from the National Longitudinal Study of Adolescent Health (Add Health) to examine the effect of body weight on the decision to become sexually active. They found that being overweight or obese was negatively related to the likelihood of having sex for the first time. Interestingly, this relationship was almost as large for males as for females. Specifically, Cawley et al. (2006) found that overweight (obese) females were 60% (32%) as likely to become sexually active as healthy-weight females, while overweight (obese) males were 76% (45%) as likely to become sexually active as healthy-weight males.

One possible explanation proposed by Cawley et al. (2006) for these results is that overweight males are crowded out of "the adolescent market for sex"(p. 72) and overweight females rationally refrain from sexual activity in an effort to preserve their reputations. Another possibility is that the negative association between body weight and sexual activity documented by Cawley et al. (2006) is spurious, due to difficult-to-measure individual- or family-level confounders. For instance, heavier individuals could be less future oriented or more physically

¹ Onyike et al. 2003 found that the relationship between being obese and depression among men was not statistically significant. Franklin et al. (2006) found that the relationship between being obese and self-esteem was stronger among girls than among boys.

mature than their counterparts in the "recommended" weight range, traits that could also affect the probability of a successful match.²

Using data drawn from Add Health, we estimate the effect of adolescent body weight on sexual activity for a sample of 14 to 17 year-olds. Ordinary Least Squares (OLS) estimates suggest that bodyweight is negatively related to the probability that female adolescents are sexually active. Among males, the relationship appears to be U-shaped: that is, both underweight and overweight males are less likely to be sexually active as compared to their counterparts of normal bodyweight. When we turn to longitudinal data and individual fixed effects are included on the right-hand side of the estimating equation, the negative relationship between bodyweight and sexual activity persists for female adolescents; the U-shaped relationship observed in the cross-section for males, however, appears to be entirely driven by difficult-toobserve factors at the individual level. When mother's obesity status and the Body Mass Index (BMI) of the respondent's sibling are used to instrument for body weight, we find that a 10-pound increase in body weight leads to a 7 percent decrease in the likelihood that female respondents were sexually active. There is no comparable effect for male adolescents. A wide set of robustness checks and falsification tests bolster the case for a causal interpretation of the instrumental variables estimates.

² Alternatively, if becoming sexually active affects either calorie intake or calories burned, then it is possible that the negative relationship is due to reverse causality. There is evidence that becoming sexually active is associated with the symptoms of depression among female adolescents (Sabia and Rees 2008; Hallfors et al. 2004); depression, in turn, may lead to changes in eating or exercise. Lagging body weight should militate against the problem of reverse causality. In fact, Cawley et al. (2006) attempted to address this concern by using lagged measures of bodyweight on the right-hand side of their estimation equation.

II. Background

The relationship between adolescent bodyweight and sexual activity is theoretically ambiguous. In a market in which there is perfect matching, sexual activity will be unrelated to bodyweight because slimmer girls will partner with slimmer boys, and overweight girls will partner with overweight boys. In fact, there is some evidence that sorting of this type takes place (Berscheid et al. 1971; Schafer and Keith 1990; McPherson et al. 2001).

However, it is also possible that some individuals will prefer to remain virgins rather than have intercourse with a partner who is overweight. Cawley et al. (2006) developed a model in which adolescents care about their reputations, and derive more utility from having intercourse with attractive partners than with unattractive partners. Built into the model is the assumption that males see their reputation as enhanced by sexual activity, whereas females may be stigmatized if they become sexually active (Coleman 1966; Anderson 1989; Holland and Eisenhart 1990; Eyre et al. 1998; Holland et al. 1998; Kirkman et al. 1998). It predicts that, to the extent that adolescent females in the healthy-weight range are viewed as more attractive, they can be selective when choosing a sex partner because they have a broader range of potential partners, whereas overweight females may elect to remain virgins rather than suffer the adverse reputational effects associated with sexual activity. In addition, it predicts that although males have an unambiguous incentive to become sexually active, overweight males may be crowded out of the market.³ Finally, Cawley et al. (2006) noted that if overweight adolescents derive utility from intimacy,

³ The Cawley et al. (2006) model assumes that having sex with unattractive partners does not impose a reputational cost.

they may be willing have sex with overweight members of the opposite sex even at the cost of a reduced reputation.

The empirical evidence on the relationship between adolescent bodyweight and sexual initiation is mixed. Cawley (2001) used data from the 1979 National Longitudinal Survey of Youth. He found no evidence of a relationship between body weight and sexual activity, a result echoed by a number of studies drawing on smaller, non-representative samples (Halpern et al. 1999; Kallen and Doughtry 1984). In contrast, two more-recent studies have found evidence of a link between body weight and sexual behavior. Using data from Add Health on females ages 12 to 17, Halpern et al. (2005) found that a higher body mass index was associated with a lower probability of being in a sexual relationship. Cawley et al. (2006) used data from the 1997 National Longitudinal Survey of Youth (NLSY) and Add Health. They found evidence in both data sets that being overweight or obese was negatively related to the probability of dating. Although the relationship between body weight and becoming sexual activity was not significant in the NLSY, they found that overweight or obese adolescents in the Add Health data were less likely to become sexually active between interviews as compared to their healthy-weight counterparts. Moreover, the relationship between body weight and sexual activity was almost as large for male as for females.

We make several contributions to the existing literature on bodyweight and sexual behavior. Neither Cawley et al. (2006) nor Halpern et al. (2005) treated adolescent body weight as endogenously determined. Therefore, the negative relationship between bodyweight and sexual behavior documented by these authors could be due to unobservables or reverse causality. In an effort to account for the potential endogeneity of body weight, we employ two empirical strategies. First, we

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experiment with including individual fixed effects in our estimating equation to control for all time-invariant unobservables at the individual level; second, we use an instrumental variables approach. Our hope is that these methods will produce more credible estimates of the effect of bodyweight on the decision to become sexually active.

In addition, Cawley et al. (2006) and Halpern et al. (2005) relied on selfreported body weight and height. Because the Add Health interviewers measured the height and weight of respondents as part of the Wave II in-home interview process, we are able to avoid the use of self-reported weight and height in the majority of our specifications. Avoiding the use of self-reported weight and height is particularly important when there is reason to expect non-random measurement error. If, for instance, adolescents who underreported their body weight were also less likely to be sexually active, then the estimates produced by Cawley et al. (2006) could have been biased toward zero.⁴

III. The Data and Measures

The data used in our analysis come from the National Longitudinal Study of Adolescent Health, conducted by the Carolina Population Center at the University of North Carolina at Chapel Hill. The Adolescent Health data collection effort began with the identification of more than 26,000 high schools from across the United States. Eighty were selected from this population, and most were matched with a junior high or middle school from the same community, bringing the total number of participating schools to 132. From the student rosters of these 132 schools, a core sample was randomly chosen to be administered the Adolescent

⁴ Halpern et al. (2005) noted that the use of measured weight produced qualitatively similar results as those reported.

Health Wave I (baseline) in-home survey. In addition to this core sample, oversamples of black students with college-educated parents, Cuban and Puerto Rican students, and other groups were administered the Wave I in-home survey.

The Wave I in-home survey was completed by 20,746 adolescents between April and December of 1995. The Wave II in-home survey was conducted approximately one year later, between April and August of 1996. Our sample consists of 9,891 adolescents aged 14 to 17 at the time of the Wave II in-home survey. Six hundred fifty-two respondents with missing information on measured height, weight, virginity status, and pubertal development at Wave II were dropped from the cross-sectional analyses. In the longitudinal analysis, the sample was limited to respondents with non-missing information on height, weight, and virginity status at both Wave I and II.⁵

Our key independent variables are: (1) the respondent's weight in pounds (*Weight*) and (2) the respondent's body mass index (*BMI*) which was calculated as the respondent's weight in kilograms divided by his or her height in meters squared. In addition, we constructed relative measures of body weight using information from the Center for Disease Control: the respondent's percentile standing in the national weight-for-age distribution (*PctWeight*), and the respondent's percentile standing in the national BMI-for-age distribution (*PctBMI*), as well as the respondent's Z-score from these distributions (*ZWeight*, *ZBMI*). Moreover, to allow a nonlinear effect of body weight, we constructed a set of dichotomous variables indicating whether the respondent was underweight (0-5th percentile in the BMI-for-age distribution), at-risk of being overweight (85th to 95th percentile in the BMI-for-age distribution), or obese (95th to 100th percentile in the BMI-for-age distribution).

⁵ Further information regarding the Adolescent Health data collection effort is available from a variety of sources (Harris et al. 2006, Harris et al. 2002).

Our dependent variable, SEX_i , is dichotomous and based on the respondent's answer to the following question asked at Wave II:

Have you ever had sexual intercourse? When we say sexual intercourse, we mean when a male inserts his penis into a female's vagina.

The variable SEX_i is equal to 1 if a respondent indicated that they had had intercourse. If a respondent indicated that they were a virgin, the variable SEX_i is equal to 0. As Cawley et al. (2006) noted, self-reports of sexual behavior have been found to be relatively accurate, despite the sensitive nature of such subject matter (see, for example, Jaccard et al. 1995, 2002).

IV. Identification Strategies

We begin our analysis by using ordinary least squares (OLS) to benchmark the findings of Cawley et al. (2006).⁶ Specifically, we estimate the following equation:

(1)
$$Sex_i = \beta_0 + \beta'_1 X_i + \beta_2 Weight_i + \varepsilon_{i}$$

where *Sex* is defined above, *Weight* is replaced by *BMI*, *PctWeight*, *PctBMI*, *ZWeight* and *ZBMI* in alternative specifications, and both the respondent's height and weight are from measurements taken by the Add Health interviewer at Wave II. Drawing on data from the Add Health parental questionnaire, which was administered at Wave I, X_i includes parental education, household income, and measures of family structure. Other controls from the Wave I data are: the respondent's PPVT score (a measure of cognitive ability), his or her race, measures of

⁶ Logit and probit estimation strategies produced marginal effects that were comparable to those obtained using linear probability models.

religiosity, whether the respondent attended a public school, number of biological siblings, and whether the respondent had an older sibling. Controls from the Wave II data are: height as measured by the Add Health interviewer, attractiveness of the respondent as rated by the Adolescent Health interviewer, a scaled puberty index (and age of menarche for female respondents), age dummies, and an indicator for whether the respondent had ever been married.⁷

While we include a large set of controls in X_i , the estimate of β_2 will be biased if there are unobserved characteristics associated with body weight and sexual activity. For example, more impulsive adolescents may have greater difficulty controlling both their dietary and sexual appetites. It may also be the case that causality runs from sex to obesity. There is evidence that sexual activity is associated with the symptoms of depression among adolescent females (Hallfors et al. 2004; Sabia and Rees 2008), which could in turn lead to poor eating habits.

To address concerns, we exploit the longitudinal nature of the Add Health data. Using information from Waves I and II of the Add Health, we estimate:

(2)
$$SEX_{it} = \alpha + \beta_1 \mathbf{X}_{it} + \beta_2 Body Weight_{it} v_i + \varepsilon_{it},$$

where *t* indexes time, and v_i represents an individual-specific fixed effect. The time-varying controls included in X_i are: age, the puberty measures, and height. Identification of the relationship between body weight and sexual activity (β_2) comes from within-person variation over time (that is, between Waves I and II). Because measured weight and height are available only at Wave II, we rely on self-reported weight and height when estimating equation (2).⁸

⁷Table 1 of the appendix presents descriptive statistics for the variables used in the analysis.

⁸ Respondents were asked their height and weight at both the Wave I and Wave II interviews.

The advantage of the individual fixed effects model is that it controls for individual timeinvariant unobservables such as personality or self-discipline. However, there are a few wellknown problems associated with this empirical strategy. First, with the inclusion of personspecific fixed effects on the right-hand side of the estimating equation, the parameters are identified by within-person variation in body weight between Waves I and II. As noted, the second wave of the Adolescent Health study was administered approximately one year after the first. Any longer-term effect of body weight on virginity is captured by the individual intercepts and therefore is not reflected in the estimate of β_2 . Second, fixed-effects estimates are subject to omitted variable bias if the omitted variable in question is correlated both with changes in body weight from Wave I to Wave II and changes in virginity status over the same period. Third, it is likely that some portion of the observed changes in body weight from Wave I to Wave II is due to measurement error. It has been shown that measurement error of this type leads to estimates that are biased toward zero (Freeman 1984). Finally, the inclusion of individual fixed effects on the right-hand side of the estimation equation does not account for reverse causality.

An alternative identification strategy avoids the problems outlined above, but requires us to identify one or more instruments, represented by the vector \mathbf{Z}_{i} , correlated with body weight but uncorrelated with the error term of equation (1). If the body weight equation is given by

(3)
$$Weight_i = \gamma_0 + \gamma'_1 X_i + \gamma'_2 Z_i + \varepsilon_i,$$

then β_2 can be obtained using instrumental variables (two-stage least squares) estimation. This identification strategy will produce a consistent estimate of the effect of body weight on adolescent virginity provided that appropriate instruments can be found.

Following Cawley (2004), we rely on the assumption that genetics, rather than household environment, is the most prominent influence on body weight. This assumption is supported by a number of studies showing that show that the BMI of adoptees is essentially uncorrelated with the BMI of their adoptive parents (Stunkard et al. 1986; Vogler et al. 1995), and that the correlation in weight between biologically unrelated adopted children is statistically indistinguishable from zero (Grilo and Pogue-Geile 1991).⁹ Our instruments are the percentile BMI-for-age of the respondent's biological sibling, and an indicator of whether the respondent's biological mother was obese.¹⁰

We assess the validity of our instruments in several ways. First, we use a Hansen test of overidentifying restrictions to explore whether the instruments are correlated with the residuals of equation (1). Second, we test the robustness of the findings to the choice of instruments and controls for observable measures of parental involvement in the adolescent's sex life and sibling's sexual experience. Finally, we conduct a set of falsification tests on a set of outcomes correlated with adolescent virginity status, but that should, in theory, have no relation to body weight. These outcomes are: an indicator for whether the respondent smoked regularly in the last 30 days, whether he or she binge drank at least once per month in the past year, and whether he or she skipped school in the past year.

Table 1 presents descriptive statistics for our dependent variable, measures of body weight and the instruments for the OLS sample and for sample used in the instrumental variables

⁹ See also Sacerdote's (2007) study of Korean-American adoptees. Sacerdote (2007) found that heritability explained 30.8 percent of the variation in BMI, whereas shared family environment only explained 11.5 percent.

¹⁰ This second instrument is based on an item in the parental survey, which was usually completed by the respondent's biological mother. The parent was asked, "[d]oes the adolescent's biological mother now have [the health problem] of obesity?" *Mother Obese* is equal to 1 if the answer to this question was yeas, and equal to 0 if the answer was no.

(IV) analysis. The IV sample is much smaller than the full sample because it is restricted to respondents who had a full or half biological sibling participating in the Add Health.

In the OLS sample, 40.4 percent of females and 39.4 percent of males were sexually active. The mean weight of females in the sample was 135.9 pounds, and 25.6 percent were overweight or obese. For males, the average weight was 156.4 pounds, and 27.1 percent were overweight or obese. Appendix Table 1 presents descriptive statistics for the control variables in the vector X_{i} .¹¹

V. Results

OLS Estimates. Table 2 presents OLS estimates of the effect of body weight on virginity status using height and weight measured by the Wave II interviewer.¹² The first column presents results for female respondents and the second for male respondents. Each estimate in Table 2 is from a separate regression. All regressions are unweighted (as is common in the behavioral economics literature) but results are unchanged when weights are employed. Standard errors are clustered at the school-level to account for the Add Health research design.

Panel I of Table 2 shows the estimated effect of weight on the probability of being sexually active. Among female respondents, an additional pound of weight is associated with a 0.0014 decrease in the probability of sexual activity; a one percentile-point increase along the weight-for-age distribution is associated with a 0.0013 decrease in the probability of sexual activity; and a one standard deviation increase in weight is associated with a decrease of 0.036 in

¹¹ In addition to these controls, the vector \mathbf{X} included a set of indicators for missing values for each of the control variables.

¹² The focus of Table 2 (and subsequent tables) is on the estimates of the relationship between body weight and sexual activity. Coefficient estimates for the control variables are available upon request.

the probability of sexual activity. The results are similar when the BMI measures are used (Panel II). For example, a one percentile-point increase along the BMI-for-age distribution is associated with a 0.0012 decrease in the probability of being sexually active, and a one standard deviation increase is associated with a 0.035 decrease in this same probability. If the relationship between body weight and sexual activity for female respondents is allowed to be non-linear, we find that overweight and obese females are more likely to be virgins than their counterparts in the healthy range (those between the 5^{th} to 85^{th} percentiles). Being overweight is associated with a 0.071 decrease in the probability of being sexually active, and being obese is associated with a 0.099 decrease in this probability.

For male respondents, the pattern of results is quite different. When weight (or BMI) is entered linearly, there is little evidence that heavier male respondents were less likely to be sexually active than their slimmer counterparts. However, the results in Panel III suggest that, relative to males in the recommended weight range, both underweight and obese males were significantly less likely to be sexually active. In other words, there appears to be a U-shaped relationship between body weight and sexual activity for males. Cawley et al. (2006) also found evidence of a U-shaped relationship between body weight and sexual activity among male respondents to the Add Health.

Taken together, the results in Table 2 are consistent with those of Cawley et al. (2006) and Halpert et al. (2005). However, it is unclear whether the OLS estimates are causal in nature or simply reflect the influence of unobservables. Next, we explore the sensitivity of these estimates to the addition of controls for unmeasured heterogeneity.

Individual Fixed Effects Estimates. To examine the extent to which the results in Table 2 are driven by person-level heterogeneity, we use data from both Wave I and Wave II of the Add

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Health to estimate equation (2). However, as noted above, measured height and weight are not available in Wave I. As a consequence, we are forced to rely on self-reported height and weight when estimating equation (2).

We begin by presenting standard OLS estimates in which measured height and weight are replaced by self-reported height and weight (Table 3). The negative relationship between weight (or BMI) and sexual activity among female respondents documented in Table 2 persists in both Waves I and II (columns 1-2), suggesting that reporting error in these measures is not systematically correlated with virginity status. Next, we add individual fixed effects to the right hand-side of the estimating equation in order to identify the effect of body weight using withinperson variation. This exercise suggests that the negative association between body weight and sexual activity for females is not due to time-invariant unobservables at the individual level. Although the estimated coefficient of *Weight* is insignificant and smaller than that obtained using OLS, a one percentile-point increase in the weight-for-age distribution is associated with a 0.0011 decrease in the probability of being sexually active, and a one standard deviation increase is associated with a 0.026 decrease in this probability. A similar pattern of results is found using the BMI measures. However, when we allow body weight to have a non-linear effect, the fixed effects estimates become much smaller and are statistically indistinguishable from zero. There are two potential explanations for this finding. First, it may be that females within the recommended weight range are driving the effects documented in Panels I and II. It is also possible that there is insufficient person-specific variation across the weight categories between waves, which may lead to imprecise estimates.¹³

¹³ For example, only 6.2 percent of female respondents in our sample who were of normal weight at Wave I were obese or at risk for obesity by Wave II.

Among male respondents (columns 4-6), the fixed effects estimates provide little evidence of a negative relationship between weight or BMI and sexual activity, and, in contrast to the OLS estimates, individual fixed effects estimates show a much smaller relationship between being underweight or obese on the probability of being sexually active. Again, it is possible that this result is due to insufficient within-person variation.¹⁴

In Table 4, we restrict the fixed effects sample to respondents who were virgins at Wave I. This restriction allows us to more cleanly interpret β_2 as the effect of body weight on the likelihood that virgins became sexually active by Wave II.¹⁵ The results in Table 4 are similar to those reported in Table 3. We conclude that the negative relationship between body weight and sexual activity among female adolescents cannot be explained by time-invariant unobservables. However, because these estimates may be contaminated by reverse causality, time-varying unobservables, or measurement error, we next turn to an instrumental variables approach.

Instrumental Variables Estimates. Our instrumental variables analysis uses data from Wave II, and weight and height are based on the measurements taken by the Add Health interviewer. The sample is restricted to respondents with a biological sibling who also participated in the Add Health.

Table 5 presents evidence on the relevance of our two key instruments (the biological sibling's ranking in the BMI-for-age distribution and an indicator of whether the youth's biological parent was obese). The results show that the instruments are individually and jointly powerful predictors of body weight. For females, first-stage F-statistics on the instruments range

¹⁴ For example, only 6.8 percent of male respondents who were of normal weight at Wave I were obese or at risk for obesity by Wave II.

¹⁵ 362 respondents in the sample who reported having had sex at Wave I but claimed to be virgins at Wave II. We drop these respondents from all of our fixed effects analysis, but the results were qualitatively similar if these individuals were coded as being sexually active in both waves.

from 46.2 to 101.7, and for males from 42.8 to 58.5, which easily satisfy the instrument relevance standards proposed by Bound et al. (1994) and Staiger and Stock (1997).

In Table 6, we present second-stage results using weight in pounds (columns 1-2), BMI (columns 3-4), and an indicator for overweight or obese (columns 5-6). OLS estimates are also presented for the sake of comparison. Across all models, overidentification tests provide evidence that the instruments are valid.

For females (Panel I), 2SLS estimates show consistent evidence that increased body weight reduces the probability of being sexually active. A 10-pound increase in weight is associated with a 7.2 percent (0.028/0.387) decrease in the likelihood of being sexually active. The 2SLS estimate using BMI produces a similar result (column 4), as does using an indicator of overweight or obese (column 6). We find that being overweight or obese reduces the probability of being sexually active among females by 0.246.

For males, however, the pattern of results is again different (Panel II). Although OLS estimates for this sample suggest a negative association between weight and sexual activity, the 2SLS estimates show that each additional pound of weight is associated with a statistically insignificant 0.001 *increase* in the probability of being sexually active; and a one kilogram/meter² increase in BMI is associated with a 0.007 increase in this probability. Similarly, according to the OLS estimate, overweight or obese male adolescents had a 0.090 lower probability of being sexually active (column 5), but the corresponding 2SLS estimate is positive and statistically indistinguishable from zero (column 6).

Table 7 shows results for percentile and Z-score measures of weight and BMI. The findings are consistent with those in Table 6. For females, a one percentile increase in standing in the weight or BMI distribution decreases the probability of virginity loss by 0.003, and a one

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standard deviation increase in standing in the weight or BMI distribution decreases the probability of virginity loss by approximately 0.08. For males, however, there is no evidence of a causal effect of body weight on sexual abstinence.¹⁶

Robustness of Estimates. In Table 8, we examine the robustness of the 2SLS estimates of the effect of BMI on sexual activity. Panel I shows results for females and Panel II for males. We begin with a discussion of results for females. Column (1) shows our baseline 2SLS estimate from column (2) of Table 6. In column (2), we drop the parental indicator of obesity and use only on the sibling's measured BMI as an instrument. We do this out of concern that parental reports of obesity may not be exogenous to their child's health outcomes.¹⁷ 2SLS estimates that rely on sibling's BMI alone (column 2) are slightly smaller and less precise, but continue to show the same negative effect of BMI on the probability of sexual activity.

Another concern with using parental obesity as an instrument is that it could proxy for health-related problems of the infant or for risky behaviors by the mother at the time of the child's birth. Early childhood health problems could have long-run psychological or physical health consequences that persist in later adolescence and affect future health outcomes. To address this possibility, we control for the respondent's birthweight in column (3). The 2SLS estimate continues to show a negative effect of BMI on the probability of virginity loss.

¹⁶In Appendix Table 3, we explore whether the 2SLS estimates presented in Table 6 could mask an unobserved nonlinear relationship between body weight and sexual activity. Column (1) shows 2SLS estimates of the effect of being obese or overweight, restricting the sample to respondents who were either overweight or obese or in the recommended weight range; column (2) shows 2SLS estimates of the effect of being overweight, restricting the sample to respondents who were either overweight or in the recommended weight range; column (3) shows 2SLS estimates of the effect of being obese, restricting the sample to respondents who were either obese or in the recommended weight range; and column (4) shows 2SLS estimates of being underweight, restricting the sample to respondents who were underweight or in the recommended weight range. The results of these regressions were consistent with those reported in Tables 6 and 7.

¹⁷ If parents' willingness to report obesity problems is correlated with unobserved family-level psychological factors that affect child outcomes, then this instrument may not be exogenous (Sabia 2007).

Next, we examine whether parental obesity is correlated with contemporaneous parental behaviors that could be correlated with unmeasured determinants of adolescent virginity status. For example, it might be the case that parents who were particularly caring ensured that neither they nor their children were overweight and were also more involved with other aspects of their children's lives, including their sexual choices. In column (4), we add a set of indicators for parental involvement in various aspects of their child's social life. These indicators come from respondents' answers to the following parental questionnaire items:

- 1. How many parents of your child's friends have you talked to in the last four weeks? (0-6, where 6 is coded as 6 or more)
- 2. How often would it be true for you to make each of the following statements about your child:
 - a. You get along well with him/her. (=1 "always"; =2 "often"; =3 "sometimes"; =4 "seldom"; =5 "never")
 - b. You and your child make decisions about his/her life together. (=1 "always";
 =2 "often"; =3 "sometimes"; =4 "seldom"; =5 "never")
- Please tell me whether you are a member of a Parent/Teacher organization? (=0 "no"; = 1; yes)
- 4. Please tell me whether the following is true with regard to your present neighborhood: You live here because there is less drug use and other illegal activity by adolescents in this neighborhood. (=0 "no"; = 1 "yes")

When we add indicators for each of these controls to the estimating equation, the results are

almost identical to those presented in Table 6.

Next, in column (5), we add controls for parental and household-level substance use,

which could also be correlated with parental supervision. These controls were constructed from

the answers to the following questions:

- How often in the last month have you had five or more drinks on one occasion? (=1 "Never"; =2 "Once"; =3 "Twice"; =4 "Three times"; =5 "Four times"; =6 "Five or more Times")
- 2. Are there any cigarette smokers in your household? (=0 "no"; =1 "yes")

Adding a set of dichotomous controls for binge drinking and smoking does not appreciably change the 2SLS estimate of the estimated effect of BMI on sexual activity.

Estimates in column (6) show 2SLS estimates controlling for direct measures of parental communication with their child about sexuality. The addition of a set of indicators based on parental responses to the following questions has no appreciable effect on the 2SLS estimates:

- Have you talked to your child about birth control? (=1 "not at all"; =2 "somewhat";
 =3 "a moderate amount"; =4 "a great deal")
- 2. Have you talked to your child about sex? (=1 "not at all"; =2 "somewhat"; =3 "a moderate amount"; =4 "a great deal")

Finally, we explore the possibility of a direct effect of sibling body weight on sexual behavior. It may be the case that siblings of healthier body weight more easily attract greater numbers of members of the opposite sex, generating "spillover partners" for the respondent. Thus, in column (6), we try to ameliorate this concern by controlling for whether any of the respondent's biological siblings who participated in the Add Health study were sexually active. Our main finding is unchanged.

In sum, the results in Panel I of Table 8 show consistent evidence of a negative relationship between bodyweight and sexual activity for females. These findings suggest that the instruments we employ are not capturing unmeasured adolescent health, parental involvement, parental supervision, or the sexual behavior of siblings.

For males (Panel II), there is no evidence of a negative effect of BMI on virginity loss. In fact, the added controls for parental involvement/supervision and the sexual behavior of siblings actually result in a larger positive estimated effect, which is marginally significant in the some specifications. Specifically, we find that a one kilogram per meter-squared increase in BMI

leads to a marginally significant 0.016 *increase* in the probability of virginity loss for males. The pattern of results in Table 8 confirms that body weight affects male and female sexual options differently, and suggests that our instruments do not simply proxy for unmeasured family-level characteristics.

Falsification Tests. The final method of exploring the exogeneity of our instruments is through a set of falsification tests. If parental and sibling body weight are simply proxying for unmeasured parental monitoring or discipline, then our instrumental variables identification strategy would be expected to generate significant "effects" of body weight on adolescent risky behaviors that should not, in theory, be influenced by body weight. In Table 9, we estimate the relationship between body weight and three alternative outcomes: whether the respondent smoked regularly for 30 days in the last year, whether the respondent became drunk at least once per month in the last year, and whether the respondent had skipped school in the current academic year. Each is measured dichotomously and was generated from answers to the following Wave II survey items:

- 1. Since the month of the last interview, have you smoked cigarettes regularly, that is, at least one cigarette every day for 30 days. (=0 "no"; =1 "yes")
- 2. Over the past 12 months, on how many days did you get drunk or "very, very high" on alcohol? (=1 "every day or almost every day"; =2 "3 to 5 days a week"; =3 "1 or 2 days a week"; =4 "2 or 3 days a month"; =5 "once a month or less"; =6 "1 or 2 days in the past 12 months"; =7 "never")
- 3. During the [current] school-year, how many times have you skipped school for a full day without an excuse? (coded = 0 if 0; = 1 if positive days reported)

Each of the above outcomes may be correlated with body weight and may affect body weight, but there is little theoretical reason to imagine that any should be *affected by* body weight. The results of our falsification tests, shown in Table 9, show no evidence that body weight—measured by weight in pounds, BMI, or an indicator for overweight or obese—affects smoking, drunkenness, or unexcused absences from school for either males or females. In fact, the coefficient signs on body weight are often the reverse of what one might expect if parent's or sibling's body weight were proxying for unmeasured lack of discipline or supervision. For example, a one-pound increase in weight results in a statistically insignificant 0.001 *decrease* in the probability of frequent female drunkenness and a 0.002 *decrease* in the probability of skipping school. We view these results as further evidence that our instruments can be thought of as exogenous.

Effects on Other Sex-Related Outcomes. In Table 10, we restrict our focus to female respondents and explore whether bodyweight influenced the use of contraception at most recent sexual intercourse, pregnancy, and sexually transmitted infection.¹⁸ 2SLS estimates suggest that an increase in BMI is associated with a lower probability of having unprotected sex (column 1). Specifically, an additional BMI point is associated with a 0.010 percentage-point decline in the probability of not using birth control at most recent intercourse. The estimated effects on pregnancy and sexually transmitted infection are negative, but not statistically significant at conventional levels (columns 2-3).

Finally, in columns (3)-(6) of Table 10, we restrict our sample to female respondents who were sexually active at Wave II in order to explore whether, within this group, bodyweight impacts the use of contraception, pregnancy, or sexually transmitted infections. We hypothesize that bodyweight could influence bargaining power within a relationship, leaving overweight

¹⁸ Each of these outcomes was dichotomous in nature and generated from the following questionnaire items:

^{1.} Did you or your partner use any method of birth control when you had sexual intercourse most recently?

^{2.} Have you ever been pregnant? Be sure to include if you are currently pregnant and any past pregnancy that ended in an abortion, stillbirth, miscarriage, or a live birth after which the baby died.

^{3.} Actual blood-tests: Chlamydia, Gonorrhea, and Trich array results (if positive =1; if negative =0).

females with less control over contraceptive decisions. The estimated coefficients in columns (3)-(6) are negative, as expected, but are not statistically significant at conventional levels.

VI. Conclusions

During the 1990s and early 2000s, there was a sharp decline in the share of teenagers who reported being sexually active. In 1991, 54 percent of teenagers reported that they had had sexual intercourse at least once; by 2005 this figure had fallen to 47 percent (Youth Risk Behavior Survey 1991; 2005). Over the same period, teenage obesity increased by almost 70 percent (Behavior Risk Factor Surveillance System 1991; 2004). Public health officials lauded the former trend and expressed deep concern about the latter (see, for example, Mokdad et al. 2003), but did not suggest that they could be linked in a causal sense.

There is, however, cross-sectional evidence that obesity and adolescent sexual activity are related. A recent study by Cawley et al. (2006) found that heavier adolescents were less likely to be sexually active than their slimmer counterparts. One interpretation of this finding is that it reflects a causal relationship, consistent with the hypothesis that overweight youths are viewed as less desirable partners in the adolescent sex market (Halpern et al.1999; Sobal 2004). Another possibility is that unobservable factors at the individual level are driving the results found by Cawley et al. (2006). In other words, the estimates produced by Cawley et al. (2006) are consistent with the hypothesis that unmeasured factors such as self-discipline or personality are correlated with both sexual activity and weight. In an effort to distinguish between these hypotheses, we employ data from the Add Health and a

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number of empirical strategies, including individual fixed effects and instrumental variables models.

Fixed effects estimates suggest that the negative correlation between body weight and sexual activity observed among female adolescents cannot be explained by time-invariant unobservables. Female Add Health respondents who gained the most weight between Waves I and II had a corresponding lower probability of becoming sexually active. For males, however, the negative relationship between body weight and sexual activity is not robust to adding individual fixed effects to the right-hand side of the estimation equation. IV estimates confirm this basic pattern of results. They suggest that a 10-pound weight gain leads to approximately a 7 percent decline in the probability that female respondents were sexually active. In contrast, when the sample was restricted to males, IV estimation produced little evidence that body weight was related to sexual activity.

The results of this paper provide further evidence that overweight females face a different set of challenges than do overweight males, and these challenges extend to finding an acceptable match in what Cawley et al. (2006) labeled the "adolescent sex market." These findings also suggest that public policies designed to combat adolescent obesity—such as limiting the sale of "junk foods" in schools and mandating increased physical education requirements¹⁹—may have the unintended consequence of increasing sexual activity among adolescent females.²⁰ While increased body weight has been linked to depression (Franklin et al. 2006), low self-esteem (Onyike et al. 2003), and decreased academic achievement (Sabia, 2007), becoming sexually active at an early age has also been linked to these same outcomes (Sabia and

¹⁹ For example, in July 2007, a California law with similar provisions was enacted.

²⁰ We do note however, that our IV estimates should be interpreted as local average treatment effects (imbens and Angrist, 1994). An effective school-based anti-obesity program could affect a different set of marginal overweight females than our instruments do.

Rees Forthcoming; 2008; Hallfors et al. 2004). Therefore, although overweight adolescents who manage to reduce their weight will likely enjoy better physical health, our findings suggest that claims of second-order benefits with regard to mental health and academic achievement may be overstated. The obviously complicated relationships between gender, body weight, sexual activity, and psychological wellbeing is ripe for exploration in future work.

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	Females		Males		les
	OLS	IV	OL	.S	IV
	Sample	Sample	Sam	Iple	Sample
-	Cample	Cample	Can	ipic	Cample
<u>Dependent Variable</u>					
Sex	0.404	0.387	0.3	94	0.370
	(0.491)	(0.487)	(0.4	89)	(0.483)
Independent Variables					
Weight in Pounds	135.9	133.9	156	6.4	153.9
	(32.9)	(30.5)	(37	.2)	(34.9)
Percentile Weight-for-Age	61.6	60.3	64	.7	63.8
	(28.5)	(28.8)	(27	.8)	(27.0)
Z-Score Weight-for-Age	0.407	0.339	0.5	66	0.525
	(1.05)	(1.07)	(1.1	0)	(1.04)
Body Mass Index	22.9	22.6	22	.9	22.6
	(5.13)	(4.87)	(4.8	31)	(4.59)
Percentile BMI-for-Age	59.5	57.3	59	.6	58.5
	(28.9)	(29.1)	(29	.7)	(29.3)
Z-Score BMI-for-Age	0.330	0.250	0.3	53	0.311
	(1.05)	(1.06)	(1.1	3)	(1.10)
Underweight	0.031	0.043	0.0	39	0.039
(0-5th Percentile)	(0.174)	(0.203)	(0.1	94)	(0.194)
Overweight	0.142	0.133	0.1	32	0.127
(85th-95th Percentile)	(0.349)	(0.340)	(0.3	38)	(0.333)
Obese	0.114	0.101	0.1	39	0.127
(95th-100th Percentile)	(0.318)	(0.301)	(0.3	46)	(0.333)
Instruments					
Sibling's Percentile BMI-for-age		56.8 (29.3)			59.1 (29.8)
Biological mother obese		0.213 (0.410)			0.199 (0.400)
Ν	4946	1000	494	45	1023

Table 1. Means of Dependent, Independent, and Instrumental Variables, by Gender

*** Statistically significant at the 1% level; ** at 5% the level; * at the 10% level.

Notes: Standard deviations are in parentheses. The dependent and independent variables are measured using Wave II of the National Longitudinal Study of Adolescent Health. The sibling percentile BMI-forage is also measured at Wave II. The indicator of obesity by the biological mother is measured at Wave I when the parental questionnaire was administered. Height and weight variables of the respondent and the respondent's sibling are measured by the interviewer at Wave II. Sample includes those aged 14 to 17 at the time of the Wave II interview.

Table 2. OLS Estimates of Relationship between Body Weight and Sexual Activity, by Gender

	Females	Males		
	Panel I	·Weight		
	i uner i	Weight		
	(1)	(2)		
Weight in Pounds	-0.0014*** (0.0003)	-0.0002 (0.0002)		
Percentile Weight-for-Age	-0.0013*** (0.0003)	0.0002 (0.0003)		
Z-Score Weight-for-Age	-0.036*** (0.009)	0.001 (0.007)		
	Panel II: BMI			
Body Mass Index	-0.0083*** (0.0015)	-0.0011 (0.0013)		
Percentile BMI-for-Age	-0.0012*** (0.0003)	0.0002 (0.0002)		
Z-Score BMI-for-Age	-0.035*** (0.007)	0.003 (0.006)		
	Panel III: No	on-linear BMI		
Underweight (0-5th Percentile)	-0.004 (0.036)	-0.050 (0.034)		
Overweight (85th-95th Percentile)	-0.071*** (0.017)	-0.006 (0.021)		
Obese (95th-100th Percentile)	-0.099*** (0.024)	-0.046** (0.019)		
Ν	4946	4945		

*** Statistically significant at the 1% level; ** at 5% the level; * at the 10% level.

Notes: Standard errors corrected for clustering at the school level are in parentheses. Estimates are from unweighted OLS regressions based on data from Waves I and II of the National Longitudinal Study of Adolescent Health. The sample includes respondents aged 14-17 at the time of the Wave II survey. All models include the full set of controls listed in Appendix Table 1, and use measured height and weight.

Table 3. OLS and Individual Fixed Effects Estimates of Relationship between Body Weight and Sexual Activity, by Gender

		Females			Males	
			Panel I:	Weight		
	OLS (Wave I) (1)	OLS (Wave II) (2)	IFE (3)	OLS (Wave I) (4)	OLS (Wave II) (5)	IFE (6)
Weight in Pounds	-0.0013***	-0.0015***	-0.0007	-0.0002	-0.0002	0.0002
	(0.0002)	(0.0003)	(0.0005)	(0.0002)	(0.0002)	(0.0005)
Percentile Weight-for-Age	-0.0009***	-0.0011***	-0.0011***	0.0004	0.0006*	-0.0000
	(0.0002)	(0.0003)	(0.0004)	(0.0003)	(0.0003)	(0.0005)
Z-Score Weight-for-Age	-0.030***	-0.033***	-0.026**	0.002	0.003	0.003
	(0.007)	(0.009)	(0.012)	(0.007)	(0.007)	(0.016)
		Panel II: BMI				
		01.0				
	(Wave I)	(Wave II)	IFE	(Wave I)	(Wave II)	IFE
	(1)	(2)	(3)	(4)	(5)	(6)
Body Mass Index	-0.0071***	-0.0086***	-0.0049*	-0.0014	-0.0009	0.0006
	(0.0013)	(0.0016)	(0.0027)	(0.0015)	(0.0005)	(0.0030)
Percentile BMI-for-Age	-0.0009***	-0.0010***	-0.0009**	0.0003	0.0004*	0.0000
	(0.0002)	(0.0003)	(0.0004)	(0.0002)	(0.0002)	(0.0004)
Z-Score BMI-for-Age	-0.028***	-0.032***	-0.019**	0.005	0.006	0.004
	(0.006)	(0.008)	(0.010)	(0.006)	(0.006)	(0.012)
			Panel III: No	n-linear BMI		
	OLS (Wave I) (1)	OLS (Wave II) (2)	IFE (3)	OLS (Wave I) (4)	OLS (Wave II) (5)	IFE (6)
Underweight	0.048	-0.044	-0.005	-0.123***	-0.090**	-0.023
(0-5th Percentile)	(0.036)	(0.034)	(0.022)	(0.031)	(0.044)	(0.027)
Overweight	-0.013	-0.059***	0.005	0.022	0.016	-0.002
(85th-95th Percentile)	(0.017)	(0.017)	(0.018)	(0.017)	(0.020)	(0.018)
Obese	-0.083***	-0.121***	0.002	-0.063***	-0.059***	-0.015
(95th-100th Percentile)	(0.018)	(0.023)	(0.029)	(0.018)	(0.020)	(0.025)
Ν	4891	4891	4891	4507	4507	4507

*** Statistically significant at the 1% level; ** at 5% the level; * at the 10% level.

Notes: Standard errors corrected for clustering at the school level. Estimates are from unweighted OLS and IFE regressions based on data from Waves I and II of the National Longitudinal Study of Adolescent Health. The sample includes respondents aged 14-17 at the time of the Wave II survey. OLS models include the full set of controls listed in Appendix Table 1. Fixed effects models include controls for age, puberty, and height. All models use self-reported height and weight.

	Females				Males			
			Danol	1: Woight				
			Fallel	i. weigin				
	Pounds (1)	Percentile (2)	Z-Score (3)	Pounds (4)	Percentile (5)	Z-Score (6)		
Weight	-0.001** (0.0006)	-0.002*** (0.0006)	-0.039** (0.016)	-0.0008 (0.0006)	-0.0009 (0.0007)	-0.021 (0.019)		
			Pane	el II: BMI				
	BMI (1)	Percentile (2)	Z-Score (3)	BMI (4)	Percentile (5)	Z-Score (6)		
BMI	-0.006* (0.004)	-0.001*** (0.0005)	-0.028** (0.013)	-0.001 (0.004)	-0.0003 (0.0005)	-0.006 (0.014)		
			Panel III: I	Non-linear BM	Ι			
Underweight (0-5th Percentile)		-0.015 (0.027)			-0.013 (0.031)			
Overweight (85th-95th Percentile)		0.008 (0.024)			-0.006 (0.025)			
Obese (95th-100th Percentile)		0.016 (0.038)			-0.022 0.034)			
Ν	3578	3578	3578	3262	3262	3262		

Table 4. Individual Fixed Effects Estimates Conditional on Sample of Virgins at Wave I

*** Statistically significant at the 1% level; ** at 5% the level; * at the 10% level.

Notes: Standard errors corrected for clustering at the school level. Estimates are from unweighted IFE regressions based on data from Waves I and II of the National Longitudinal Study of Adolescent Health. The sample includes respondents aged 14-17 at the time of the Wave II survey. All models include controls for age, puberty, and height, and use self-reported weight and height. The sample for this analysis is conditional on virginity at Wave I.

	Females				Males			
			Panel	1: V	Veight			
	Pounds (1)	Percentile (2)	Z-Score (3)		Pounds (4)	Percentile (5)	Z-Score (6)	
Biological Sibling BMI	0.296*** (0.031)	0.269*** (0.026)	0.010*** (0.001)		0.297*** (0.032)	0.259*** (0.028)	0.010*** (0.001)	
Biological Mother Obese	13.9*** (2.97)	11.4*** (1.77)	0.453*** (0.077)		11.7*** (2.65)	5.52*** (1.67)	0.295*** (0.070)	
F-stat on instruments	F = 63.7	F = 98.2	F = 101.7		F = 51.7	F = 47.3	F = 54.0	
			Pane	Panel II: BMI				
	BMI (1)	Percentile (2)	Z-Score (3)		BMI (4)	Percentile (5)	Z-Score (6)	
Biological Sibling BMI	0.050*** (0.005)	0.309*** (0.028)	0.011*** (0.001)		0.043*** (0.005)	0.333*** (0.032)	0.012*** (0.001)	
Biological Mother Obese	2.45*** (0.509)	11.9*** (11.9)	0.452*** (0.081)		1.69*** (0.381)	6.56*** (2.15)	0.306*** (0.082)	
F-stat on instruments	F = 63.4	F = 98.8	F = 98.5		F = 50.3	F = 56.0	F = 58.5	
	F	Panel III: O	verweight (′ 85 t	h-100th Bl	Al Percentil	e)	
		Overwgt (1)				Overwgt (2)		
Biological Sibling BMI		0.003*** (0.0005)				0.003*** (0.0004)		
Biological Mother Obese		0.165*** (0.036)				0.179*** (0.035)		
F-stat on instruments		F = 46.2				F = 42.8		
Ν	1000	1000	1000		1023	1023	1023	

Table 5. Estimates of Effect of Instruments on Body Weight

*** Statistically significant at the 1% level; ** at 5% the level; * at the 10% level.

			Panel I:	Females		
	Weight (Pounds)		BI	BMI		t or Obese
	(1) OLS	(2) 2SLS	(3) OLS	(4) 2SLS	(5) OLS	(6) 2SLS
Body Weight	-0.0014*** (0.0005)	-0.0028** (0.0014)	-0.008** (0.003)	-0.017** (0.008)	-0.100*** (0.037)	-0.246** (0.125)
F-stat on instruments in first stage J-stat on Hansen overid test p-value on overid test N	N = 1000	F = 63.7 J = 0.408 p = 0.52 N = 1000	N = 1000	F = 63.4 J = 0.373 p = 0.54 N = 1000	N = 1000	F = 46.2 J = 0.360 p = 0.55 N = 1000
			Panel I	I: Males		
	Weight (Pounds)	В	MI	Overweigh	t or Obese
	(1) OLS	(2) 2SLS	(3) OLS	(4) 2SLS	(5) OLS	(6) 2SLS
Body Weight	-0.0009** (0.0005)	0.0010 (0.0013)	-0.006** (0.003)	0.007 (0.009)	-0.090*** (0.034)	0.070 (0.104)
F-stat on instruments in first stage J-stat on Hansen overid test p-value on overid test N	N = 1023	F = 51.7 J = 0.652 p = 0.42 N = 1023	N = 1023	F = 50.3 J = 0.645 p = 0.42 N = 1023	N = 1023	F = 42.8 J = 0.780 p = 0.38 N = 1023

Table 6. OLS and 2SLS Estimates of Relationship between Body Weight and Sexual Activity, by Gender

*** Statistically significant at the 1% level; ** at 5% the level; * at the 10% level.

Table 7. 2SLS Estimates of Relationship between Percentile and Z-Score Body Weight and Sexual Activity, by Gender

	Panel I: Females							
	We	ight	BI	MI				
	(1)	(2)	(3)	(4)				
	Weight-for-Age	Weight-for-Age	BMI-for-Age	BMI-for-Age				
	Percentile	Z-Score	Percentile	Z-Score				
Body Weight	-0.003*	-0.083**	-0.003*	-0.076**				
	(0.002)	(0.042)	(0.001)	(0.039)				
F-stat on instruments in first stage	F = 98.2	F = 101.7	F = 98.8	F = 98.5				
J-stat on Hansen overid test	J = 0.480	J = 0.457	J = 0.565	J = 0.561				
p-value on overid test	p = 0.49	p = 0.50	p =0.45	p = 0.45				
N	N = 1000	N = 1000	N = 1000	N = 1000				
		Panel I	: Males					
	We	ight	BI	MI				
	(1)	(2)	(3)	(4)				
	Weight-for-Age	Weight-for-Age	BMI-for-Age	BMI-for-Age				
	Percentile	Z-Score	Percentile	Z-Score				
Body Weight	0.002	0.030	0.001	0.030				
	(0.002)	(0.035)	(0.001)	(0.035)				
F-stat on instruments in first stage	F = 47.3	F = 58.5	F = 56.0	F = 58.5				
J-stat on Hansen overid test	J = 0.427	J = 0.471	J = 0.402	J = 0.471				
p-value on overid test	p = 0.51	p = 0.49	p = 0.53	p = 0.49				
N	N = 1023	N = 1023	N = 1023	N = 1023				

*** Statistically significant at the 1% level; ** at 5% the level; * at the 10% level.

			Pa	nel I: Female	s		
:					-		
	Baseline 2SLS	IV: Sibling BMI only	(1) + control for birthweight	(3) + controls for parental involvement	(4) + controls for parental risky behaviors	(5) + parental sex talks	(6) + sibling virginity
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
BMI	-0.017** (0.008)	-0.014 (0.010)	-0.022** (0.009)	-0.020** (0.009)	-0.021** (0.009)	-0.022** (0.009)	-0.019** (0.009)
F-stat on instruments J-stat on overid test p-value on overid test N	F = 63.4 J = 0.373 p = 0.54 N = 1000	F = 107.0 N = 1000	F = 53.8 J = 0.005 p = 0.94 N = 882	F = 44.8 J = 0.001 p = 0.97 N = 834	F = 42.1 J = 0.001 p = 0.97 N = 833	F = 42.0 J = 0.025 p = 0.87 N = 825	F = 42.3 J = 0.023 p = 0.86 N = 825
			P	anel II: Males	5		
	Baseline 2SLS	IV: Sibling BMI only	(1) + control for birthweight	(3) + controls for parental involvement	(4) + controls for parental risky behaviors	(5) + parental sex talks	(6) + sibling virginity
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
BMI	0.007 (0.009)	0.010 (0.010)	0.013 (0.009)	0.014 (0.009)	0.016* (0.009)	0.015* (0.009)	0.016* (0.009)
F-stat on instruments J-stat on overid test p-value on overid test N	F = 50.3 J = 0.645 p = 0.42 N = 1023	F = 99.6 N = 1023	F = 44.4 J = 0.194 p = 0.66 N = 916	F = 43.3 J = 0.693 p = 0.41 N = 865	F = 43.5 J = 0.079 p = 0.78 N = 863	F = 45.4 J = 0.402 p = 0.53 N = 854	F = 46.0 J = 0.230 p = 0.63 N = 854

Table 8. Robustness of 2SLS Estimates of Effect of BMI on Sexual Activity

*** Statistically significant at the 1% level; ** at 5% the level; * at the 10% level.

Table 9. Faisification Tests 25L5 Estimates of Effect of Body weight on other Risky Benavior
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		Females				Males	
		i cinaico		=		males	
	Smoking	Drunk ≥	Skip	-	Smoking	Drunk ≥	Skip
	last 30 d	once/mo	school		last 30 d	once/mo	school
	(1)	(2)	(3)	_	(4)	(5)	(6)
NA7 * 1 /	0.004	0.004			0.004	0.004	
Weight	0.001	-0.001	-0.002		-0.001	-0.001	-0.000
	(0.001)	(0.001)	(0.001)		(0.001)	(0.001)	(0.001)
F-stat on instruments	F = 63.7	F = 65.2	F = 63.7		F = 52.5	F = 51.7	F = 51.8
J-stat on overid test	J = 0.058	J = 1.53	J = 0.053		J = 0.135	J = 1.32	J = 1.28
p-value on overid test	p = 0.81	p = 0.22	p = 0.82		p = 0.71	p = 0.25	p = 0.26
Ν	N = 997	N = 998	N = 1000		N = 1016	N = 1023	N = 1021
		Females				Males	
				-			
	Smoking	Drunk ≥	Skip	-	Smoking	Drunk ≥	Skip
	last 30 d	once/mo	school		last 30 d	once/mo	school
	(1)	(2)	(3)	-	(4)	(5)	(6)
BMI	0.006	-0.006	-0 009		-0.006	-0.006	-0.001
Divit	(0,006)	(0.006)	(0.003)		(0.008)	(0,006)	(0,009)
	(0.000)	(0.000)	(0.000)		(0.000)	(0.000)	(0.000)
F-stat on instruments	F = 63.4	F = 65.1	F = 63.4		F = 51.5	F = 50.3	F = 50.4
J-stat on overid test	J = 0.052	J = 1.57	J = 0.044		J = 0.136	J = 1.33	J = 1.28
p-value on overid test	p = 0.82	p = 0.21	p = 0.83		p = 0.71	p = 0.25	p = 0.26
Ν	N = 997	N = 998	N = 1000		N = 1016	N = 1023	N = 1021
		Females				Males	
				-			
	Smoking	$Drunk \geq$	Skip		Smoking	$Drunk \ge$	Skip
	last 30 d	once/mo	school		last 30 d	once/mo	school
	(1)	(2)	(3)	-	(4)	(5)	(6)
Overweight or Obese	0 091	-0 084	-0 136		-0 075	-0.081	-0.003
	(0.091)	(0.085)	(0.112)		(0.089)	(0.068)	(0.108)
			- ··			- · · · ·	- ·
F-stat on instruments	F = 45.9	F = 47.4	F = 46.7		F = 43.1	F = 42.8	F = 42.7
J-stat on overia test	J = 0.052	J = 1.56	J = 0.044		J = 0.088	J = 1.12	J = 1.26
p-value on overlu (est N	μ = 0.02 N = 007	h = 0.21 N = 008	p = 0.63 N = 1000		μ = 0.77 N = 1016	p = 0.29 N = 1023	µ = 0.20 N – 1∩21
/ •	11 - 331	IN - 330	N - 1000			11 - 1023	11 - 1021

*** Statistically significant at the 1% level; ** at 5% the level; * at the 10% level.

	U	nconditiona	l	Conditional			
	Unprotected Sex at Recent Intercourse	Pregnant	Sexually Transmitted Infection	Unprotected Sex at Recent Intercourse	Pregnant	Sexually Transmitted Infection	
	(1)	(2)	(3)	(4)	(5)	(6)	
BMI	-0.010* (0.005)	-0.001 (0.004)	-0.002 (0.004)	-0.024 (0.018)	0.006 (0.013)	-0.005 (0.010)	
F-stat on instruments J-stat on overid test p-value on overid test N	F = 61.0 J = 0.073 p = 0.79 N = 929	F = 64.4 J = 0.742 p = 0.39 N = 997	F = 61.1 J = 0.015 p = 0.90 N = 719	F = 20.3 J = 0.168 p = 0.68 N = 316	F = 28.0 J = 2.13 p = 0.14 N = 384	F = 30.7 J = 0.025 p = 0.87 N = 274	

*** Statistically significant at the 1% level; ** at 5% the level; * at the 10% level.

Appendix Table 1. Means of Control Variables for Females¹

Control Variables		Other Christian	0.198 (0.399)	Age of Menarche	12.1 (1.30)
Log Household Income	10.4 (0.821)	Non-Christian Relig	0.043 (0.203)	Look younger than most	0.078 (0.268)
Parent Completed High School	0.301 (0.459)	Black	0.233 (0.423)	Look younger than some	0.103 (0.304)
Parent Trade School	0.092 (0.293)	Asian	0.058 (0.233)	Look older than some	0.284 (0.451)
Parent Some College	0.191 (0.393)	Indian	0.014 (0.118)	Look older than most	0.134 (0.341)
Parent College Ed	0.146 (0.353)	Hispanic/Other	0.163 (0.370)		
Parent Post-College Education	0.099 (0.299)	Height (inches)	64.5 (2.73)		
Single Parent	0.065 (0.247)	Age at Wave II ²	15.7 (1.08)		
Separated	0.055 (0.229)	Public School	0.923 (0.266)		
Widowed	0.036 (0.186)	Small School Size	0.169 (0.375)		
Divorced	0.147 (0.355)	Medium School Size	0.398 (0.489)		
PVT Score	99.6 (14.9)	Number biological siblings	1.57 (1.40)		
Rural	0.167 (0.373)	Whether have older sibling	0.498 (0.500)		
Suburban	0.535 (0.469)	Very attractive	0.169 (0.375)		
West	0.229 (0.420)	Attractive	0.399 (0.490)		
Midwest	0.265 (0.441)	Unattractive	0.030 (0.169)		
South	0.365 (0.481)	Very unattractive	0.009 (0.096)		
Catholic	0.249 (0.432)	Ever Married ³	0.009 (0.070)		
Baptist or Methodist	0.399 (0.490)	Parent's Age	41.2 (6.54)		

¹Sample size is 4,946. Standard deviations are in parentheses. Height, puberty measured, attractiveness, and marital status are are measured at Wave II. All other control variables are measured at Wave I.

²Age dummies are included in each regression.
³Marriage only measured for those aged 15 and older.

Appendix Table 2. Means of Control Variables for Males¹

Control Variables		Other Christian	0.194 (0.398)	Look younger than most	0.119 (0.323)
Log Household Income	10.5 (0.814)	Non-Christian Relig	0.045 (0.206)	Look younger than some	0.108 (0.310)
Parent Completed High School	0.286 (0.452)	Black	0.212 (0.409)	Look older than some	0.252 (0.434)
Parent Trade School	0.097 (0.296)	Asian	0.070 (0.256)	Look older than most	0.130 (0.337)
Parent Some College	0.206 (0.404)	Indian	0.016 (0.127)		
Parent College Ed	0.159 (0.366)	Hispanic/Other	0.171 (0.377)		
Parent Post-College Education	0.091 (0.288)	Height (inches)	69.1 (3.39)		
Single Parent	0.054 (0.225)	Age at Wave II ²	15.7 (1.08)		
Separated	0.043 (0.203)	Public School	0.922 (0.267)		
Widowed	0.031 (0.174)	Small School Size	0.165 (0.371)		
Divorced	0.158 (0.365)	Medium School Size	0.406 (0.491)		
PVT Score	101.0 (15.2)	Number biological siblings	1.57 (1.40)		
Rural	0.171 (0.377)	Whether have older sibling	0.501 (0.500)		
Suburban	0.543 (0.498)	Very attractive	0.092 (0.289)		
West	0.228 (0.419)	Attractive	0.340 (0.474)		
Midwest	0.253 (0.435)	Unattractive	0.050 (0.219)		
South	0.367 (0.482)	Very unattractive	0.010 (0.097)		
Catholic	0.257 (0.437)	Ever Married ³	0.001 (0.022)		
Baptist or Methodist	0.372 (0.483)	Parent's Age	41.2 (6.59)		

¹Sample size is 4,945. Standard deviations are in parentheses. Height, puberty measured, attractiveness, and marital status are are measured at Wave II. All other control variables are measured at Wave I.

 $^{2}\mbox{Age}$ dummies are included in each regression.

³Marriage only measured for those aged 15 and older.

Appendix Table 3. 2SLS Estimates of Nonlinear Relationship between Body Weight and Sexual Activity, by Gender

	Panel I: Females				
	Overweight or Obese vs Normal BMI	Overweight vs Normal BMI	Obese vs Normal BMI	Underweight vs Normal BMI	
	(1)	(2)	(3)	(4)	
Body Weight	-0.274** (0.123)	-0.320 (0.205)	-0.397** (0.182)	0.504 (0.520)	
F-stat on instruments in first stage J-stat on Hansen overid test p-value on overid test N	F = 43.4 J = 0.200 p = 0.66 N = 957	F = 15.9 J = 0.067 p = 0.80 N = 856	F = 25.6 J = 0.002 p = 0.97 N = 824	F = 7.6 J = 0.382 p = 0.54 N = 766	
	Panel II: Males				
	Overweight or Obese vs Normal BMI	Overweight vs Normal BMI	Obese vs Normal BMI	Underweight vs Normal BMI	
	(1)	(2)	(3)	(4)	
Body Weight	0.033 (0.107)	0.179 (0.204)	0.010 (0.141)	-0.918 (0.566)	
F-stat on instruments in first stage J-stat on Hansen overid test p-value on overid test N	F = 39.7 J = 0.232 p = 0.63 N = 983	F = 15.1 J = 0.190 p = 0.66 N = 853	F = 28.7 J = 0.796 p = 0.37 N = 853	F = 4.1 J = 0.574 p = 0.45 N = 763	

*** Statistically significant at the 1% level; ** at 5% the level; * at the 10% level.