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The Effect of Weight on Mental Health: New Evidence Using Genetic IVs

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Abstract

Average body mass index (BMI) and depression prevalence grew over the last several decades, increasing medical expenditures. There is a strong correlation between obesity and depression but limited evidence on the causal effect of weight on mental health. I use an index of genetic risk for high BMI as a source of exogenous variation in weight to provide novel evidence on the effect of weight on mental health. This is one of the first studies to use genetics as an instrument for BMI and to examine the causal relationship between weight and depression. Results are mixed; I find a meaningful and significant effect of weight on suicidal ideation but no effects on counselling and an index of depression. The effect on suicidal ideation is concentrated in white females. From respondent and interviewer opinions of respondent attractiveness, social stigma is a mechanism through which weight affects mental health for white women.

1. Introduction

In the United States, there has been an across the board shift in the weight distribution. The prevalence of obesity (BMI ≥ 30) has increased dramatically from less than 20% in the early 1960s to over 30%, with a similar decrease in healthy weight (BMI 18.5–24.9) (Centers for Disease Control and Prevention, 2003; Fryar et al., 2012). As seen in Appendix Figure A1 and Appendix Figure A2, no group has avoided increases in obesity (Fryar et al., 2012). Commensurate with the rise in weight has been a marked increase in the rate of depression, from 3.3% in 1991–92 to 7.1% in 2001–02 (Compton et al., 2006). Increases in weight and frequency of mental illness are imposing large costs on society. Medical expenditures on weight-related care have reached \$200 billion per year in the United States, and Americans spend \$100 billion annually on mental health treatments (Cawley and Meyerhoefer, 2012; Mark et al., 2007). These medical expenditures cause negative externalities to other members of insurance risk pools and taxpayers in the case of public insurance. Additionally, mental health may serve as an important pathway through which weight affects labor market outcomes.¹ Mental illness also can have negative spillovers to family members, in terms of

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¹See Averett (2014) and Cawley (2007) for reviews of the literature on the effect of weight on wages and employment.

decreased earnings and damaged relationships which can lead to divorce (Lépine and Briley, 2011).

The rise in weight – particularly the severity and the prevalence of obesity – has led to a great deal of interest in the effects of weight on physical health (Fryar et al., 2012; World Health Organization, 2017). However, there is much less research about the causal effects of weight on mental health. The influence of body mass index (BMI) on mental health has important ramifications for how we measure the overall impact of the ongoing obesity epidemic. This paper aims to help fill the gap in the literature on the effect of weight on mental health by using a genetic index of risk for high body mass index to instrument for BMI. In addition, this research addresses the possible mechanisms – body image, disrupted social relationships including stigma, and health problems – through which weight affects mental health. Understanding the empirical relevance of these various mechanisms is important in shaping policies to address depression.

Not only have the increases in weight and depression coincided over time, but the correlational evidence is quite clear: there is a strong positive association between high BMI and depression (Luppino et al., 2010; Pratt and Brody, 2014; Scott et al., 2010; Zhao et al., 2009). The correlation between weight and depression could operate through three non-mutually exclusive potential pathways: (1) higher BMI harms mental health, (2) poor mental health causes weight gain – such as through changes in diet or as a side effect of anti-depressants, or (3) a third factor – such as parental SES – affects both mental health and weight. This paper tests the first pathway, whether being heavier harms mental health. It is important to distinguish between these three relationships. For instance, if higher BMI has a negative effect on mental health, it has important ramifications for how we calculate the cost of the obesity epidemic. Similarly, efforts to reduce BMI can have important spillovers that improve mental health; as communities, policymaker, and employers invest more resources to fight increasing BMIs, it will be useful to consider the mental health benefits of any success. Additionally, paying special attention to overweight and obese people during mental health screenings could better identify individuals with increased risk of mental health problems.

Because weight is not randomly assigned and typically is based on a number of factors that researchers cannot observe, overcoming problems associated with endogeneity is very difficult in this literature. Consequently, there is a dearth of studies that use credible causal methods to estimate the effect of weight on mental health. Two recent papers make progress on this difficult endogeneity problem by using biological relatives' BMI/obesity as instruments for respondents' weight. Sabia and Rees (2015) find that increased weight adversely affects mental health, primarily for females, while Bjørngaard et al. (2015) find higher weight increases depression but not anxiety. While this instrument provides a clear advancement relative to prior associational work, it cannot disentangle the causal effects of BMI from the effects stemming from other common factors within the household.

This paper contributes to the literature by leveraging individual level genetic information to examine the effect of weight on mental health within an instrumental variables (IV) strategy. The use of genetic information – derived from biological samples and laboratory analysis –

as an instrument for BMI is in contrast to much of the previous literature that relied on biological relatives to proxy for genetics (Bjørngaard et al., 2015; Sabia and Rees, 2015).² I use the National Longitudinal Study of Adolescent to Adult Health (Add Health), which contains data on height and weight, information on mental health, and an index of genetic predisposition for high BMI. The BMI Genetic Risk Score, the instrument used in this study, is based on 31 individual pieces of genetic information known as Single Nucleotide Polymorphisms (SNPs) that are related to elevated BMI. The SNPs included in the BMI Genetic Risk Score are based on a meta-analysis of genome-wide association studies (GWAS) and verification by geneticists (Speloites et al., 2010).

The central identifying assumption underlying my approach is that the BMI Genetic Risk Score is only related to mental health through weight. The main concern is that the SNPs in the BMI Genetic Risk Score are correlated with other variables, such as family socio-economic status, which also independently affect mental health. I show that the instrument is unrelated to a series of individual background characteristics, such as parental education and income, and I also control for parental obesity to account for concerns related to the correlation of parent and child obesity and mental health. By using a genetic index as the instrument, I use only the variation in BMI related to those genes in the index (Speloites et al., 2010). While one can never completely prove that the exclusion restriction holds, it allows me to relax the assumption associated with using a biological relative as an instrument for obesity, that shared environmental factors or genetics unrelated to weight are uncorrelated with mental health outcomes.

My results for the effect of weight on mental health are mixed. I find an economically meaningful and statistically significant effect of weight on suicidal ideation at the 10% level, but not use of counseling services or a depression index. The effect on suicidal ideation is primarily in white females. A concentration of effects in females is consistent with Sabia and Rees (2015) as well as with the literature on the effect of weight on labor market outcomes (Averett, 2014). However, I find a weaker relationship between weight and mental health than previous research using sibling BMI as an instrument. When I examine mechanisms, I find that social stigma, as measured by the interviewer's opinion of the respondent's attractiveness, is a mechanism through which weight affects mental health for this group.

This paper is organized as follows: Section 2 presents the data, including the instrument and the measures of BMI and mental health; Section 3 explains the research design and method; Section 4 presents empirical results; Section 5 explores potential mechanisms; and Section 6 concludes with a discussion.

²As of the start of 2016, only four studies (Ding et al., 2009; Fletcher and Lehrer, 2011; Norton and Han, 2008; von Hinke et al., 2016) used genetics to instrument for BMI in the economics literature (von Hinke et al., 2016). Importantly, von Hinke et al. (2016) note that the earlier studies used genes correlated with neurotransmitters, which may violate the exclusion criterion. Both von Hinke et al. (2016) and I use genetic information based on Speloites et al. (2010) that appears not to be directly connected to neurological function. As of June 2017, Google Scholar indicates 14 papers have cited von Hinke et al. (2016). Of the 14, only one is an economics study that uses genetics as an instrument for BMI, a working paper by Böckerman et al. (2016).

2. Data

This paper uses data from the National Longitudinal Study of Adolescent to Adult Health (Add Health). Add Health, a national panel study, began data collection when participants were in 7th through 12th grade. The first two waves of data collection occurred while respondents were adolescents in 1995 and in 1996; waves 3 and 4 were collected during early adulthood in 2001–2002 and in 2008 when respondents were aged 18–34. Add Health contains a wide variety of information, including details about the respondent's school reported by an administrator and information about family and household reported by an adult household member, as well as neighborhood and community characteristics from administrative datasets such as the Centers for Disease Control Sexually Transmitted Disease File.

Importantly, Add Health collected information about height, weight, and mental health in all four waves. The BMI Genetic Risk Score was collected in wave 4 and is time invariant. Most control variables, including parental characteristics, were collected at baseline and are treated as fixed. This information is in Appendix Table A1 as well as summary statistics on the data aggregated across waves. Respondents are quite young and results might not extrapolate to older populations. However, young adults have higher rates of depression than older adults (Compton et al., 2006), so this may be the most appropriate group to examine.

I calculate the respondent's BMI, the main variable of interest in this analysis, using measures of height and weight collected by Add Health field interviewers in waves 2–4 and self-reported information in wave 1 when measured values are not available.³ Add Health contains a rich set of mental health outcomes, including information on counseling, suicidal ideation, suicide attempt, injury from suicide attempt as well as a subset of questions from the Center for Epidemiologic Studies Depression Scale (CES-D), which I standardized to have mean of zero and standard deviation of one.^{4,5,6} The CES-D is an established tool to measure symptoms of depression and has been tested in multiple settings for validity and reliability (Lewinsohn et al., 1997; Radloff, 1977). Suicidal ideation can be used as a useful measure of utility. One theory is that a fully rational individual who wants to commit suicide has a negative expected lifetime utility (Hamermesh and Soss, 1974). Another theory is that

³Estimates with obesity (BMI>30) and waist circumference available upon request and have similar conclusions. Results based on obesity are not presented because the statistical methods used assume continuous treatment. Also, empirical evidence suggests the effects on mental health are across the weight distribution, so dichotomizing BMI overstates the effect of obesity on mental health; the instrument affects mental health not just through obesity but through variation in BMI within obese and non-obese categories. Effects across the BMI distribution is consistent with the research on wages, particularly for women (Gregory and Ruhm, 2011). Results based on waist circumference are not presented because the genetic index is designed for BMI rather than waist circumference.

⁴Completed suicide information is not yet available at this time, because the genetic information that serves as the instrument was collected in the most recent wave of the Add Health data. Only 2.3% of respondents report a suicide attempt in the past year, and less than 1% injured themselves during an attempt. This lack of variation in these outcomes requires a much larger sample to detect the effect of weight, so estimates on these outcomes are not presented here, but available upon request.

⁵Counseling is based on the question: "In the past year, have you received psychological or emotional counseling?" Suicidal ideation is based on the question: "During the past 12 months, did you ever seriously think about committing suicide?" The depression index is standardized to be mean 0, standard deviation 1. Based on how often the following were true in the past week: You were bothered by things that don't usually bother you; You felt that you could not shake off the blues, even with help from your family and your friends; You felt you were just as good as other people; You had trouble keeping your mind on what you were doing; You felt depressed; You felt that you were too tired to do things; You enjoyed life; You felt sad; You felt that people disliked you. Each question takes on a value of one to three, and the index is the sum of the values.

⁶The pair-wise correlations between mental health outcomes examined here are: corr(suicidal ideation, counseling)=0.189; corr(suicidal ideation, depression index)=0.305; corr(counseling, depression index)=0.173.

professed suicidal ideation can serve as an expression of low utility (unhappiness), perhaps in order to extract sympathy and resources (Cutler et al., 2001). To examine potential mechanisms, I use information on respondents' own sense of attractiveness as a measure of self-image and interviewer-rated attractiveness as a proxy for stigma.⁷

An important characteristic of the Add Health study is the sampling design. Add Health randomly sampled clusters of high schools and collected data from a subsample of students in those schools. This sample design enabled Add Health to develop data about school environment and peer networks. Add Health oversampled certain subpopulations of interest, including the children of college-educated black adults and individuals with physical disabilities, who may have a relatively small presence in a school. Generally, when a study uses a sample design that is not a simple equal probability sample, analyses should account for both clustering and oversampling.

In this study, I cluster at the school level but use no sample weights. I cluster at the school level as opposed to the level of treatment assignment (individual or family) because all individuals and almost 80 percent of families are completely subsumed within schools. By clustering at the larger level, I account for the correlation within the smaller clusters, and according to Cameron and Miller (2015) "[t]he consensus is to be conservative and avoid bias and use bigger and more aggregate clusters." Add Health personnel recommended that no available sample weights be used in the analysis of the BMI Genetic Risk Score sample (National Longitudinal Study of Adolescent to Adult Health, personal communication, 2015). Additionally, if the sample weights are unrelated to the outcomes conditional on controls, unweighted and weighted regressions are both consistent, but weighting can reduce precision (Solon et al., 2015). The results from this analysis are thus local to the sample population and are not representative at the national level.

Instrument – BMI Genetic Risk Score

While the literature on the causal effect of weight on mental health is thin, many studies have used instruments for BMI to look at the effect of weight on other outcomes, such as employment and wages (Averett, 2014; Averett and Korenman, 1996; Cawley, 2004). Most of these studies use the BMI of a biological relative to instrument for the respondent's BMI. The relative's BMI serves as a proxy for shared genetics that are related to weight (Averett, 2014). Researchers have started to use respondents' own genetic information as collecting and analyzing genetic samples has become more common in social science datasets (Böckerman et al., 2017; Tyrrell et al., 2016; von Hinke et al., 2016).

The BMI Genetic Risk Score, the instrument in this study, is based on 31 Single Nucleotide Polymorphisms⁸ previously identified as associated with increased BMI by Speloites et al.

⁷Respondent-rated attractiveness is based on the question "How attractive are you?" Takes value 1 if response "not at all attractive" or "slightly attractive", 0 if "moderately attractive" or "very attractive". Interviewer-rated attractiveness based on the field interviewers answer to the question "How physically attractive is the respondent?" Takes value 1 if response "very unattractive", "unattractive" or "about average"; 0 if "moderately attractive" or "very attractive".

⁸There are four nucleotides: cytosine, adenine, guanine, and thymine. DNA (deoxyribonucleic acid) is composed of a sequence of these nucleotides. If a nucleotide occurs in a particular position with at least moderate frequency (more than one in one hundred people) then this location-nucleotide combination is a SNP (for instance, if more than one percent of the population has cytosine in a certain location in their DNA, that would be a SNP) (Nature.com, 2014).

(2010). The score can range from 0 to 62, as a person can have 0, 1, or 2 risky alleles for each of the 31 SNPs.⁹ To determine which SNPs were associated with increased BMI, Speloites et al. (2010) first performed a meta-analysis of 46 genome-wide association studies. She and her co-authors then performed their own analysis on the 19 SNPs from the meta-analysis and found an additional 12 SNPs associated with BMI. In wave 4, Add Health collected the genetic information used to calculate the BMI Genetic Risk Score using saliva samples. Saliva samples were collected from 1,946 respondents, 1,886 have valid genetic data, and 1,733 individuals have all information required for valid BMI Genetic Risk Scores, though not all respondents with BMI Risk Scores were present in all waves of data. Of the 1,733 respondents with valid BMI Genetic Risk Scores, there are 595 sibling pairs, 30 sibling trios, 1 sibling quad, and 449 stand-alone respondents. The Siblings Pair dataset is based on approximately 1,600 individual from whom genetic samples were collected, which is described in the BMI Genetic Risk Score documentation (National Longitudinal Study of Adolescent to Adult Health, 2017).

3. Method

I obtain estimates of correlation between BMI and mental health using ordinary least squares models. While OLS models are important to see associations between weight and mental health, they can suffer from bias in the form of reverse causality (mental illness causes weight gain) and unobserved confounders such as self-control. Von Hinke et al. (2016) carefully outline an argument for the credibility of a genetic index as an instrument for weight based on the instrumental variable assumptions: independence, exclusion, non-zero correlation of instrument and treatment, and monotonicity. I address the same issues and concerns in a new context: mental illness. I combine the discussion of independence and exclusion in terms of “validity”; likewise, I combine monotonicity and non-zero correlation of instrument and treatment as “strength”. I discuss the empirical evidence regarding the IV assumptions in the Results section.

While the issue of instrument strength can be empirically examined, instrument validity – that the BMI Genetic Risk Score is only related to mental health by affecting weight – is not as clear-cut. One benefit of genetic instruments involves the concept of Mendelian randomization; genetics are randomly passed from parents to children (Lawlor et al., 2008). Therefore, after controlling for parental genetics, children’s genetics are as-if randomly assigned. While I am unable to condition on parents’ genes, I do control for parental characteristics including self-reported obesity. Potential threats to the validity assumption that I cannot investigate with the data in Add Health include pleiotropy (when a gene influences multiple characteristics) and biological mechanisms, though I address these issues with evidence from existing research.

⁹As of June 14, 2017 the online documentation for the BMI Genetic Risk Score is incorrect (http://www.cpc.unc.edu/projects/addhealth/documentation/restricteduse/datasets/GRS_BMI.pdf). It states that the score is standardized (mean 0, standard deviation 1) by race. However, based on communication with Add Health staff, this description applies to how the data were used in Domingue et al. (2014) (National Longitudinal Study of Adolescent to Adult Health, personal communication, 2016). The fact that the BMI Genetic Risk Score is not standardized is confirmed in a frequency table at the bottom of the online documentation.

In terms of pleiotropy, if a SNP that is related to weight also is directly related to mental health outcomes, the SNP would not be a valid instrument. As I cannot examine this concern with the data available, I follow von Hinke et al. (2016) by checking if the SNPs in the BMI Genetic Risk Score are also found to be related to mental illness in GWAS. Based on literature reviews, the SNPs in the BMI Genetic Risk Score are not directly linked to mental illness such as major depression, bipolar disorder, or schizophrenia (Flint and Kendler, 2014; Gurung and Prata, 2015; Ripke et al, 2013). In aggregate, if BMI affects mental health, then there is an indirect link between the SNPs and mental health; however, with current information on genetic mechanisms, there is not a definitive method to disentangle a potential direct effect of the SNPs on mental health from an indirect effect through weight. While not much is known about the biological mechanisms of SNPs generally, the current evidence indicates that the SNPs in the BMI Genetic Risk Score impact weight through mechanisms that are consistent with the IV assumptions, such as energy homeostasis (involved in hunger) and gastric inhibitory polypeptide (involved in insulin secretion) (Locke et al., 2015).

To estimate the causal effect in the IV approach, I estimate two stage least squares (2SLS) regression models. The estimating equations are:

First Stage:

$$\text{BMI}_{it} = \alpha_0 + \alpha_{\text{riskscore}} \text{GeneticRiskScore}_i + \alpha_x X_{itjk} + \zeta_{itjk}$$

Second Stage:

$$Y_{it} = \beta_0 + \beta_{\text{BMI}} \widehat{\text{BMI}}_{it} + \beta_x X_{itjk} + \varepsilon_{itjk},$$

where each observation occurs at time t for individual i , who is a member of family j , and sampled from school k . Therefore, each observation nests within individuals who are nested within both schools and families, and most families are nested within schools (78%).

In the first stage, the endogenous regressor of interest (BMI) is regressed on the instrument (BMI Genetic Risk Score). In the second stage, the outcome (depression index, counseling, suicide ideation) is regressed on the fitted value of BMI from the first stage. I also estimate reduced form models in which I regress mental health outcomes directly on the BMI Genetic Risk Score. These reduced form models relax the functional form assumptions. Though these models have no clear policy implication, they are included as standard good form. To estimate the mechanism by which BMI affects mental health, I estimate the same 2SLS models and use interviewer- and self-rated attractiveness as the outcomes. In all models, I control for individual and area characteristics to increase precision. Controls include age, age squared, native born, wave, race/ethnicity, parent education, self-reported parent obesity (continuous measure not available), county unemployment rate, gender, intelligence (picture vocabulary and number recall).

4. Results

As previously mentioned, the credibility of the instrument rests on two main assumptions. The first assumption is that the instrument is highly correlated with BMI. By construction, the BMI Genetic Risk Score is correlated with BMI. Further, this assumption can be tested empirically. As seen in Figure 1, the BMI Genetic Risk Score and BMI have a strong positive relationship. The mean BMI Genetic Risk Score in the sample is 27 and the standard deviation is 3.44, so for every five SNPs (or about 1.4 standard deviations) that are associated with increased BMI, BMI increases by 2 units. For a 6-foot tall individual, a BMI increase of 2 units corresponds to a weight gain of 15 pounds. Additionally, I present first stage F-statistics from the two stage least squares models. All F-statistics are above the traditional cut-off of 10 for weak instruments (Stock and Yogo, 2005), and many F-statistics are in the 20s and 30s, indicating that the BMI Genetic Risk Score is a strong instrument for BMI.

Figure 1 also presents evidence of monotonicity. While the relationship between the BMI Genetic Risk Score and BMI is somewhat noisy, the relationship appears largely linear and increasing; a higher BMI Genetic Risk Score is associated with higher average BMI. The one exception is for BMI Genetic Risk Score from 21 to 23. This is likely not caused by an actual violation of the monotonicity assumption, but due to the fact that the mean BMI for each bin is fairly imprecise due to few observations. As a robustness test, those with BMI Genetic Risk Score below 24 are excluded.¹⁰

Linearity between outcomes and the predicted BMI based on first-stage regressions (Appendix Figures A3–A5) can be assessed from appendix graphs. These graphs are important because the effect of predicted BMI on outcomes is estimated in the second stage of 2SLS models and assumes linearity. Relationships appear linear, though with limited data points. From Appendix Figure A3 there is some indication that a relationship between predicted BMI and suicidal ideation is largely caused by effects at low predicted BMI. However, very few respondents have predicted BMI below 24 (3%), and so estimates are unlikely to be driven by changes at low predicted BMI. This is reflected in the fact that probability of suicidal ideation at other predicted BMI values lay close to the fitted line. Additionally, effect sizes are largely robust to dropping respondents with BMI Genetic Risk Scores under 24, which includes all respondents with low predicted BMI.¹¹

Table 1 presents first stage estimates of the effect of the BMI Genetic Risk Score on BMI.¹² There are several important takeaways. First, estimated effects of the BMI Genetic Risk Score are stable across racial and gender groups: a unit increase in the BMI Genetic Risk Score increases BMI by approximately 0.3 units. Second, all F-statistics are over the traditional cut-off of 10, and often above 20. Third, the marginal R-squared, the additional

¹⁰First stage estimates increase, while reduced form estimates are substantively the same but are not statistically significant due to loss of precision (results available upon request).

¹¹See footnote 10 for discussion about results when respondents with BMI Genetic Risk Scores below 24 are excluded.

¹²Ideally, the data would have sufficient sample size to include household fixed effects. In this case, the identifying variation would be based solely on differences between siblings. However, including household fixed effects leaves very little variation and uses much of the statistical power. While the estimated relationship is fairly stable, F-statistics drop to below ten, and the partial R^2 falls to about 1%.

percent of total variance in BMI explained by controlling for the BMI Genetic Risk Score, ranges from 2–5% (in square brackets).

The second IV assumption – validity or that the instrument is unrelated to the outcomes except through BMI – cannot be tested directly. The two ways this assumption could fail are: (1) the effect of the instrument on the outcome operates through a mechanism besides BMI (exclusion) and (2) some third factor causes changes in the instrument and the outcome (independence). I empirically assess both of these possible threats to validity by examining the relationship between the BMI Genetic Risk Score and other predictors of depression.

To assess the relationship between the BMI Genetic Risk Score and other characteristics, I compare respondents with high and low genetic risk. This is a common approach to investigate the validity of an instrument (McClellan et al., 1994). Appendix Table A2 contains the mean and standard error of the mean of characteristics that are related to BMI and mental health for those with low (below the median of 27) and high (above the median) BMI Genetic Risk Scores in wave 1. In addition to race (addressed in regards to respondent characteristics below), the only significant difference among parental characteristics is that those with higher BMI risk are more likely to have obese parents, which is expected because parents and children share genes. A concern with the heritability of risk of high BMI is that parents of heavier children are heavier and thus might earn less. In this case, the BMI Genetic Risk Score could violate the validity assumption by impacting heavier children's mental health through socioeconomic status. However, childhood household income and parents' education are similar across levels of BMI risk.

Another concern is that parental weight can impact parental mental health, and depressed parents could affect their children's mental health. The concern is that shared genes related to BMI result in correlated child/adult weight, and that higher adult weight could affect child mental health. The effect of parent mental health, net of the effect of parent weight, on child mental health would not violate the IV assumptions. In order to prevent bias from shared genetic disposition for high BMI, I control for a measure of parental obesity in all models as well as parental education. Ideally, I would control for parent mental health to increase precision, but such measures are not available in Add Health.

Appendix Table A2 also shows differences in picture vocabulary test and grade point average that are statistically significant, but the differences are quite small in magnitude. A difference of 2.3 in picture vocabulary test is only 1/6 of a standard deviation, and the difference in GPA is less than 1/10 of a standard deviation. Additionally, academic achievement is endogenous (affected by weight), and so differences in GPA are plausibly a consequence of higher BMI's effect on mental health (Sabia and Rees, 2015). There is a four percentage point difference in prevalence of native born, but both groups are almost completely born in the United States. I include picture vocabulary and native born in all models. Importantly those with higher BMI risk have higher BMI but very similar heights; estimated effects of BMI will be based on variation in weight instead of height. Whites and blacks differ in base rates of the SNPs of interest, because most genetic studies of the effect of SNPs on BMI use white samples as well as due to potential population stratification (different prevalence of genes caused by historic separation of the ancestors of white and

black individuals). Thus, I perform all analyses separately for whites and blacks, as suggested by von Hinke et al. (2016).

Appendix Table A3 shows a composite measure of the potential bias from violating the validity assumption for the black and white pooled sample. First, predicted outcomes are based on regressing a mental health measure on observable characteristics. Then the predicted value for the mental health outcome, which is a linear combination of observable characteristics, is regressed on the genetic risk score controlling for parent obesity. The estimates in Table A3 are the association between the BMI Genetic Risk Score and variation in observable characteristics predictive of mental health issues.

Though the relationships are statistically significant, the correlations are very small. For suicidal ideation, the correlation is 1/3 the reduced form estimate and 1/10 the two stage least squares estimate of the effect of BMI, and thus any bias from violating the validity assumption is unlikely to explain the results for suicidal ideation. The other correlations are also very small and comparable in magnitude to the reduced form estimates, which are not statistically significantly different from zero. Importantly, once the analysis is stratified by race (Appendix Table A4 and Appendix Table A5) the estimates are much smaller and many of the associations are not statistically significant; the significant relationships found in Appendix Table A3 load on race. Since the main results are also stratified by race, the balance in Appendix Tables A4 and A5 further mitigate concerns about the validity assumption.

Since results from reduced form linear probability models are similar to the marginal effect obtained from probit models, I present linear probability models, which are easier to interpret.¹³ Tables 2–4 present ordinary least squares (left panel) associations and two stage least squares (right panel) estimates of the effect of BMI on mental health outcomes. Ordinary least square estimates are the association between BMI and the outcome. The 2SLS estimates are the causal effect of a one-unit increase in BMI on the outcome. Comparing OLS and 2SLS estimates can reveal potential bias from using an endogenous measure of treatment.¹⁴ However, differences could also be due to the fact OLS correlations are average treatment effects (ATE) and IV estimates are local average treatment effects (LATE) based only on variation in BMI due to genetic predisposition.¹⁵ If high BMI caused by genetics is more difficult to reverse or prevent, then intuitively the LATE in this context (effect of BMI on mental health specifically caused by genetics) would be larger than the correlation.

Table 2 presents OLS and 2SLS estimates of the effect of BMI on suicide ideation. Effects of suicidal ideation are of particular interest, because it is the most severe measure of mental health problems I examine. From the right panel, BMI has a statistically and economically significant effect on suicidal ideation. Consistent with research on BMI's effect on labor

¹³For example, see Appendix Table A6 for LPM and Appendix Table A9 for probit marginal effects.

¹⁴The direction of the bias is ambiguous ex ante. The direction of omitted variable bias depends on the correlation between the omitted variable with both the treatment and the outcome, while bias from reverse causality – mental health affecting weight – could be up or down due to the effect of depression on diet (American Psychiatric Association, 1994).

¹⁵While OLS estimates are average treatment effects and 2SLS estimates are local average treatment effects, the 2SLS estimates are based on variation across the BMI distribution (see Figure 1).

market outcomes and mental health, the effects are concentrated in women and particularly white women (Averett, 2014; Sabia and Rees, 2015). Each unit increase in BMI causes a 0.87 percentage points increase in suicidal ideation, off a mean of 9.7 percent. The effect is almost double for white women at 1.4 percentage points. For women and whites, the estimated effects are much larger than the correlations. The estimated correlations and estimated effects of BMI are smaller and not statistically significant for males and blacks. However, IV point estimates are still positive and of moderate magnitude for all groups; a one-unit increase in BMI causes half a percentage point increase in suicidal ideation for men. Additionally, the estimate for black women is almost as large as for white women but is not statistically significant, perhaps due to a smaller sample size in that subpopulation.

While suicidal ideation is a fairly severe indication of depression, changes in the depression index and use of counseling services can reflect effects of weight on people who have relatively good mental health. The estimated effect of BMI on the depression index and on use of counseling service in the past year are small and exclusively not statistically significant. The largest 2SLS estimate for the effect of BMI on the depression index (Table 3) is 0.031 for white females. While the estimated effect for women is much larger than the estimated correlation of 0.011 based on OLS, it is still a small fraction of the standard deviation of the depression index ($SD=1$). A 5'4" women who weighs 130 pounds (~22 BMI) could increase her weight by 50 pounds (~8 BMI), and the resulting increase in her depression index would be much smaller than a standard deviation in this outcome ($8 \text{ BMI} \times 0.0308 \approx 0.25$). Results for a dichotomized depression score (above vs below average) also result in small estimates that are not statistically significant (results available upon request).

The results for the effect on using counseling services (Table 4) tell a similar story, though men are more responsive to weight for this outcome. Again the IV estimates are of greater magnitude than the OLS estimates, but both 2SLS and OLS estimates are generally small compared to the standard deviation of the outcome ($SD=0.279$). However, the effects on men are of economically significant magnitude but are imprecise. The largest estimated effect is for black males, for whom a one-unit increase in BMI would cause a not statistically significant 1.58 percentage point increase in the probability of using counseling services in the past year. The estimated effect of BMI on counseling is often of the opposite sign from the correlation – indicating potential bias of an endogenous measure of BMI – though all estimated effects and correlations are not significant.

The lack of effect of BMI on these two indicators of less severe¹⁶ mental health problems – depression index and counseling – suggests that those of fair-to-good psychological well-being are unresponsive to weight. In order to test this empirically, I examine the effect of BMI on suicidal ideation for those with high depression index scores (discussed below). Since the data only contain information on the extensive margin of counseling, I am unable to examine the effect of BMI on frequency of counselling. If the intensive margin is

¹⁶This not to say depression cannot be a serious health concern, but that use of counseling services at the extensive margin – the marginal person goes from no use to some – and the topics in the depression index – such as inability to shake off the blues or increased irritability – intuitively fall to the left of suicidal ideation in terms of severity.

responsive to BMI, these estimates could understate the effect of BMI, particularly for people in worse mental health.

Appendix Tables A6–A8 contain estimates of reduced form IV models. Though these models have no clear policy implication, they are included for completeness. In these models, the outcome is regressed on the instrument (BMI Genetic Risk Score) along with a set of controls. The estimates represent the effect of a one-unit increase in the BMI Genetic Risk Score or the presence of one more allele associated with high BMI on mental health outcomes. The results are consistent with the 2SLS regressions above. The BMI genetic risk score has a statistically significant and economically meaningful effect on suicidal ideation, particularly for white females, but not for the black sample or on men. The estimated effects on counseling and the depression index are small and not significantly different from zero.

I also ran a number of analyses to examine heterogeneous effects. Since statistically significant effects of BMI are found only on suicidal ideation, this suggests that BMI influences people who are already in relatively worse mental health. In order to examine this empirically, I estimate the effect of BMI on suicidal ideation for those with above average depression index scores (the index is standardized to mean 0). In Appendix Table A13 are results for the high depression index subsample as well as the full sample. Results are consistent with the hypothesis that individuals with worse mental health are more responsive to weight: Point estimates are much high for the high depression index subsample, and effects are still concentrated in white women.

Two other questions of interest remain: Is there heterogeneity by age and how does the BMI Genetic Risk Score compare to using the traditional instrument of sibling BMI? Results for adults (age>18) are compared to the full sample in Appendix Tables A14–A16. Again, BMI only has statistically significant effects on suicidal ideation, and the effect continues to be concentrated in white women. Interestingly, the effect of BMI on suicidal ideation is larger for adults, which should address concerns that effects are driven by endogenous misreporting of height and weight in wave 1.

In Appendix Tables A17–A19, I compare estimates using sibling BMI as an instrument to estimates using the BMI Genetic Risk Score. In order to have more comparable analyses, I limit the sample to respondents with BMI Genetic Risk Scores and at least one sibling with BMI information.¹⁷ When restricting the sample, estimates based on the BMI Genetic Risk Score are less precise and similar to the main analysis, except the effects on black women are somewhat unstable.

Pooled gender estimates for suicidal ideation are not statistically significant using sibling BMI as an instrument, because effects for males have decreased. Estimates using the BMI Genetic Risk Score show a moderate if imprecise increase in suicidal ideation for men, but instrumenting with sibling BMI results in reversal of sign – a moderate decrease in suicidal ideation. Effects for white women are similar between instruments, but effects are only

¹⁷In order for an observation to be included, the respondent and a sibling must have BMI information in the same wave. If a sibling pair has BMI information in one wave of data collection, they will be included for that wave. If one sibling is missing information in a different wave, they will not be included in that wave.

statistically significant using sibling BMI in the restricted sample. The estimated effect of weight on the depression index is larger and becomes significant for white women using sibling BMI, and the effect for black men is large but very imprecise. Regardless of instrument, there is no significant effect on counseling use, and many estimates remain precise zeros. Analysis on unrestricted samples are available in Appendix Tables A20–A22. When using sibling BMI as an instrument, results are similar but smaller for the full sample, and effects on the depression index are no longer statistically significant for white women.

5. Mechanism of BMI Effect on Mental Health

I now explore potential mechanisms through which weight affects mental health. The psychological literature notes three possible mechanisms by which weight can cause worse mental health: body image, disrupted social relationships including stigma, and health problems. First, body image may be important in societies with beauty premiums; being overweight can lower self-esteem and self-worth as the feeling of being attractive decreases (Granberg, 2011). Second, stigma towards heavier people can contribute to depression. Research indicates that heavier people, particularly women, have less successful marriage/romantic partner market outcomes (Averett and Korenman, 1996) and are treated differently in any number of other social interactions. Lastly, high BMI can decrease mental well-being through weight's detrimental effect on physical health. Obesity causes cardiovascular disease and diabetes (Colditz et al., 1995; Hubert et al., 1983), which in turn can decrease the ability to engage in enjoyable activities and to effectively take care of oneself. Comorbidities increase stress through the financial burden of increased medical care costs. For instance, the lifetime medical cost of developing diabetes can reach \$100,000 (Zhuo et al., 2014).

It is important to note that stigma and self-image are two different issues. Stigma harms mental health primarily through worse treatment, such as difficulty making friends (Granberg, 2011). While stigma could affect self-image if heavier people know they are treated differently because of their weight, stigma may manifest itself in ways that do not affect self-image or in ways that the heavier person does not realize are related to weight/attractiveness. For instance, if stigma plays a role in the effect of weight on labor market outcomes for women such as in Cawley (2004), women may be unaware that their weight and appearance are factors. Therefore, stigma may impact a person and their mental health but not necessarily through self-image. I am using interviewer-rated attractiveness of the respondent as a proxy for stigma.

Given the mixed evidence of weight's effect on mental health, investigating the impact of weight on stigma and self-image is important; if these potential mechanisms are unaffected, the evidence that weight harms mental health would be further weakened. Additionally, determining the mechanism by which weight affects mental health can help target policies. For instance, groups that suffer from poor body image could be more responsive to mental health interventions. While broader informational campaigns on the social effects of weight may increase awareness among those who stigmatize obesity, and reduce judgmental behavior and statements. I hypothesize that health problems are unlikely to be a driving factor as respondents are quite young, 12 to 34 years old.¹⁸ Therefore, I examine body

image and stigma as viable mechanisms for this sample. Table 5 contains estimates of the effect of BMI on one's body image, and Table 6 contains estimates of the effect of BMI on the interviewer's perspective of a respondent's attractiveness, which I use as a proxy for stigma. I use this measure to proxy for stigma as I assume that weight's role in what is considered attractive is an important part of the stigma associated with weight.

For white females, the group in which the effect of weight on mental health is concentrated, the mechanism appears to be social stigma. Interviewer-rated attractiveness is highly responsive to weight for this group, while self-rated attractiveness is largely unaffected; this suggests that stigma and differential treatment may manifest in ways that white women are unaware relate to weight/attractiveness. Additionally, women's self-image could be increasingly protected from the effects of weight due to both public service and marketing campaigns promoting positive body image, which have tended to focus on women. For instance, almost 20 years ago the American Dietetic Association began promoting body size acceptance (Parham, 1999), while the Dove Campaign For Real Beauty started in the mid-2000s (Bahadur, 2014). Such campaigns may help protect self-image, but could also reduce reporting of poor self-image due to weight. If a shift towards body acceptance prevents women from reporting poor self-image, the measure used in this study may not capture true problems with self-image.

A unit increase in BMI causes a 2.35 percentage point increase in the probability of the interviewer finds a white female unattractive. In contrast, the point estimate for the effect of BMI on self-rated attractiveness is only 0.32 percentage points. For neither outcome do white males have statistically significant results, which is consistent with white males' earnings and mental health being largely unaffected by weight, as shown in this research and other studies examining mental health and labor market outcomes (Averett, 2014; Sabia and Rees, 2015).

Black males' self-image and exposure to social stigma are both responsive to increased BMI. The meaningful effect of BMI on mental health for white females and lack of effect for black males in light of the large effects on potential mechanisms is somewhat surprising. While these mechanisms do not manifest themselves in statistically significant effects on the measures of mental health examined here for black males, I estimate BMI has a meaningful but imprecise effect on counseling for this group. Self-image and stigma may also serve as mechanisms for mental health outcomes not examined in this study for this group. Another possible explanation for this difference between black males and white females is that feeling attractive and being perceived as attractive may have a greater effect on the mental health of white women.

¹⁸Appendix Table A23 provides confirmation. First, the prevalence of poor health and diabetes is very low; half a percent of respondents report being in poor general health and two percent have diabetes. Second, the effect of BMI on self-described health and on diabetes (only available in waves 3 and 4) are very small. The exception is for the moderate protective effect of BMI on black respondents', which is the opposite direction one would expect for a mechanism between weight and mental health. Importantly there is no statistically significant effect on diabetes, an objective health outcome highly related to weight.

6. Conclusion

Overall, I conclude that the evidence on the causal relationship between weight and mental health is mixed. While my findings do not indicate that weight impacts use of counselling services or a depression index, I find a statistically and economically significant effect of weight on suicidal ideation. This effect is concentrated in certain groups, particularly white women. A concentration of effects of weight in white women is consistent with the research on labor market outcomes (Averett, 2014; Cawley, 2004) and the smaller literature on the effect of weight on mental health (Bjørngaard et al., 2015; Sabia and Rees, 2015). However, my findings are less strong than the previous research on mental health that uses sibling BMI as an instrument for own BMI (Sabia and Rees, 2015). Based on respondent and interviewer opinions of respondent attractiveness, social stigma is a possible mechanism by which high BMI affects mental health for white women.

The effect on suicidal ideation is statistically significant at a cut-off of 0.10. While there are many regressions investigating heterogeneous effects by race and gender, there are three main hypothesis tests on the pooled sample (one for each mental health outcome). If there were only statistically significant effects in one subsample and none for the pooled sample, then there may be concerns about multiple comparisons (that a significant finding is due to the combination of many tests and type I error). However, after accounting for three comparisons, the adjusted p-value for the effect of BMI on suicidal ideation for the pooled sample is slightly above the traditional cut-off.¹⁹

These findings add to the scant literature that uses econometric methods to examine the causal effect of weight on depression. The effect of weight on mental health also has important implications for the total cost of the ongoing obesity epidemic in the United States. This causal relationship could serve as an important mechanism of weight's effect on labor market outcomes. Additionally, this paper is one of the first economic studies to use measures of genetic information to instrument for BMI. Another contribution is the empirical examination of mechanisms by which weight impacts mental health outcomes, which can have policy implications for educating society about the consequences of stigmatizing weight.

This study is limited to a young sample under 35 years old, so future work should consider the effects of weight on an older population as well as how potential mechanisms operate differently for this group. As more genetic data respondents become available in survey data, particularly in data sets with parent and offspring pairs, the credibility of genetic indices as

¹⁹Using the procedure discussed in Tukey et al. (1985), the adjust p-value for the 2SLS effect on suicidal ideation for the pooled sample is 0.105. The adjusted p-value is

$$p_{adj} = 1 - (1 - p_{unadj})^{\sqrt{k}} = 1 - (1 - 0.062)^{\sqrt{3}} = 0.105,$$

where p_{unadj} is the unadjusted p-value and k is the number of tests (3). This adjustment is appealing because it intuitively balances type I and type II error. This method takes into account multiple hypothesis tests and results in adjusted p-values that approach 1 as the number of tests becomes large. However, it is less conservative than the Bonferroni adjustment of multiplying p-values by k , which can result in adjusted p-values over 1.

instruments will continue to improve, because offspring genetic risk is as-if randomly assigned after fully controlling for parental genetic risk. This advancement will further improve genetic information as instruments. Larger data sets with genetic information will provide the required statistical power to analyze the effect of weight on more severe but less common outcomes such as suicide attempts and completed suicides.

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Appendix

Appendix Figure A1.

Trends in obesity rates among adults by race, men

Source: Fryar et al., 2012. Data: NHANES III; Continuous NHANES (1999–00 through 2009–10)

Appendix Figure A2.

Trends in obesity rates among adults by race, women

Source: Fryar et al., 2012. Data: NHANES III; Continuous NHANES (1999–00 through 2009–10)

Appendix Figure A3.

Relationship between Suicidal Ideation and Predicted BMI

Data Source: The National Longitudinal Study of Adolescent to Adult Health

Predicted BMI range restricted due to small numbers of observations at extreme values

Appendix Figure A4.

Relationship between Counseling and Predicted BMI

Data Source: The National Longitudinal Study of Adolescent to Adult Health

Predicted BMI range restricted due to small numbers of observations at extreme values

Appendix Figure A5.

Relationship between Depression Index and Predicted BMI

Data Source: The National Longitudinal Study of Adolescent to Adult Health

Predicted BMI range restricted due to small numbers of observations at extreme values

Appendix Table A1

Summary Statistics and Data Availability by Wave

	Availability				Mean	(SD)
	Wave 1	Wave 2	Wave 3	Wave 4		
BMI Genetic Risk Score				X ¹	27.0	(3.4)
BMI	X ²	X	X	X	25.6	(6.8)
Suicidal Ideation	X	X	X	X	0.097	(0.296)
Depression Index	X	X	X	X	0.0	(1.0)
Counseling Use	X	X	X	X	0.085	(0.279)
Self-Rated Unattractiveness			X	X	0.238	(0.426)
Interviewer-Rated Unattractiveness	X	X	X	X	0.529	(0.499)
Father Obese	X ³				0.117	(0.321)

	Availability				Mean	(SD)
	Wave 1	Wave 2	Wave 3	Wave 4		
Mother Obese	X ³				0.229	(0.420)
Father Ed. (HS Grad or Less)	X ³				0.488	(0.500)
Mother Ed. (HS Grad or Less)	X ³				0.556	(0.497)
Native Born	X ¹				0.961	(0.194)
Black	X ¹				0.351	(0.477)
White	X ¹				0.495	(0.500)
Hispanic	X ¹				0.108	(0.310)
Gender	X ¹				0.479	(0.500)
Age	X	X	X	X	21.1	(5.5)
Intelligence (Number Recall)				X ³	4.065	(1.525)
Intelligence (Picture Vocab)	X ³				0.097	(0.296)

¹Time-invariant;

²Self-reported;

³Treating as time-invariant

Standard deviations in parentheses (SD)

Data: The National Longitudinal Study of Adolescent to Adult Health

Appendix Table A2

Balance Between High and Low BMI Risk

	Low BMI Risk	High BMI Risk	Difference
<i>Parental Characteristics</i>			
Parent Income (\$1,000)	41.518 (2.223)	43.063 (1.559)	1.545 p=0.581
Less than HS Mother	0.171 (0.013)	0.167 (0.014)	-0.004 p=0.819
Less than HS Father	0.147 (0.015)	0.151 (0.016)	0.004 p=0.863
HS Grad Mother	0.382 (0.017)	0.393 (0.018)	0.012 p=0.639
HS Grad Father	0.336 (0.02)	0.348 (0.021)	0.012 p=0.686
Some Post HS Mother	0.195 (0.013)	0.213 (0.015)	0.019 p=0.362
Some Post HS Father	0.182 (0.016)	0.205 (0.018)	0.023 p=0.344
College Mother	0.197 (0.014)	0.177 (0.014)	-0.02 p=0.306
College Father	0.234 (0.018)	0.213 (0.018)	-0.021 p=0.404
More Than College Mother	0.056 (0.008)	0.049 (0.008)	-0.007 p=0.564

	Low BMI Risk	High BMI Risk	Difference
More Than College Father	0.102 (0.013)	0.085 (0.012)	-0.017 p=0.348
Obese Mother	0.209 (0.014)	0.247 (0.017)	0.038 [*] p=0.078
Obese Father	0.075 (0.009)	0.162 (0.014)	0.086 ^{***} p<0.001
Alcoholism Mother	0.031 (0.006)	0.037 (0.007)	0.006 p=0.523
Alcoholism Father	0.153 (0.013)	0.157 (0.014)	0.003 p=0.861
Diabetes Mother	0.030 (0.006)	0.036 (0.007)	0.005 p=0.568
Diabetes Father	0.055 (0.008)	0.063 (0.01)	0.008 p=0.527
<i>Intelligence and Academic Achievement</i>			
Picture Vocab Test	96.987 (0.472)	99.255 (0.520)	2.268 ^{***} p=0.001
Number Recall	4.027 (0.05)	4.096 (0.054)	0.069 p=0.350
Grade Point Average	2.768 (0.024)	2.695 (0.028)	-0.073 [*] p=0.048
Less Than High School	0.111 (0.01)	0.101 (0.011)	-0.011 p=0.480
High School	0.193 (0.013)	0.181 (0.014)	-0.012 p=0.539
Some Post HS	0.363 (0.016)	0.387 (0.018)	0.024 p=0.299
JR College	0.07 (0.008)	0.066 (0.009)	-0.004 p=0.754
College	0.168 (0.012)	0.171 (0.014)	0.003 p=0.866
More Than College	0.096 (0.01)	0.094 (0.011)	-0.001 p=0.922
<i>Demographics</i>			
Native Born	0.942 (0.008)	0.982 (0.005)	0.041 ^{***} p<0.001
White	0.407 (0.016)	0.6 (0.018)	0.193 ^{***} p<0.001
Black	0.432 (0.016)	0.257 (0.016)	-0.175 ^{***} p<0.001
Hispanic	0.100 (0.01)	0.119 (0.012)	0.019 p=0.205

	Low BMI Risk	High BMI Risk	Difference
<i>Body Measures</i>			
BMI (self-reported) (Wave 1)	22.216	23.393	1.176 ***
	(0.141)	(0.188)	p<0.001
Obese (self-reported) (Wave 1)	0.055	0.11	0.055 ***
	(0.007)	(0.011)	p<0.001
Weight (lbs) (Wave 4)	182.186	195.457	13.271 ***
	(1.556)	(2.004)	p<0.001
Height (inches) (Wave 4)	66.917	67.076	0.159
	(0.132)	(0.142)	p=0.413
<i>Mental Health Outcomes</i>			
Suicidal Ideation (Wave 1)	0.136	0.146	0.010
(past 12 months)	(0.011)	(0.013)	p=0.553
Counseling (Wave 1)	0.095	0.114	0.019
(past 12 months)	(0.009)	(0.011)	p=0.192
Depression Index (Wave 1)	6.372	6.220	-0.151
	(0.138)	(0.158)	p=0.469

Data: The National Longitudinal Study of Adolescent to Adult Health; Wave 1 (2001–02)

* p<0.1,

** p<0.05,

*** p<0.01

Standard errors in parentheses (SE)

Appendix Table A3

Association between BMI Risk Score and Predicted Outcomes; Black and White Pooled Sample

Suicidal Ideation	Counseling	Depression Index
0.0009 ***	0.0015 ***	0.0075 ***
(0.0002)	(0.0003)	(0.0026)
N=5517	N=5566	N=5559

* p<0.1,

** p<0.05,

*** p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level

Observables included: Age, age squared, native born, wave, race/ethnicity, parent education, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Second stage also includes parent obesity

Appendix Table A4

Association between BMI Risk Score and Predicted Outcomes; White Sample

Suicidal Ideation	Counseling	Depression Index
0.0005 *	0.0003	0.0003
(0.0003)	(0.0003)	(0.0029)

Suicidal Ideation	Counseling	Depression Index
N=2898	N=2915	N=2912

*
p<0.1,
**
p<0.05,

p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level

Observables included: Age, age squared, native born, wave, race/ethnicity, parent education, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Second stage also includes parent obesity

Appendix Table A5

Association between BMI Risk Score and Predicted Outcomes; Black Sample

Suicidal Ideation	Counseling	Depression Index
-0.0003	-0.0001	0.0007
(0.0005)	(0.0003)	(0.0049)
N=1840	N=1863	N=1860

*
p<0.1,
**
p<0.05,

p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level

Observables included: Age, age squared, native born, wave, race/ethnicity, parent education, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Second stage also includes parent obesity

Appendix Table A6

Reduced Form Regressions - Effect of Genetic Risk Score on Mental Health Outcomes; Black and White Pooled Sample

	Suicidal Ideation	Counseling	Depression Index
Panel A: Males and Females Pooled			
Genetic Risk Score	0.00271 *	0.000804	0.000583
	(0.00139)	(0.00122)	(0.00605)
	N=5517	N=5566	N=5559
Panel B: Males			
Genetic Risk Score	0.00190	0.00234	-0.00319
	(0.00170)	(0.00178)	(0.00825)
	N=2679	N=2708	N=2702
Panel C: Females			
Genetic Risk Score	0.00330	-0.000644	0.00412
	(0.00208)	(0.00190)	(0.00776)
	N=2838	N=2858	N=2857

*
p<0.1,
**
p<0.05,

p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Appendix Table A7

Reduced Form Regressions - Effect of Genetic Risk Score on Mental Health Outcomes; White Sample

	Suicidal Ideation	Counseling	Depression Index
Panel A: Males and Females Pooled			
Genetic Risk Score	0.00340 *	-0.000566	0.000485
	(0.00177)	(0.00163)	(0.00877)
	N=2898	N=2915	N=2912
Panel B: Males			
Genetic Risk Score	0.00212	0.000879	-0.0101
	(0.00256)	(0.00235)	(0.0131)
	N=1415	N=1425	N=1421
Panel C: Females			
Genetic Risk Score	0.00413 *	-0.00272	0.00950
	(0.00246)	(0.00271)	(0.0100)
	N=1483	N=1490	N=1491

* p<0.1,

** p<0.05,

*** p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Appendix Table A8

Reduced Form Regressions - Effect of Genetic Risk Score on Mental Health Outcomes; Black Sample

	Suicidal Ideation	Counseling	Depression Index
Panel A: Males and Females Pooled			
Genetic Risk Score	0.00203	0.00175	0.00685
	(0.00234)	(0.00221)	(0.0124)
	N=1840	N=1863	N=1860
Panel B: Males			
Genetic Risk Score	0.00118	0.00557	0.0119
	(0.00239)	(0.00358)	(0.0165)
	N=858	N=874	N=872
Panel C: Females			
Genetic Risk Score	0.00484	0.000802	0.00242

	Suicidal Ideation	Counseling	Depression Index
	(0.00331)	(0.00274)	(0.0163)
	N=982	N=989	N=988

*
p<0.1,
**
p<0.05,

p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Appendix Table A9

Reduced Form Probit Regressions Margins - Effect of Genetic Risk Score on Mental Health Outcomes; Black and White Pooled Sample

	Suicidal Ideation	Counseling
Panel A: Males and Females Pooled		
Genetic Risk Score	0.00262 *	0.000786
	(0.00135)	(0.00119)
	N=5517	N=5566
Panel B: Males		
Genetic Risk Score	0.00190	0.00235
	(0.00164)	(0.00169)
	N=2679	N=2708
Panel C: Females		
Genetic Risk Score	0.00337 *	-0.000795
	(0.00205)	(0.00183)
	N=2838	N=2858

*
p<0.1,
**
p<0.05,

p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Appendix Table A10

2SLS Standard Errors by Clusters, Suicidal Ideation

Panel A: Males and Females Pooled			
	Full Sample	Whites	Blacks
BMI	(0.00469)	(0.00508)	(0.00657)
	[0.00472]	[0.00498]	[0.00666]
	{0.00469}	{0.00508}	{0.00657}

Panel B: Males			
	Full Sample	Whites	Blacks
BMI	(0.00548)	(0.00727)	(0.00685)
	[0.00547]	[0.00726]	[0.00663]
	{0.00548}	{0.00727}	{0.00685}

Panel C: Females			
	Full Sample	Whites	Blacks
BMI	(0.00703)	(0.00795)	(0.0100)
	[0.00710]	[0.00799]	[0.0106]
	{0.00703}	{0.00795}	{0.0100}

Standard errors clustered at the school level in parentheses (SE)

Standard errors clustered at the school and family level in square brackets [SE]

Standard errors clustered at the school and individual level in braces {SE}

Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Appendix Table A11

2SLS Standard Errors by Clusters, Depression Index

Panel A: Males and Females Pooled			
	Full Sample	Whites	Blacks
BMI	(0.0198)	(0.0258)	(0.0349)
	[0.0203]	[0.0264]	[0.0362]
	{0.0198}	{0.0258}	{0.0349}

Panel B: Males			
	Full Sample	Whites	Blacks
BMI	(0.0260)	(0.0381)	(0.0459)
	[0.0266]	[0.0390]	[0.0475]
	{0.0260}	{0.0381}	{0.0459}

Panel C: Females			
	Full Sample	Whites	Blacks
BMI	(0.0267)	(0.0327)	(0.0498)
	[0.0277]	[0.0339]	[0.0519]
	{0.0267}	{0.0327}	{0.0498}

Standard errors clustered at the school level in parentheses (SE)

Standard errors clustered at the school and family level in square brackets [SE]

Standard errors clustered at the school and individual level in braces {SE}

Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Appendix Table A12**2SLS Standard Errors by Clusters, Counseling Use**

Panel A: Males and Females Pooled			
	Full Sample	Whites	Blacks
BMI	(0.00402)	(0.00478)	(0.00639)
	[0.00406]	[0.00480]	[0.00643]
	{0.00402}	{0.00478}	{0.00639}
Panel B: Males			
	Full Sample	Whites	Blacks
BMI	(0.00601)	(0.00678)	(0.0114)
	[0.00600]	[0.00672]	[0.0115]
	{0.00601}	{0.00678}	{0.0114}
Panel C: Females			
	Full Sample	Whites	Blacks
BMI	(0.00627)	(0.00885)	(0.00823)
	[0.00630]	[0.00896]	[0.00802]
	{0.00627}	{0.00885}	{0.00823}

Standard errors clustered at the school level in parentheses (SE)

Standard errors clustered at the school and family level in square brackets [SE]

Standard errors clustered at the school and individual level in braces {SE}

Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Appendix Table A13**2SLS - Effect of BMI on Suicidal Ideation, High Depression Subsample and Full Sample (Mean=0.097)**

High Depression Index Subsample				Full Sample		
Panel A: Males and Females Pooled						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.0244 ^{**} (0.00980)	0.0318 ^{***} (0.00965)	0.00909 (0.0120)	0.00876 [*] (0.00469)	0.0103 ^{**} (0.00508)	0.00490 (0.00657)
	N=2345	N=1059	N=901	N=5437	N=2857	N=1818
Panel B: Males						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.0128 (0.0103)	0.0162 (0.0147)	0.00933 (0.0152)	0.00546 (0.00548)	0.00552 (0.00727)	0.00335 (0.00685)
	N=1006	N=448	N=390	N=2648	N=1398	N=852

	High Depression Index Subsample			Full Sample		
Panel C: Females						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.0323 ^{**} (0.0145)	0.0415 ^{***} (0.0141)	0.0190 (0.0151)	0.0116 [*] (0.00703)	0.0140 [*] (0.00795)	0.0136 (0.0100)
	N=1339	N=611	N=511	N=2789	N=1459	N=966

*
p<0.1,

**
p<0.05,

p<0.01

High depression subsample restricted to those with scores above the mean of 0

Standard errors in parentheses (SE); Standard errors clustered at the school level

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Data: The National Longitudinal Study of Adolescent to Adult Health

Appendix Table A14

2SLS - Effect of BMI on Suicidal Ideation, Adults and All Ages (Mean=0.097)

	Adults Only			All Ages		
Panel A: Males and Females Pooled						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.0111 *** (0.00432)	0.0174 *** (0.00523)	0.00251 (0.00615)	0.00876 * (0.00469)	0.0103 ** (0.00508)	0.00490 (0.00657)
	N=3252	N=1711	N=1072	N=5437	N=2857	N=1818
Panel B: Males						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.00890 (0.00577)	0.0121 (0.00860)	0.00400 (0.00763)	0.00546 (0.00548)	0.00552 (0.00727)	0.00335 (0.00685)
	N=1593	N=848	N=501	N=2648	N=1398	N=852
Panel C: Females						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.0133 ** (0.00620)	0.0216 ** (0.00916)	0.00412 (0.00901)	0.0116 * (0.00703)	0.0140 * (0.00795)	0.0136 (0.0100)
	N=1659	N=863	N=571	N=2789	N=1459	N=966

*
p<0.1,

**
p<0.05,

p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Appendix Table A15

2SLS - Effect of BMI on Depression Index, Adults and All Ages (Mean=0.0)

Adults Only				All Ages		
Panel A: Males and Females Pooled						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.0118	0.000953	0.0214	0.00283	0.00267	0.0200
	(0.0184)	(0.0251)	(0.0397)	(0.0198)	(0.0258)	(0.0349)
	N=3280	N=1724	N=1083	N=5477	N=2871	N=1838
Panel B: Males						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.00120	-0.0257	0.0332	-0.00903	-0.0259	0.0329
	(0.0272)	(0.0476)	(0.0461)	(0.0260)	(0.0381)	(0.0459)
	N=1612	N=856	N=509	N=2671	N=1404	N=866
Panel C: Females						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.0238	0.0233	0.00666	0.0148	0.0308	0.0103
	(0.0259)	(0.0302)	(0.0598)	(0.0267)	(0.0327)	(0.0498)
	N=1668	N=868	N=574	N=2806	N=1467	N=972

*
p<0.1,**
p<0.05,***
p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Appendix Table A16

2SLS - Effect of BMI on Counseling Use, Adults and All Ages (Mean=0.085)

	<i>Adults Only</i>			<i>All Ages</i>		
Panel A: Males and Females Pooled						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.00255	0.000610	0.00258	0.00316	−0.000782	0.00493
	(0.00373)	(0.00491)	(0.00588)	(0.00402)	(0.00478)	(0.00639)
	N=3286	N=1726	N=1086	N=5484	N=2874	N=1841
Panel B: Males						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks

	<i>Adults Only</i>			<i>All Ages</i>		
BMI	0.00872	0.00840	0.00950	0.00768	0.00286	0.0158
	(0.00541)	(0.00734)	(0.0104)	(0.00601)	(0.00678)	(0.0114)
	N=1616	N=858	N=511	N=2677	N=1408	N=868

Panel C: Females

	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	-0.00392	-0.0108	0.00102	-0.00158	-0.00749	0.00235
	(0.00627)	(0.00960)	(0.00886)	(0.00627)	(0.00885)	(0.00823)
	N=1670	N=868	N=575	N=2807	N=1466	N=973

*
p<0.1,
**
p<0.05,

p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Appendix Table A17

2SLS - Effect of BMI on Suicidal Ideation, by Instrument (Common Sample) (Mean=0.097)

	Sibling BMI			BMI Genetic Risk Score		
Panel A: Males and Females Pooled						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.00408	0.00436	−0.00618	0.0104 [*]	0.00794	0.00790
	(0.00392)	(0.00425)	(0.0156)	(0.00563)	(0.00559)	(0.0115)
	N=3909	N=2353	N=1016	N=3909	N=2353	N=1016
Panel B: Males						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	−0.00570	−0.00536	−0.0916	0.00602	0.00216	0.00365
	(0.00514)	(0.00501)	(0.388)	(0.00586)	(0.00644)	(0.00709)
	N=1884	N=1148	N=447	N=1884	N=1148	N=447
Panel C: Females						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.0123 ^{**}	0.0121 ^{**}	−0.000205	0.0151	0.0120	0.0395
	(0.00546)	(0.00514)	(0.0150)	(0.0111)	(0.0103)	(0.0509)
	N=2025	N=1205	N=569	N=2025	N=1205	N=569

*
p<0.1,
**
p<0.05,

p<0.01

Sample restricted to respondents with BMI Genetic Risk Scores

Standard errors in parentheses (SE); Standard errors clustered at the school level

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Appendix Table A18

2SLS - Effect of BMI on Depression Index, by Instrument (Common Sample) (Mean=0.0)

	Sibling BMI			BMI Genetic Risk Score		
Panel A: Males and Females Pooled						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.00150	0.0108	−0.0589	−0.0118	−0.0254	0.0469
	(0.0145)	(0.0157)	(0.0655)	(0.0234)	(0.0285)	(0.0631)
	N=3937	N=2366	N=1026	N=3937	N=2366	N=1026
Panel B: Males						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	−0.0157	−0.0297	0.218	−0.0350	−0.0560	0.0312
	(0.0223)	(0.0269)	(1.492)	(0.0279)	(0.0389)	(0.0367)
	N=1897	N=1153	N=452	N=1897	N=1153	N=452
Panel C: Females						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.0162	0.0410 [*]	−0.0571	0.0222	0.0119	0.0711
	(0.0198)	(0.0223)	(0.0519)	(0.0409)	(0.0413)	(0.160)
	N=2040	N=1213	N=574	N=2040	N=1213	N=574

* p<0.1,

** p<0.05,

*** p<0.01

Sample restricted to respondents with BMI Genetic Risk Scores

Standard errors in parentheses (SE); Standard errors clustered at the school level

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Appendix Table A19

2SLS - Effect of BMI on Counseling Use, by Instrument (Common Sample) (Mean=0.085)

Sibling BMI				BMI Genetic Risk Score		
Panel A: Males and Females Pooled						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.00412	0.00500	0.00652	0.00593	−0.000911	0.00639
	(0.00324)	(0.00343)	(0.0179)	(0.00505)	(0.00560)	(0.0113)
	N=3943	N=2368	N=1029	N=3943	N=2368	N=1029

	Sibling BMI			BMI Genetic Risk Score		
Panel B: Males						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	−0.00212	0.00158	−0.110	0.00765	0.00380	0.00588
	(0.00411)	(0.00464)	(0.571)	(0.00606)	(0.00763)	(0.0109)
	N=1903	N=1157	N=454	N=1903	N=1157	N=454
Panel C: Females						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.00792	0.00734	0.0113	0.00122	−0.00882	0.0108
	(0.00484)	(0.00522)	(0.0171)	(0.00893)	(0.0105)	(0.0261)
	N=2040	N=1211	N=575	N=2040	N=1211	N=575

*
p<0.1,

**
p<0.05,

p<0.01

Sample restricted to respondents with BMI Genetic Risk Scores

Standard errors in parentheses (SE); Standard errors clustered at the school level

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Appendix Table A20

2SLS - Effect of BMI on Suicidal Ideation, by Instrument (Full Sample) (Mean=0.097)

	Sibling BMI			BMI Genetic Risk Score		
Panel A: Males and Females Pooled						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.00297	0.00275	−0.000088	0.00876 *	0.0103 **	0.00490
	(0.00184)	(0.00237)	(0.00634)	(0.00469)	(0.00508)	(0.00657)
	N=44997	N=28109	N=8806	N=5437	N=2857	N=1818
Panel B: Males						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	−0.00110	−0.00300	−0.00149	0.00546	0.00552	0.00335
	(0.00261)	(0.00313)	(0.0153)	(0.00548)	(0.00727)	(0.00685)
	N=21063	N=13423	N=3779	N=2648	N=1398	N=852
Panel C: Females						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.00567 **	0.00697 **	0.000961	0.0116 *	0.0140 *	0.0136
	(0.00245)	(0.00347)	(0.00542)	(0.00703)	(0.00795)	(0.0100)

Sibling BMI			BMI Genetic Risk Score		
N=23934	N=14686	N=5027	N=2789	N=1459	N=966

*
p<0.1,
**
p<0.05,

p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Data: The National Longitudinal Study of Adolescent to Adult Health

Appendix Table A21

2SLS - Effect of BMI on Depression Index, by Instrument (Full Sample) (Mean=0.0)

Sibling BMI				BMI Genetic Risk Score		
Panel A: Males and Females Pooled						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.00368	0.00238	−0.0148	0.00283	0.00267	0.0200
	(0.00695)	(0.00834)	(0.0204)	(0.0198)	(0.0258)	(0.0349)
	N=45335	N=28284	N=8905	N=5477	N=2871	N=1838
Panel B: Males						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	−0.00762	−0.00853	−0.0125	−0.00903	−0.0259	0.0329
	(0.0104)	(0.0128)	(0.0638)	(0.0260)	(0.0381)	(0.0459)
	N=21278	N=13531	N=3837	N=2671	N=1404	N=866
Panel C: Females						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.0111	0.0102	−0.0129	0.0148	0.0308	0.0103
	(0.00873)	(0.0104)	(0.0213)	(0.0267)	(0.0327)	(0.0498)
	N=24057	N=14753	N=5068	N=2806	N=1467	N=972

*
p<0.1,
**
p<0.05,

p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Appendix Table A22

2SLS - Effect of BMI on Counseling Use, by Instrument (Full Sample) (Mean=0.085)

Sibling BMI	BMI Genetic Risk Score
Panel A: Males and Females Pooled	

	Sibling BMI			BMI Genetic Risk Score		
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.00110 (0.00192) N=45436	0.00129 (0.00224) N=28345	0.00483 (0.00516) N=8927	0.00316 (0.00402) N=5484	-0.000782 (0.00478) N=2874	0.00493 (0.00639) N=1841
Panel B: Males						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	-0.00156 (0.00271) N=21332	-0.00241 (0.00384) N=13566	-0.00358 (0.0110) N=3850	0.00768 (0.00601) N=2677	0.00286 (0.00678) N=1408	0.0158 (0.0114) N=868
Panel C: Females						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.00286 (0.00248) N=24104	0.00380 (0.00298) N=14779	0.00832 (0.00577) N=5077	-0.00158 (0.00627) N=2807	-0.00749 (0.00885) N=1466	0.00235 (0.00823) N=973

*
p<0.1,
**
p<0.05,

p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Appendix Table A23

2SLS - Effect of BMI on Health

	Poor Health (Mean=0.005)			Diabetes (Mean=0.021)		
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
Panel A: Males and Females Pooled						
BMI	0.000311 (0.00110) N=5486	0.00121 (0.00143) N=2876	-0.00278** (0.00121) N=1841	-0.00146 (0.00269) N=2676	-0.00267 (0.00334) N=1413	0.00127 (0.00375) N=890
Panel B: Males						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.000834 (0.000958) N=2677	0.000076 (0.000908) N=1408	0.000666 (0.000594) N=868	-0.000655 (0.00402) N=1294	-0.00327 (0.00612) N=690	-0.000277 (0.00472) N=413
Panel C: Females						

	Poor Health (Mean=0.005)			Diabetes (Mean=0.021)		
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	-0.000193 (0.00186)	0.00208 (0.00251)	-0.00647** (0.00286)	-0.00193 (0.00381)	-0.00243 (0.00371)	0.00467 (0.00649)
	N=2809	N=1468	N=973	N=1382	N=723	N=477

*
p<0.1,

**
p<0.05,

p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Figure 1.

Relationship between BMI and Genetic Risk Score and Number of Observations by Risk Score

Data Source: The National Longitudinal Study of Adolescent to Adult Health

BMI Genetic Risk Score range restricted due to small numbers of observations at extreme values

Table 1

First Stage - Effect of the Genetic Risk Score on BMI (Mean=25.6)

Panel A: Males and Females Pooled			
	Full Sample	Whites	Blacks
Genetic Risk Score	0.308 *** (0.0401) N=5486 F=59.194 [0.030]	0.337 *** (0.0463) N=2876 F=52.912 [0.043]	0.353 *** (0.0730) N=1841 F=23.702 [0.030]
Panel B: Males			
	Full Sample	Whites	Blacks
Genetic Risk Score	0.318 *** (0.0583) N=2677 F=30.024 [0.036]	0.348 *** (0.0686) N=1408 F=25.923 [0.049]	0.355 *** (0.106) N=868 F=11.851 [0.039]
Panel C: Females			
	Full Sample	Whites	Blacks
Genetic Risk Score	0.296 *** (0.0524) N=2809 F=31.554 [0.025]	0.311 *** (0.0694) N=1468 F=20.166 [0.034]	0.323 *** (0.0976) N=973 F=10.399 [0.021]

*
p<0.1,**
p<0.05,***
p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level

Partial R² in square brackets

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Table 2

OLS and 2SLS - Effect of BMI on Suicidal Ideation (Mean=0.097)

Ordinary Least Squares				Two Stage Least Squares			
Panel A: Males and Females Pooled							
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks	
BMI	0.00152 ** (0.000720) N=5437	0.00191 (0.00118) N=2857	0.00148 (0.00122) N=1818	0.00876 * (0.00469) N=5437	0.0103 ** (0.00508) N=2857	0.00490 (0.00657) N=1818	
Panel B: Males							
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks	
BMI	-0.000543 (0.000869) N=2648	-0.000446 (0.00124) N=1398	-0.000241 (0.00174) N=852	0.00546 (0.00548) N=2648	0.00552 (0.00727) N=1398	0.00335 (0.00685) N=852	
Panel C: Females							
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks	
BMI	0.00283 ** (0.00110) N=2789	0.00357 * (0.00186) N=1459	0.00258 * (0.00146) N=966	0.0116 * (0.00703) N=2789	0.0140 * (0.00795) N=1459	0.0136 (0.0100) N=966	

* p<0.1,

** p<0.05,

*** p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level

Standard errors stable to two-way clustering (school and family, school and individual), see Appendix Table A10

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)
Data: The National Longitudinal Study of Adolescent to Adult Health

Table 3

OLS and 2SLS - Effect of BMI on Depression Index (Mean=0.0)

Ordinary Least Squares			Two Stage Least Squares			
Panel A: Males and Females Pooled						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.00507 [*] (0.00304) N=5477	0.00589 (0.00452) N=2871	0.00745 [*] (0.00412) N=1838	0.00283 (0.0198) N=5477	0.00267 (0.0258) N=2871	0.0200 (0.0349) N=1838
Panel B: Males						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	-0.000145 (0.00400) N=2671	-0.00248 (0.00528) N=1404	0.00866 (0.00693) N=866	-0.00903 (0.0260) N=2671	-0.0259 (0.0381) N=1404	0.0329 (0.0459) N=866
Panel C: Females						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.00857 ^{**} (0.00406) N=2806	0.0112 [*] (0.00627) N=1467	0.00855 [*] (0.00503) N=972	0.0148 (0.0267) N=2806	0.0308 (0.0327) N=1467	0.0103 (0.0498) N=972

* p<0.1,
** p<0.05,
*** p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level
Standard errors stable to two-way clustering (school and family, school and individual), see Appendix Table A11
Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)
Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Table 4

OLS and 2SLS - Effect of BMI on Counseling Use (Mean=0.085)

Ordinary Least Squares			Two Stage Least Squares			
Panel A: Males and Females Pooled						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.000233 (0.000743) N=5484	0.000833 (0.00113) N=2874	-0.000460 (0.00108) N=1841	0.00316 (0.00402) N=5484	-0.000782 (0.00478) N=2874	0.00493 (0.00639) N=1841
Panel B: Males						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	-0.000845 (0.000867) N=2677	-0.000440 (0.00131) N=1408	-0.000829 (0.00193) N=868	0.00768 (0.00601) N=2677	0.00286 (0.00678) N=1408	0.0158 (0.0114) N=868
Panel C: Females						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.00136 (0.00110) N=2807	0.00180 (0.00176) N=1466	0.000486 (0.00128) N=973	-0.00158 (0.00627) N=2807	-0.00749 (0.00885) N=1466	0.00235 (0.00823) N=973

* p<0.1,

**

p<0.05,

p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level

Standard errors stable to two-way clustering (school and family, school and individual), see Appendix Table A12

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)

Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Table 5

2SLS and OLS - Effect of BMI on Self-Rated Unattractiveness (Mean=23.8%)

Ordinary Least Squares			Two Stage Least Squares			
Panel A: Males and Females Pooled						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.00750 *** (0.00128) N=2653	0.0113 *** (0.00187) N=1404	0.00550 *** (0.00170) N=883	0.0100 (0.00773) N=2653	0.000792 (0.0110) N=1404	0.0282 *** (0.00897) N=883
Panel B: Males						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.00572 *** (0.00201) N=1279	0.00547 (0.00330) N=685	0.00886 *** (0.00271) N=408	0.0153 (0.0113) N=1279	-0.00803 (0.0150) N=685	0.0410 *** (0.0152) N=408
Panel C: Females						
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks
BMI	0.00873 *** (0.00192) N=1374	0.0145 *** (0.00307) N=719	0.00343 (0.00279) N=475	0.00243 (0.0116) N=1374	0.00320 (0.0161) N=719	0.0156 (0.0112) N=475

*
p<0.1,

**
p<0.05,

p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, parent obesity, county unemployment rate, gender, intelligence (picture vocabulary and number recall)
Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)

Table 6
2SLS and OLS - Effect of BMI on Interviewer-Rated Unattractiveness (Mean=52.9%)

Ordinary Least Squares				Two Stage Least Squares			
Panel A: Males and Females Pooled							
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks	
BMI	0.0154*** (0.00122) N=5476	0.0191*** (0.00168) N=2873	0.0125*** (0.00185) N=1836	0.0108 (0.00720) N=5476	0.00936 (0.00904) N=2873	0.0272** (0.0122) N=1836	
Panel B: Males							
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks	
BMI	0.0113*** (0.00188) N=2672	0.0140*** (0.00269) N=1408	0.00876** (0.00361) N=865	0.00101 (0.0102) N=2672	-0.00718 (0.0153) N=1408	0.0455*** (0.0198) N=865	
Panel C: Females							
	Full Sample	Whites	Blacks	Full Sample	Whites	Blacks	
BMI	0.0180*** (0.00152) N=2804	0.0226*** (0.00203) N=1465	0.0150*** (0.00263) N=971	0.0189** (0.00962) N=2804	0.0235** (0.0112) N=1465	0.0135 (0.0181) N=971	

*
p<0.1,

**
p<0.05,

p<0.01

Standard errors in parentheses (SE); Standard errors clustered at the school level
Average marginal effect in square brackets [average marginal effect]

Controls: Age, age squared, native born, wave, race/ethnicity, parent education, county unemployment rate, gender, intelligence (picture vocabulary and number recall)
Data: The National Longitudinal Study of Adolescent to Adult Health (Waves 1–4)