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The relationship between body mass index and income: Using genetic variants from HUNT as instrumental variables

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Abstract

Several studies have estimated effects of body mass index (BMI) on labour market outcomes, and these studies have mixed findings. A significant challenge has been to adequately control for omitted variables, selection, reverse causality, and measurement error. We examine the impact of BMI on income using genetic variants as instrumental variables for BMI. Individual-level pre-tax income from tax records was merged with health survey data containing measured height and weight, and data on genetic variants. The analyses were stratified by sex and a variety of methods were used to explore the sensitivity and validity of the instrumental variable (IV) strategy. For females we found that BMI had a negative effect on the logarithm of income. The effect estimated from the IV models (−0.02) was larger than the effect estimated from naïve ordinary least squares regressions (−0.01). For males, the coefficients for the effect of BMI on income were imprecise, and both positive and negative coefficients were estimated depending on the estimation method. Our results suggest that females are susceptible to reduced income levels following increased BMI.

KEYWORDS
I1, I12, I14, J01, J24, J3, J7

1 | INTRODUCTION

In 2015, a total of 107.7 million children and 603.7 million adults were obese. Since 1980, the prevalence of obesity has doubled in more than 70 countries and has continuously increased in most other countries (GBD Obesity Collaborators, 2017). The economic consequences of these trends, beyond morbidity and mortality, may include reduced labour market participation and lower income for those affected (Cawley, 2004, 2015). Nevertheless, these consequences are insufficiently understood and results of existing studies vary widely. In addition, many studies have found that the relationship between obesity and labour market outcomes differs between males and females (Asgeirsdottir, 2011; Atella et al., 2008; Averett et al., 2012; Averett & Korenman, 1993; Baum & Ford, 2004; Bozoyan & Wolbring, 2011; Brunello & D'Hombres, 2007; Burkhauser & Cawley, 2008; Cawley, 2000, 2004; Garcia & Quintana-Domeque, 2007; Greve, 2008; Han

et al., 2009; Johansson et al., 2009; Kelly, 2014; Meyerhoefer & Yang, 2016; Norton & Han, 2008; Renna & Thakur, 2010; Sabia & Rees, 2012; Sarlio-Lähteenkorva & Lahelma, 1999; Shimokawa, 2008; Villar & Quintana-Domeque, 2009).

Prior studies have proposed a number of hypotheses for why elevated levels of body-mass index (BMI, the weight in kilograms divided by the square of the height in meters) may reduce income. These can be grouped into three main categories:

(1) Health effects: High BMI reduces income by being a risk factor for deliberating diseases, which may reduce work ability and therefore income (Kinge & Morris, 2010; Lakdawalla et al., 2004). This hypothesis is supported by a number of epidemiological studies, which suggest that high BMI is a cause of an expanding set of chronic diseases, including cardiovascular disease, diabetes mellitus, chronic kidney disease, many cancers, and an array of musculoskeletal disorders (GBD Obesity Collaborators, 2017; Jiang et al., 2012; Lauby-Secretan et al., 2016; Singh et al., 2013).
(2) Reduced labour market performance: High BMI reduces income by affecting certain characteristics that might reduce performance in the labour market. For example, higher BMI has been found to be associated with lower self-esteem, lower reservation wages, and higher discount rates (Komlos et al., 2004; Offer, 2001).
(3) Discrimination: Individuals with high BMI may be discriminated against due to their physical attributes (Agerström & Rooth, 2011; Finkelstein et al., 2007; Pingitore et al., 1994; Popovich et al., 1997; Rooth, 2009). As discussed by Morris (2007) there are three main reasons for this. First, prejudice by employers, reflecting their disapproval of workers with obesity and the psychological costs incurred when dealing with them (McLean & Moon, 1980). Second, there may be stereotyping by employers, arising from a belief that persons with obesity are less productive (Everett, 1990). Third, discrimination may arise through uncertainty or a lack of knowledge about the productivity of workers with obesity (Pagan & Davila, 1997).

A significant challenge in prior literature has been the endogenous relationship between BMI and labour market outcomes. Low socioeconomic status might lead to weight gain and cause obesity (Meyerhoefer & Yang, 2016). For example, income might affect BMI if individuals with a lower income become obese, in part, because they cannot afford healthy food and therefore rely on calorie-dense fast foods (Averett, 2011). Omitted variables might also bias estimates if unobserved variables such as an individual's discount rate and/or ability and motivation are correlated with both BMI and income (Komlos et al., 2004; Meyerhoefer & Yang, 2016; Morris, 2006). Finally, measurement error might affect the findings as most datasets contain self-reports of weight and height and individuals tend to underreport their weight, with heavier individuals underreporting to a greater extent (Cawley et al., 2015).

To mitigate endogeneity the "first generation studies" typically used an earlier measure of BMI, rather than a contemporaneous one, as lagged BMI cannot be affected by current income. However, this is not a perfect strategy because lagged BMI may capture some omitted variable or variables related to both past and contemporaneous BMI (Averett, 2011). Extensions on these early studies have used sibling-fixed effects models that differ out a family specific error term or individual-fixed effects models, which remove all time-invariant heterogeneity at the individual level. However, such models have frequently suffered from a lack of power and rely on unobserved factors being person-specific and fixed over time. Unobservable factors influencing both weight and income may, however, vary over time. Furthermore, sibling-fixed effects may be biased due to variables that are not shared between the siblings and omitted from the regressions (Averett, 2011; Frisell et al., 2012).

More recent studies have relied on instrumental variable (IV) analysis, which is preferred to the strategies discussed above if a valid instrument can be found. There are essentially two types of variables that have been used as instruments for BMI in IV-analyses. One is to use the mean BMI in the local area as an instrument for respondent BMI. In these studies the idea is that individual BMI is affected by peer effects (Morris, 2006, 2007).

However, this may be an invalid instrument if respondents sort into neighbourhoods based on unobserved variables (Cawley, 2015). The second is to use the BMI of a biological relative as an instrument for the BMI of the respondent (Brunello & D'Hombres, 2007; Cawley, 2004; Kinge, 2016). Here, the idea is that the BMI of biologically related individuals mainly reflects genetic factors, making it an instrument indirectly based on genes. If shared household environments affect both the biological relative and the respondent, it might threaten the identification strategy. However, most studies in behavioural genetics do not find this to be a threat to the identification strategy (Cawley, 2015). Another potential threat to validity is that the genes that affect BMI may also affect other variables correlated with the error term in the income equation (Cawley, 2015).

Recently, data on genetic variants associated with BMI have become available. These genetic variants are determined at conception, and therefore less likely to be confounded by environmental factors. Researchers have exploited

this by using these genetic variants as instruments for BMI. Two prior studies have used genetic variants as instruments to investigate labour market outcomes. Norton and Han (2008) use a US dataset (The National Longitudinal Study of Adolescent Health [AddHealth]) to examine the effect of obesity on wages. The dataset contains genetic information for a subset of respondents, which was used as an instrument for BMI based on the biomedical literature (Norton & Han, 2008). In contrast to other studies, their preferred specification yields no effect of obesity on wages or employment, in neither men nor women. However, their sample was small, and may therefore have lacked power to detect these effects. In addition, the survey participants were relatively young (early to mid-twenties), so it is difficult to generalize these results and to compare them to the results of other studies. A second study used Finish data and instrumented for BMI using a genetic risk score constructed based on genetic variants associated with BMI (Böckerman et al., 2019). The findings were inconclusive, with both positive and negative coefficients, and imprecise. However, lack of power due to a small sample size, might be a contributing factor to the findings in this study. A lack of power was also the reason for not including sex-stratified results in the main manuscript.

Finally, a notable literature has used experimental designs. For examples, studies have sent out weight-manipulated photos or showed videos of applicants in different obesity categories, to study discrimination against the obese (Agerström & Rooth, 2011; Finkelstein et al., 2007; Pingitore et al., 1994; Popovich et al., 1997; Rooth, 2009). The applicants with obesity were less likely to be called for interviews and were perceived as being less capable for the job, than applicants with normal weight.

In the present study we combine data on income from tax records with newly available data containing genotyped data from biological samples and measured height and weight.

Based on these data, the following sets of analyses were conducted: (1) adjusted ordinary least squares (OLS) regressions of BMI on income; (2) 2SLS (two-stage least squares) IV-regressions using genetic variants as instruments; (3) two sample IV methods, combining data from two samples.

2 | METHODS

2.1 | Data

We used data from two rounds of the Nord-Trøndelag Health Studies (The HUNT studies). These population-based cohort studies were conducted in a geographical region in Central Norway between August 1995 and June 1997 (HUNT 2) and between October 2006 and June 2008 (HUNT 3). In these studies, the height and weight of participants was measured, and biological materials were collected for genotyping performed using Illumina HumanCoreExome arrays. The adult section of the HUNT study invited all inhabitants aged 20 years or older in the area of Nord-Trøndelag to participate, hence individuals were sampled independently, and no clusters of groups were sampled together. Detailed information about The HUNT Studies is available from Holmen et al. (2003) and Krokstad et al. (2013). We linked the data from the HUNT studies, using a de-identified key, to national data available from Statistics Norway: The Norwegian Tax Administration Database, The Norwegian Population Register and the National Education Database. The Norwegian Tax administration database contains information about income, defined as the sum of income from wages and self-employment, from 1967 to 2016. The Norwegian Population Registry contains information about whether individuals were alive and living in Norway during 1997 and 2008.

We used data from the rounds of HUNT from which the most recent data on height and weight were available. In total 50,910 participants below 67 years of age were alive and included in our study; 13,176 participants from HUNT 2 and 37,734 participants from HUNT 3. In total 780 participants (236 from HUNT 2 and 544 from HUNT 3) were excluded because they were registered as dead or not living in Norway at the time the study was conducted.

2.1.1 | Income measure

Income was measured at the individual level. To measure income, we used pensionable income from The Norwegian Tax Administration Database. Pensionable income is the sum of personal income from wages and self-employment. For participants of HUNT 2 we used income reported in 1997 and for participants from HUNT 3 we used income reported in 2008. Income was adjusted to 2016 price levels using the Consumer Price Index (Statistics Norway, 2017), and converted from Norwegian Kroner (NOK) to 2016 Euros (€ 1 = NOK 9.29) (Norges Bank, 2019).

2.2 | The genetic variants and the instrumental variable assumptions

The current study exploits the random assignment of an individual's genotype from his/her parental genotypes at conception (Lawlor et al., 2008). Under certain assumptions, which we discuss in detail below, associations between genetic variants and an outcome of interest cannot be due to simultaneity or omitted variable bias from behavioural and environmental factors, including the in-utero environment. As a result, genetic variants can seemingly be used to estimate causal effects of BMI on various outcomes (Lawlor et al., 2008). In the following we discuss the assumptions for an IV analysis and potential violations of these.

2.2.1 | Relevance

The relevance assumption is likely to hold. Around 40–70% of inter-individual variability in BMI, has been attributed to genetic factors (Maes et al., 1997; Visscher et al., 2012; Zaitlen et al., 2013). Locations (or loci) where the DNA sequence varies between people are called polymorphisms. The most commonly studied form of polymorphism is a single nucleotide polymorphism (SNP): a single base-pair variation at a particular DNA locus (see Appendix A in the Supplementary File, for a description). This study uses SNPs that have been found to be associated with BMI in genome wide association studies (GWAS) (Locke et al., 2015) as instruments for BMI. A GWAS is a study of associations between specific phenotypes and SNPs across the genome, in a hypothesis-free way and corrected for multiple testing. The strength of instruments using SNPs based on GWAS studies are generally higher than studies using one particular genetic variant (Dixon et al., 2020; von Hinke et al., 2016).

2.2.2 | Independence

The independence assumption that the association between the genetic instrument and BMI is independent of measured and unmeasured confounders, is only partly testable. Although genes are randomly allocated at conception, and therefore assumed to be independent of confounders, there are still some issues. First, individuals are more likely to mate with individuals with similar phenotypes (assortative mating). Second, offspring genes are conditional on parental genes (dynastic effects). Third, having one genetic variant can increase your chance of having another genetic variant (linkage disequilibrium) (Palmer et al., 2012). Fourth, differential ancestry can lead to a higher frequency of some genetic variants in particular sub-populations (population stratification) (Price et al., 2006). Lastly, non-confounding variables can also modify the effect of genetic variants on income. Several sensitivity analyses are conducted to explore these potential violations of the independence assumption.

2.2.3 | Exclusion

The exclusion restriction requirement, that the genetic variants should only affect income via BMI, cannot be tested. We cannot be certain that one or more of the 97 genetic variants we have used have no association with phenotypic traits that are associated with income. However, since most genes are pleiotropic (i.e., affect more than one phenotypic trait), it is not unlikely that such an association exists. This issue is known as pleiotropy, which is a well-known potential threat to the third condition. For this reason, several methods, which we conduct in our sensitivity analyses, have been developed to assess the effect of bias from horizontal pleiotropy on the IV-estimates.

2.3 | Estimation strategy

The main estimation method, to estimate the effects of BMI on income, was the model suggested by Norton and Han (2008):

$$\Pr(w_i > 0) = \alpha^e \mathrm{BMI} + X_i\beta^e + \varepsilon_i^e, \quad (1)$$

$$\ln(w_i) = \alpha^w BMI + X_i\beta^w + \varepsilon_i^w \quad \textit{if income is positive}, \tag{2}$$

where BMIi is individual i's BMI and X is a vector of control variables. The subscripts e and w refer to the probability of a positive income (e) and income (w). ε is the error term. The aim was to estimate consistent estimates of αe and αw. For the probability of income > 0 we followed Norton and Han (2008) and used a linear probability model (LPM) and for logged income we used an OLS model. The distribution of logged wages was roughly normal. In these analyses we adjusted for (i) birth year and study period, and (ii) birth year, study period, educational level, smoking status, marital status, and urbanity.

One of the benefits of the primary estimation method is that it separates out the binary outcome of earning an income or not, from the size of income given that someone is an income earner. Nevertheless, these models condition on the dependent variable, which is a potential consequence of the instrument. Hence, a second model was also fitted, in which we included both earners and non-earners and calculated the log of income +1:

$$\ln(w_i + 1) = \alpha BMI + X_i\beta + \varepsilon_{i,} \tag{3}$$

Next, we conducted IV-analyses using 2SLS regression with three different genetic instruments, stratified by sex. These genetic instruments were selected based on 97 genetic variants found to be associated with BMI in GWAS (Locke et al., 2015). All, but one (rs12016871) of these variants were available in our dataset, and for this variant we followed Brandkvist et al. (2019) and used the variant rs4771122 as a proxy. Three genetic instruments were constructed: an unweighted genetic risk score (GRS), a weighted GRS, and an instrument which involved including the two variants (FTO [rs1558902] and MC4R [rs6567160]) as two dummy variables. FTO and MC4R were the two variants with the strongest associations with BMI in GWAS (Locke et al., 2015). The unweighted GRS was calculated by summing the number of BMI-increasing alleles for each participant. The weighted GRS was calculated separately for males and females. First, we weighted the number of BMI-increasing alleles by multiplying the respective sex-specific beta-coefficients for each variant (reported by Locke et al., 2015), and then we summed the product for each participant. By summing the genetic variants into a genetic risk score we construct a single instrument and the IV-estimator is just identified (Palmer et al., 2012). The first-stage equation in our 2SLS models was:

$$BMI_i = GenesIV_i\theta + X_i\beta^{BMI} + v_i, \tag{4}$$

where GenesIVi represents one of the three genetic instruments and v_i is the error term. The correct standard errors from the 2SLS, which account for the two-step nature of the estimation, can then be obtained using the asymptotic covariance matrix given by Wooldridge (2002) (p. 95). We used the first-stage F-statistic to evaluate the relevance assumption, and consider the instrument to be weak if F < 10 (Staiger & Stock, 1994).

In all our analyses, we adjusted for study period (HUNT 2 or 3), and birth year (categorical).

2.4 | Sensitivity analyses

We conducted two-sample analyses and analyses excluding outliers mainly to explore the effects of horizontal pleiotropy, and within-family analyses to investigate violations of the IV- assumptions due to assortative mating, dynastic effects, and population stratification (Brumpton et al., 2019).

2.4.1 | Two-sample methods

Two-sample methods involve combining independent data sources from populations with similar ancestry (Burgess et al., 2017). The associations and standard errors between (1) the genetic variants indexed from j to J (G_j) and the outcomes ($\hat{\delta}_{yj}$), and (2) the genetic variants and the exposure ($\hat{\delta}_{xj}$) can be obtained from two different samples and combined using a two sample IV-estimator (Angrist & Krueger, 1992). We use HUNT data for the SNP-outcome

association ($\hat{\delta}_{yj}$, $\hat{\sigma}_{yj}$), and one of the most recent and largest genome-wide association studies by Locke et al. (2015) for the SNP-BMI association ($\hat{\delta}_{xj}$).

We use four two sample IV-estimators, which allow us to explore the validity of our genetic variants as instruments. To make the estimates interpretable as the marginal effect of a one-unit increase in BMI on income, we followed Budu-Aggrey et al. (2018) and Dixon et al. (2020) and divided the estimates from the various two-sample methods by the median standard deviation (4.6) reported by Locke et al. (2015).

Inverse variance weighted

With a single genetic variant Gj, the effect of the risk factor on the outcome can be estimated as a simple ratio of association estimates: $(\hat{\delta}_{yj})/(\hat{\delta}_{xj})$ (Lawlor et al., 2008). With no clustering or sample weights the standard error of this estimate can be calculated as $(\hat{\sigma}_{yj})/(\hat{\delta}_{xj})$ (Burgess & Bowden, 2015; Burgess et al., 2013). With multiple genetic variants, the ratio estimates calculated from each genetic variant can be averaged using an inverse-variance weighted formula. that is the inverse-variance weighted (IVW) estimate (Burgess et al., 2013). In the IVW approach it is assumed that the ratio estimates all provide independent evidence on the causal effect. The IVW estimate is similar to the 2SLS estimate, when the genetic variants are uncorrelated (Burgess & Thompson, 2015). The IVW estimate can be obtained from a linear regression of the genetic associations with the outcome ($\hat{\delta}_{yj}$) on the genetic associations with the risk factor ($\hat{\delta}_{xj}$) using IVWs ($(\hat{\delta}_{yj})^{-2}$) when there is no intercept term in the regression:

$$\hat{\delta}_{yj} = \theta_{IVW}\hat{\delta}_{xj} + \varepsilon_j, \tag{5}$$

where ($\hat{\delta}_{yj}$) and ($\hat{\delta}_{xj}$) are data in the model, ϵ_j is a residual term and θ_{IVW} is the parameter of interest.

If the exclusion restriction holds, the heterogeneity among the effect estimates for each of the genetic variants should not be greater than the heterogeneity one would expect from random variation. This can be tested using Cochran's Q statistic (Cochran, 1950). The Cochran's Q statistic is closely related to the Sargan (1958) over-identification test and can identify failure of the instrumental variable assumptions, but not which genetic variant or variants that are causing the failure or why the genetic variants are invalid (Dixon et al., 2020).

MR-Egger

The MR-Egger and IVW estimators are similar as both estimate the mean of the ratio estimates. However, in MR Egger analyses the intercept term is estimated as part of the analysis, and not constrained to zero (as is the case in the IVW analysis) (Bowden et al., 2015; Hemani et al., 2018):

$$\hat{\delta}_{yj} = \theta_{0EGGER} + \theta_{1EGGER}\hat{\delta}_{xj} + \varepsilon_j, \tag{6}$$

where the parameter θ_{0EGGER} represents the intercept term and θ_{1EGGER} is the slope (i.e., the MR-egger estimate). The MR-Egger intercept term can be used as a test for pleiotropy (MR-Egger intercept test), and a non-zero intercept term is indicative of directional pleiotropy. In the presence of pleiotropy, and given that the underlying assumptions for MR-Egger regression hold, the MR-Egger slope estimate can be interpreted as a pleiotropy-adjusted effect (Bowden, et al., 2016).

MR-Egger estimators are less powerful and efficient than the other two-sample estimators because they require both an intercept and a slope parameter in the estimation (Dixon et al., 2020). In addition to the IV-assumptions, both the MR-Egger and IVW estimators require that the InSIDE (Instrument Strength Independent of Direct Effect) assumption holds and that there is no measurement error. The InSIDE assumption is difficult to test, and is not an unlikely problem. Measurement error may be revealed from a regression dilution statistic (I^2_{GX}). If $I^2_{GX} < 90\%$ this may indicate bias due to measurement error. MR-Egger can be conducted with a simulation extrapolation (SIMEX) correction that can be used to adjust for this bias (Bowden et al., 2016).

Weighted median

The weighted median estimator uses the median ratio estimate of all available instruments and does not rely on the InSIDE assumption (Bowden et al., 2015; Dixon et al., 2020). This estimator requires that at least 50% of the variants are valid in order to be unbiased. Given that this assumption holds, invalid instruments contribute no weight and are less biased than the 2SLS and IVW estimators.

Weighted modal

Finally, the weighted modal method classifies the genetic variants into clusters based on similar effects (i.e. modes). The weighted modal estimator will be consistent if the largest cluster consists of valid genetic variants. All other genetic variants could essentially be invalid. The weighted modal estimate requires that there is Zero Modal Pleiotropy (ZEMPA) (Hemani et al., 2018), that is that the most common effect is a consistent estimate of the true causal effect (Hartwig et al., 2017).

2.4.2 | Effects of outlier removal

Findings from the two-sample analyses were inspected to identify genetic variants that had an outlying effect on income, and the effect of removing these genetic variants from our 2SLS analyses was explored.

2.4.3 | Within-family analyses

A 2SLS regression with family fixed effects was conducted to investigate the effect of only including individuals that are biologically related. This method can reveal bias due to assortative mating, dynastic effects, and population stratification (Brumpton et al., 2019). These analyses require more power, and were therefore not stratified by sex.

2.4.4 | Non-linear models

Fitting non-linear models in the present IV setting is complicated as the instruments explain a relatively small proportion of variance in BMI, and therefore non-linear effects may not be detectable. Hence, to estimate non-linearities we have used an approach suggested by Staley and Burgess (2017). The sample was divided into 10 stratums by using residual BMI. We then calculate linear IV-regression model estimates in each stratum of the population as a ratio of coefficients: the association between the GRS and income divided by the association between the GRS and BMI. This effect is referred to as the localized average causal effect. We performed meta-regression of the localised average causal effect estimates against the mean of the BMI in each stratum in a flexible semiparametric framework by using the derivative of fractional polynomial models of degrees 1 and 2. We report two tests for non-linearity: a quadratic and a fractional polynomial test, which assess whether a non-linear model fits the localised average causal effect estimates better than a linear model.

3 | RESULTS

3.1 | Descriptive information

In total, 50,910 participants <67 years of age were included in the analyses (Table 1). The mean age was 46.26 (S.D. = 12.93) years for males, and 45.63 (S.D. = 13.03) years for females. The mean BMI for males was 27.22 (S.D. = 3.84) kg/m^2, and the mean BMI for females was 26.62 (S.D. = 4.90) kg/m^2.

For males, mean income, the proportion with a positive income, and mean income given a positive income, was lowest for participants in the underweight category and highest for participants in the overweight category (Table 2).

For females, mean income, and the proportion with a positive income was lowest for participants with underweight and highest for participants categorized as normal weight. The mean income given a positive income was lowest for females with underweight, and highest for females with normal weight.

3.2 | Results from conventional analyses

For males, there was neither an apparent association between BMI and the probability of having a positive income, nor between BMI and log income, when birth year and study period were included in the model (Table 3). When additional covariates were included, we did not find an association between BMI and the probability of income, but BMI was associated with significantly higher log income.

TABLE 1 Descriptive information by sex for adults <67 years of age

Variable	Category	Sex	
		Male N (%)	Female N (%)
Total		24,166	26,744
Age	18–24	1859 (7.69)	2260 (8.45)
	25–44	8724 (36.10)	10,009 (37.43)
	45–66	13,583 (56.21)	14,475 (54.12)
	Missing	0 (0)	0 (0)
Marital status	Married/registered partner	13,148 (54.41)	14,912 (55.76)
	Unmarried	8167 (33.80)	7411 (27.71)
	Divorced/separated	2562 (10.60)	3329 (12.45)
	Widow/widower	247 (1.02)	1048 (3.92)
	Missing	42 (0.17)	44 (0.16)
Education	Primary school	4499 (18.66)	5343 (20.04)
	Secondary school	14,029 (58.19)	12,751 (47.81)
	Higher education, short	4098 (17.00)	7681 (28.80)
	Higher education, long	1485 (6.16)	893 (3.35)
	Missing	0 (0)	0 (0)
Urbanity	Urban	15,787 (65.33)	17,579 (65.73)
	Rural	8273 (34.23)	8995 (33.63)
	Missing	106 (0.44)	170 (0.64)
Smoking status	Smoker	6892 (28.52)	8548 (31.97)
	Former smoker	7003 (28.98)	7215 (26.98)
	Never smoker	9977 (41.29)	10,720 (40.08)
	Missing	294 (1.22)	261 (0.98)
[a]BMI category	Underweight	77 (0.32)	239 (0.89)
	Normal weight	6756 (27.96)	11,186 (41.83)
	Overweight	12,288 (50.85)	9599 (35.89)
	Class I obesity	4177 (17.29)	4014 (15.01)
	Class II obesity	867 (3.59)	1706 (6.38)
	Missing	0 (0)	0 (0)

Abbreviation: BMI, body mass index.

[a]The BMI-categories were defined as follows: Underweight = BMI < 18.5, normal weight = BMI: 18.5–24.9, overweight = BMI: 25.0–29.9, class I obesity = BMI: 30.0–34.9, and class II obesity = BMI ≥ 35.0.

For females, BMI was associated with a lower probability of having a positive income, and with a lower log income. The effects were slightly reduced, but remained statistically significant, when additional covariates were added to the model.

3.3 | IV estimators

3.3.1 | Two-stage least squares

In all the IV-analyses, the F-statistic was high and above 10 for all instruments (Table 4). The F-statistic was consistently highest for the weighted GRS, followed by the unweighted GRS, and FTO & MC4R.

TABLE 2 Mean income, proposition with positive income, and income among those with positive income, by BMI-category for adults <67 years of age, stratified by sex

Sex	[a]BMI-category	Mean (SD) income (€)	Prop. with income>0	Mean (SD) income given income>0 (€)
Males	Underweight	17,670.95 (18,498.80)	0.73	24,297.55 (17,576.35)
	Normal weight	40,587.04 (39,389.58)	0.92	44,326.88 (39,098.90)
	Overweight	46,858.23 (44,003.06)	0.92	51,072.73 (43,533.56)
	Class I obesity	43,922.51 (38,338.86)	0.89	49,265.39 (37,221.31)
	Class II obesity	35,218.91 (28,530.15)	0.86	41,041.39 (26,635.20)
Females	Underweight	22,302.29 (21,220.66)	0.81	27,475.50 (20,308.61)
	Normal weight	31,164.99 (25,850.35)	0.90	34,581.05 (24,932.55)
	Overweight	30,784.12 (28,631.21)	0.86	35,683.71 (27,854.71)
	Class I obesity	26,674.91 (26,562.14)	0.80	33,200.96 (25,719.17)
	Class II obesity	22,880.94 (23,398.86)	0.75	30,543.73 (22,288.49)

Abbreviation: BMI, body mass index.

[a]The BMI-categories were defined as follows: Underweight = BMI < 18.5, normal weight = BMI: 18.5–24.9, overweight = BMI: 25.0–29.9, class I obesity = BMI: 30.0–34.9, and class II obesity = BMI ≥ 35.0.

TABLE 3 Sex-specific results from OLS analysis of the effect of BMI on (ii) log income given income is positive, and (iii) log income +1; for participants aged <67 years

Analysis	Covariates	Sex	Coef. (SE)	P-val.	95% CI
Prob. income>0	I	Males	−0.0007 (0.0005)	0.128	−0.0015; 0.0002
		Females	−0.0049 (0.0004)	10.001	−0.0057; −0.0042
	II	Males	−0.0004 (0.0005)	0.385	−0.0013; 0.0005
		Females	−0.0042 (0.0004)	<0.001	−0.0050; −0.0034
Log income given income>0	I	Males	0.0003 (0.0015)	0.832	−0.0026; 0.0032
		Females	−0.0106 (0.0011)	<0.001	−0.0127; 0.0085
	II	Males	0.0037 (0.0015)	0.011	0.0008; 0.0066
		Females	−0.0061 (0.0011)	<0.001	−0.0082; −0.0040
Log income +1	I	Males	−0.0070 (0.0048)	0.149	−0.0164; 0.0025
		Females	−0.0581 (0.0040)	<0.001	−0.0660; −0.0503
	II	Males	−0.0008 (0.0048)	0.873	−0.0102; 0.0086
		Females	−0.0468 (0.0039)	<0.001	−0.0545; −0.0390

Notes: I. Birth year and study period. II. Birth year, study period, educational level, smoking status, marital status, and urbanity.

Abbreviation: BMI, body mass index; OLS, ordinary least squares.

For men, BMI did not have a significant effect on the likelihood of earning an income when using the GRSs (Table 4; Figure 1). When using the FTO & MC4R as instruments, there was a positive effect of BMI on the likelihood of income and on the log of income +1. Among males with a positive income, an effect of BMI on log income was not found.

For females, BMI did not influence the likelihood of having a positive income when using any of the instruments (Table 4). Among females with a positive income, BMI had a negative effect on log income, and this effect was significant when using the GRSs, but not when using the FTO & MC4R instrument. The results of the model using the full population and the log of income +1 as the dependent variable were quantitatively similar (Table 4).

For ease of interpretation of the income effect resulting from changes in BMI in women, we follow Manning (1998) and use a heteroskedastic smearing estimator, correcting for birth year and education. A female with obesity could be expected to earn € 5053 less per year, compared with a normal weight female.

TABLE 4 Sex-specific results from 2SLS IV analyses with three different sets of instruments, estimating the effect of BMI on (i) the likelihood of positive income, (ii) log income among income earners, and (iii) log income +1; for adults <67 years of age

			First-stage		Second-stage				
Analysis	**Sex**	**Sex**	**Coef. (SE)**	**P-val.**	**Coef. (SE)**	**P-val.**	**95% CI**	**F-stat.**	**P-val.**
Prob. income>0	Males	Unweighted GRS	0.093 (0.004)	<0.001	−0.004 (0.003)	0.191	−0.009; 0.002	604.267	<0.001
		Weighted GRS	3.980 (0.145)	<0.001	−0.001 (0.003)	0.613	−0.006; 0.004	756.377	<0.001
		FTO & MC4R	0.394 (0.034) & 0.259 (0.039)	<0.001 & <0.001	0.010 (0.005)	0.053	<-0.001; 0.021	87.74	<0.001
	Females	Unweighted GRS	0.108 (0.005)	<0.001	−0.003 (0.003)	0.271	−0.009; 0.002	528.704	<0.001
		Weighted GRS	4.588 (0.171)	<0.001	−0.003 (0.002)	0.189	−0.008; 0.002	722.457	<0.001
		FTO & MC4R	0.464 (0.042) & 0.434 (0.047)	<0.001 & <0.001	−0.002 (0.004)	0.610	−0.011; 0.006	103.789	<0.001
Log income	Males	Unweighted GRS	0.092 (0.004)	<0.001	0.006 (0.009)	0.505	−0.012; 0.025	552.273	<0.001
		Weighted GRS	3.904 (0.149)	<0.001	0.008 (0.008)	0.368	−0.009; 0.024	684.477	<0.001
		FTO & MC4R	0.376 (0.035) & 0.247 (0.040)	<0.001 & <0.001	0.010 (0.018)	0.588	−0.025; 0.445	74.931	<0.001
	Females	Unweighted GRS	0.107 (0.005)	<0.001	−0.028 (0.008)	>0.001	−0.043; −0.013	480.047	<0.001
		Weighted GRS	4.581 (0.178)	<0.001	−0.020 (0.006)	0.002	−0.032; −0.007	665.333	<0.001
		FTO & MC4R	0.445 (0.044) & 0.422 (0.049)	<0.001 & <0.001	−0.017 (0.012)	0.175	−0.041; 0.007	89.106	<0.001
Log income +1	Males	Unweighted GRS	0.093 (0.004)	<0.001	−0.032 (0.031)	0.303	−0.092; 0.029	604.267	<0.001
		Weighted GRS	3.980 (0.145)	<0.001	−0.006 (0.028)	0.831	−0.060; 0.048	736.377	<0.001
		FTO & MC4R	0.394 (0.034) & 0.259 (0.039)	<0.001 & <0.001	0.114 (0.057)	0.047	0.002; 0.227	87.739	<0.001
	Females	Unweighted GRS	0.108 (0.005)	<0.001	−0.054 (0.029)	0.060	−0.110; 0.002	528.704	<0.001
		Weighted GRS	4.488 (0.171)	<0.001	−0.048 (0.025)	0.053	−0.096; 0.001	722.457	<0.001
		FTO & MC4R	0.464 (0.042) & 0.434 (0.047)	<0.001 & <0.001	−0.035 (0.045)	0.437	−0.124; 0.054	103.789	<0.001

Abbreviation: GRS, genetic risk score.

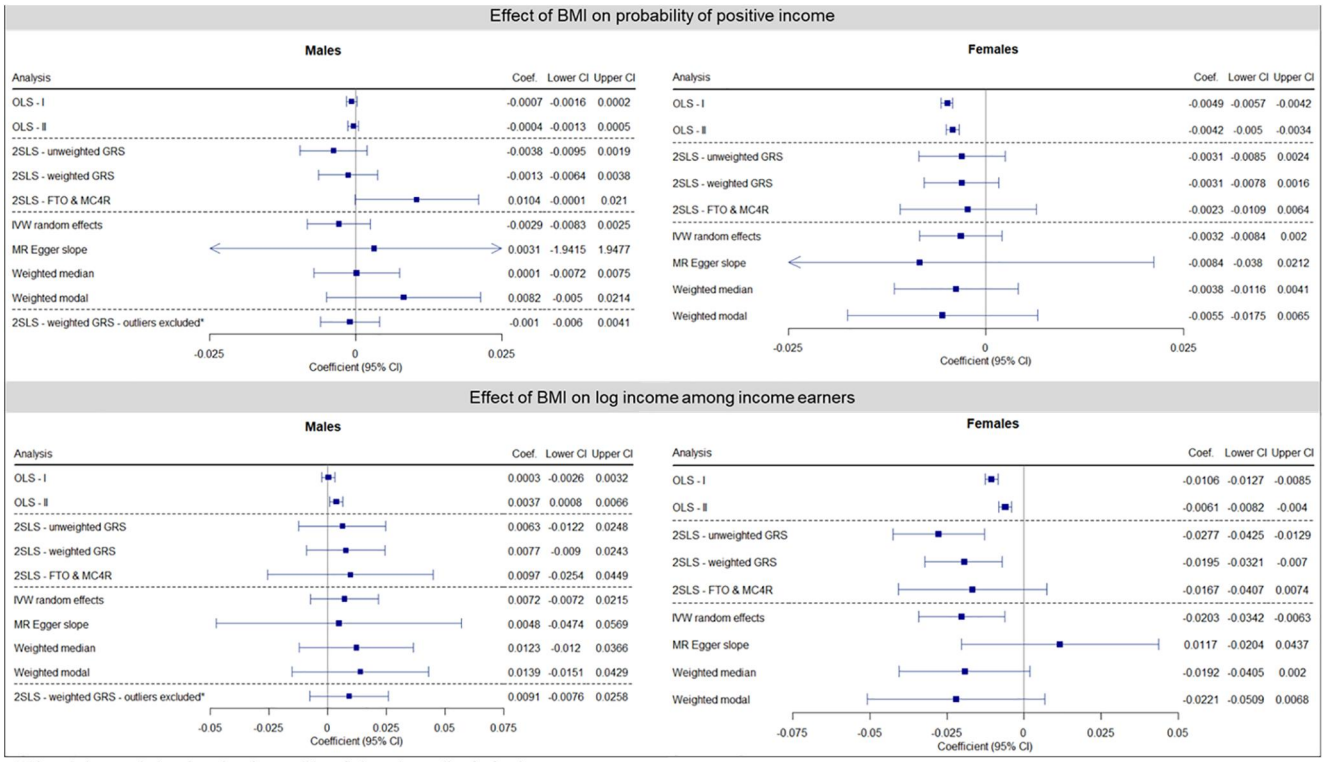

FIGURE 1 Coefficients and 95% confidence intervals from the two-sample analyses for the effect of body mass index on having a positive income (top panel), and for the effect of BMI on log income among income earners (bottom panel), for males (left), and females (right). GRS, genetic risk score [Colour figure can be viewed at wileyonlinelibrary.com]

3.3.2 | Two-sample estimators

The effect of BMI on the probability of income-males

For males, the effect of BMI on the probability of having a positive income was not significant when using any of the four two-sample estimators (Figure 1).

The IVW estimate was negative, which is the same as what was found in the OLS analyses (Table 3), and in the 2SLS (Table 4) analyses using GRSs as instruments.

The MR-Egger and weighted modal estimates were positive, and the weighted median estimate was close to zero. The 2SLS estimate was positive when using the FTO and MC4R instrument, implying that the direction of the association between obesity and the likelihood of a positive income differs between genetic variants. Multiple peaks could also be seen upon inspection of the modal density plot (Supplementary file, Figure S2). The highest peak was above zero, the second peak was near zero, and the last peak was below zero.

Cochran's Q was 119.1 (p-value: 0.048), indicating heterogeneity bias, but the MR- Egger intercept test was non-significant (Coef. = −0.000079, p-value = 0.955), suggesting that heterogeneity was not due to directional pleiotropy.

The effect of BMI on the probability of income-females

For females, BMI was consistently associated with a lower probability of having a positive income, when using each of the four two-sample estimators. However the effect estimates were imprecise and non-significant (Figure 1). The two-sample results were also consistent with what was found in all the OLS (Table 3) and 2SLS (Table 4) analyses.

Cochran's Q was 104.0 (p-value: 0.248) suggesting that bias due to heterogeneity was small, and the MR-Egger intercept test suggested that bias due to pleiotropy was negligible (Coef. = 0.00052, p-value = 0.886).

The effect of BMI on log income–males

For males, the effect of BMI on log income was positive, but close to zero and imprecise when using the four two-sample estimators (Figure 1). This was consistent with the findings from the OLS (Table 3) and 2SLS analyses (Table 4).

Cochran's Q was 76.0 (p-value: 0.924) and the MR-Egger intercept test was (Coef. = 0.00024, p-value = 0.942), suggesting that bias due to heterogeneity and directional pleiotropy was negligible.

The effect of BMI on log income–females

For females, the effect of BMI on income, was negative and significant when using the IVW estimator (p-value = 0.004), negative and weakly significant (p-value = 0.075) when using the weighted median estimator, and negative and non-significant when using the weighted modal estimator (Figure 1). This is in line with the findings from the OLS (Table 3) and 2SLS (Table 4) analyses.

The MR-Egger estimate, however, showed a positive association between BMI and log income, but the estimate was non-significant and imprecise.

Cochran's Q was 97.3 (p-value: 0.415) suggesting that bias due to heterogeneity was small, but the MR-Egger intercept test suggested that there was bias due to directional pleiotropic effects (Coef. = −0.00435, p-value = 0.036).

While the MR-Egger intercept suggests that the true effect is likely to be more positive than what has been estimated from the IVW and 2SLS estimators, the MR-Egger slope estimate may not be an accurate estimate of the causal effect. The modal density plot (Supplementary File, Figure S2), exhibited multiple clear peaks, which may indicate that clusters of genetic variants yield different effects. If these clusters of genetic variants act through the same confounders, then the InSIDE assumption required for the MR-Egger slope estimate to be reliable may have been violated, and as a result the MR-Egger slope estimate can be considered unreliable (Burgess & Thompson, 2017).

In situations where the InSIDE assumption is violated, the median and mode-based estimates may be more reliable. However, the exhibition of multiple peaks also violates the ZEMPA assumption, hence the modal estimate is also unreliable. As a result, the weighted median estimate can be considered the most reliable estimate of the effect of BMI on log income for females.

Additional two-sample analyses

The I^2_{GX} statistic was 83.99% for males, and 85.27% for females, suggesting that there was some measurement error in the estimates. To investigate the effect of measurement error we conducted MR-Egger estimation with correction for attenuation bias (MR-Egger SIMEX). In all the analyses the results were similar to the uncorrected MR-Egger estimates.

In all the analyses, for both males and females, the two-sample analyses performed with all ages included, gave similar coefficients and p-values as the analyses with age restricted to participants below 67 years of age.

3.4 | Outlier removal

From studying the funnel (Supplementary File, Figure S3) and forest (Supplementary File, Figure S4) plots for males, we identified one left-lying outlier (rs10733682) when assessing the effect of BMI on the probability of having a positive income, and one left-lying outlier when assessing the effect of BMI on log income (rs2836754). The results of the analyses without outliers were similar to the results of the main analysis (Figure 1).

For females, no clear outliers were found when studying the funnel (Supplementary File, Figure S3) and forest (Supplementary File, Figure S4) plots.

3.5 | Within family–weighted GRS

The coefficients estimated from the within family and full population analyses were similar (Table 5), suggesting that bias due to assortative mating, dynastic effects and population stratification was of low concern.

3.6 | Non-linear models

Using the full base case sample of unrelated individuals, the data suggest that non-linearity might be present. However, there was evidence consistent with the null for both a quadratic term (males: p = 0.10, females: p = 0.41), and for a

TABLE 5 Within family IV-analysis with weighted GRS as the instrument

Analysis	Population	Coef. (SE)	P-val.	95% CI
Prob. income>0	Full population	−0.002 (0.002)	0.172	−0.006; 0.001
	Within-family	−0.004 (0.004)	0.321	−0.012; 0.004
Log income given income>0	Full population	−0.008 (0.005)	0.119	−0.019; 0.002
	Within-family	−0.008 (0.011)	0.461	−0.030; 0.013
Log income +1	Full population	−0.032 (0.018)	0.085	−0.068; 0.004
	Within-family	−0.035 (0.043)	0.416	−0.120; 0.050

Abbreviation: GRS, genetic risk score;

fractional polynomial term (males: $p = 0.22$, females: $p = 0.48$). Some non-linearities were observed especially at lower BMI levels for males (Supplementary File, Figure S8)

3.7 | Probit estimation of the binary response

We estimate LPM, using probit (see Appendix B, Supplementary File, page 3, for methodological details). Although somewhat lower standard errors were observed it does not alter the interpretation of the findings (Supplementary File, Table S1).

4 | DISCUSSION

This study estimates the effect of BMI on income in Norway using adjusted OLS and IV methods, as well as a range of sensitivity analyses. Measurement error was minimized by using income from national registries and BMI based on measured height and weight.

In men we do not find an effect of BMI on the probability of having a positive income and find a positive non-significant effect of BMI on log income. Non-linear analysis suggests that this positive effect of BMI on income in men is at lower levels of BMI. For females we found a negative non-significant effect of BMI on the probability of having a positive income and find a negative significant ($p < 0.05$) effect of BMI on log income.

Over the last decades, several studies have examined the impact of BMI on income and other labour market outcomes using data from several developed countries (see e.g., Averett, 2011; Meyerhoefer &Yang, 2016 for reviews). A recurrent finding in some, but not all, studies is that BMI has a significant negative effect on income for women, while the results are inconclusive for men (Cawley, 2004; Morris, 2006). Cawley (2004), also found that for white women the estimates from IV models based on biological siblings, were larger than the OLS estimates. This is also in line with the sex-specific results reported by Böckerman et al. (2019) in the supplementary material of their article.

With reference to prior literature, there are predominantly three potential explanations for why BMI could be negatively associated with income in women, but not in men. First, weight-based discrimination in the labour market might impact females more or differently than males (Meyerhoefer & Yang, 2016). A Swedish study demonstrated that job applications, which were sent along with a photo that was manipulated to show the applicant as obese, had significantly lower callback, than those sent without photo manipulation (Rooth, 2009). This effect was stronger in women, than in men. Comparable studies and results have been found in other countries (Pingitore et al., 1994). Another study where HR professionals that regularly made career decisions were asked to evaluate individuals that only differed in terms of gender, ethnicity and BMI, found that obese females were less likely to be hired and less likely to be hired for a supervisory position, compared with obese males (Giel et al., 2012).

Second, obesity has been found to affect self-esteem more in women, than in men, and reduced self-esteem might affect labour market performance (Averett, 2011).

However, studies on the magnitude of this effect suggest that it is small. (Averett, 2011; Mocan & Tekin, 2011).

Third, the effects of BMI on health differs by sex. Cawley (2003) explores this hypothesis. Based on US data he suggested that a differential impact of BMI on disability and physical health is the primary explanation for an effect of BMI on income in women and not in men. In similitude, Kinge and Morris (2010) show more pronounced associations

of BMI with health-related quality of life in women, compared with men. Obesity related health problems may reduce worker productivity and increase absenteeism. The effect of BMI income and workforce participation may, however, be biased by the healthy-worker effect, which may differ between males and females.

The effect of BMI on household income was not studied in this paper, Studying household income might be relevant if the aim is to measure the financial position of the individual. BMI could be associated with household income through various mechanisms. First, directly as it affects individual income. Second, indirectly by affecting family members income. It could reduce the partner's income, as obesity related diseases might increase the care burden of the family. that is family member taking time off work in order to receive health care (Drummond et al., 2015). Lastly, it could increase the partner's income if the partner has to work more to compensate for the reduced income of the individual.

The primary estimation strategy used in this study, based on Norton and Han (2008), are two equations for the labour market outcomes: pr (income > 0) and ln (income) given that the individuals are income earners. A potential limitation is the selection into employment. This means that we do not know how those who are not employed would have responded to changes in BMI if they were employed, which will in turn make our estimates less valid in a population with different levels of unemployment. Some studies have attempted to account for this using Heckman selection models. Such models should be fitted with variables that solely affect the probability of employment but are unrelated to wages. Models without such variables rely solely on functional form for identification and are unstable (Dow & Norton, 2003). Nevertheless, a useful extension of this work would be to find valid identifying variables and estimate selection models.

This study had some limitations. First, our understanding of BMI is limited, and BMI is trait heterogeneous (i.e., the genetic pathways leading to high BMI are heterogeneous). For instance, some people may have genetic variants that lead to an increased probability of poor diet, others may be predisposed to low metabolism or overeating. Ideally, we should seek to identify genetic variants that are associated with different underlying causes and assess the individual impact of each of these on BMI and income. In our analyses, the effect of clusters of genetic variants seem to differ between males and females, and the effect of BMI on income differed between the clusters. To further increase our understanding of the effects of BMI on income, future studies should seek to identify which variants have negative and which have positive effects on income, separately for males and females. Second, although we have done our best to test the validity IV-assumptions, we need an improved understanding of how each of the genetic variants work to fully understand the limitations of their use (Cawley et al., 2011). Third, it is possible that the health consequences of acquiring obesity due to a genetic predisposition differ from the health consequences of obesity acquired through environmental factors (e.g., social transmission). Finally, while the estimations reported are informative, by showing the impact of changes in BMI near the mean, they can be misleading if the relationship between BMI and employment is in fact non-linear. However, the instruments explain a relatively small proportion of variance in BMI (Dixon et al., 2020). Hence, non-linear effects may not be detectable, and this is a question for further research.

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ETHICAL STATEMENT

The study was approved and participant consent was waived by the Regional Committee for Medical and Health Research Ethics Central Norway.

ETHICAL APPROVAL

The study was approved by the Regional Committee for Ethics in medical research (2016/537/REK midt).

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from third parties. Restrictions apply to the availability of these data, which were used under license for this study. Please contact the corresponding author for more information about data accessibility.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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