NBER WORKING PAPER SERIES

THE IMPACT OF LATE-CAREER JOB LOSS AND GENOTYPE ON BODY MASS INDEX

Lauren L. Schmitz
Dalton Conley

Working Paper 22348
http://www.nber.org/papers/w22348

NATIONAL BUREAU OF ECONOMIC RESEARCH
1050 Massachusetts Avenue
Cambridge, MA 02138
June 2016

This study was funded in part by the Russell Sage Foundation (grant number 83-15-29) and by an NIA training grant to the Population Studies Center at the University of Michigan (T32 AG000221). The Health and Retirement Study (HRS; accession number 0925-0670) is sponsored by the National Institute on Aging (grant numbers NIA U01AG009740, RC2AG036495, and RC4AG039029) and is conducted by the University of Michigan. Additional funding support for genotyping and analysis were provided by NIH/NICHD R01 HD060726. The views expressed herein are those of the authors and do not necessarily reflect the views of the National Bureau of Economic Research.

The Impact of Late-Career Job Loss and Genotype on Body Mass Index
Lauren L. Schmitz and Dalton Conley
NBER Working Paper No. 22348
June 2016
JEL No. I1,J63,J69

ABSTRACT

This study examines whether the effect of job loss on body mass index (BMI) at older ages is moderated by genotype using twenty years of socio-demographic and genome-wide data from the Health and Retirement Study (HRS). To avoid any potential confounding we interact layoffs due to a plant or business closure—a plausibly exogenous environmental exposure—with a polygenic risk score for BMI in a regression-adjusted semiparametric differences-in-differences matching framework that compares the BMI of those before and after an involuntary job loss with a control group that has not been laid off. Results indicate genetically-at-risk workers who lost their job before they were eligible for Social Security benefits, or before age 62, were more likely to gain weight. Further analysis reveals heterogeneous treatment effects by demographic, health, and socioeconomic characteristics. In particular, we find high risk individuals who gained weight after a job loss were more likely to be male, in worse health, single, and at the bottom half of the wealth distribution. Across the board, effects are concentrated among high-risk individuals who were not overweight prior to job loss, indicating unemployment at older ages may trigger weight gain in otherwise healthy or normal weight populations.

Lauren L. Schmitz
Population Studies Center
University of Michigan
426 Thompson St.
Ann Arbor, MI 48104
lschmitz@umich.edu

Dalton Conley
Department of Sociology
Princeton University
153 Wallace Hall
Princeton, NJ 08544
and NBER
dconley@princeton.edu

Introduction

High body mass index (BMI) continues to be a pressing public health concern in the United States. Obese adults are at risk for multiple chronic conditions, including type two diabetes, cardiovascular disease, and certain cancers (Bhaskaran et al. 2014; Lewis et al. 2009). Recent statistics show almost 75% of the adult population aged 20 years and older in the US is overweight (BMI ≥25 kg/m2)—42% of which are classified as obese (BMI ≥30 kg/m^2) (Ogden, et al. 2012).

Although in large part lifestyle changes have fueled the obesogenic environment and obesity epidemic in westernized societies over the past 30 years, more work is needed to understand the joint contribution of biological and social forces to life course trends in body weight. In particular, research that incorporates genetic, behavioral, and social factors into the same framework may help to more accurately explain the complex etiology of cardiovascular risk factors like BMI. This study takes advantage of twenty years of genetic and socio-demographic data from the Health and Retirement Study (HRS) to examine whether a significant social stressor—an unexpected job loss at older ages—amplifies polygenic risk for weight gain.

An aging population coupled with the recent economic downturn in an era of falling wages, rising earnings inequality, and high involuntary joblessness makes understanding the health effects of unemployment at older ages particularly important. The US labor market has been plagued by high levels of unemployment since the 1980s, reaching crisis levels during the Great Recession. Older workers in particular can no longer expect the same benefits accrued during the 1950s-1970s golden age of reliable growth and economic security. Rates of job loss have risen considerably for all workers in recent years, but are increasing faster for older workers than younger workers. Between 1981 and 2009, three-year job loss rates for workers aged 55

and over rose from 10% to more than 14% (Farber 2015). Post displacement, older workers also have a harder time finding new employment and spend more time unemployed than their younger counterparts (Johnson 2012). Workers aged 55-64 are about 16 percentage points less likely than workers aged 35-44 to be employed after a job loss and reemployed job losers aged 45-64 take three to four weeks longer to find jobs than those aged 20-29 (Farber 2015).

A growing body of evidence has linked late-career job loss to a range of adverse health and chronic disease outcomes in the US and abroad (Deb et al. 2011; Gallo et al., 2006; Gallo et al., 2004; Gallo et al. 2000; Marcus 2014; Strully 2009). However, the majority of studies on the effect of job loss on health do not deal with the endogeneity of job loss, or the possibility that layoffs may be correlated with worker characteristics, health behaviors, or poor health (Burgard et al. 2007; Jusot et al. 2008; Roelfs et al. 2011). In addition, while most studies have found adverse health effects from involuntary unemployment, there is no consensus, and several studies have not been able to uncover an average treatment effect (e.g. Salm 2009). A major limitation of these studies is they are not able to account for heterogeneous treatment effects by genotype, or the possibility that changes in BMI induced by a job loss vary across the spectrum of genetic risk.

That said, selection problems are further compounded with the addition of genetic data into the analysis. The majority of gene-environment (G × E) interaction studies use endogenous measures of the environment that cannot adequately address both the non-random distribution of genes across environments and the possibility that genes may be acting as proxies for other unobserved gene-environment correlations (rGE) or G × E interactions. In this case, endogenous measures of job loss could be intertwined with a host of unobserved genetic or environmental influences that are associated with health and changes in BMI. Therefore, methods that exploit

exogenous measures of "G" and "E" are needed to properly identify G × E effects (Conley 2009; Fletcher and Conley 2013; Schmitz and Conley 2015).

To address these shortcomings, we interact business closures—a plausibly exogenous environmental exposure— with a polygenic risk score for BMI in a regression-adjusted semiparametric differences-in-differences matching framework that compares the BMI of those before and after an involuntary job loss with a control group who has not been laid off. Business closures are considered more exogenous than layoffs or firings because they are typically the byproduct of external, firm level decisions to restructure or relocate business. However, it is still possible that workers with unhealthy behaviors or poor health, for example, could select into more vulnerable or volatile industries. Combining propensity score matching with differences-in-differences estimation makes the model more robust to selection on observables and unobservables with time invariant effects, such as ability or worker preferences.

Results show genetically-at-risk workers who were not overweight prior to job loss were more likely to gain weight than comparable high-risk workers who were continuously employed. These effects are concentrated among workers who are male, in worse health, single, and have below median net worth. Across the board, weight gain is only significant in the population of older workers who are not eligible for Social Security (i.e. before age 62), indicating unemployment in the years leading up to retirement may be particularly detrimental to long-term cardiovascular health.

Background

The Genetics of BMI

BMI is an inexpensive, non-invasive measure of obesity that has been found to be as predictive of cardiovascular disease risk as other, more difficult to measure anthropomorphic measures such as abdominal adiposity (i.e. waist circumference [WC], waist-hip ratio [WHR], or waist-height ratio [WHtR]) (Taylor et al. 2010). Twin studies on the genetic determination of BMI have found a strong heritable component (h^2 of ~40-70%) and previous meta-analyses of genome-wide association studies (GWAS) have identified more than 100 genome-wide significant loci associated with BMI (Locke et al. 2015; Shungin et al. 2015; Speliotes et al. 2010).

The largest cluster of highly significant loci is located in the FTO gene region (fat mass and obesity associated gene) on chromosome 16. Growing evidence from epidemiological and functional studies suggests FTO increases obesity risk through subtle changes in food intake and preference. The BMI-increasing allele of FTO SNPs has been found to be associated with increased intake of dietary fat or protein, increased appetite and reduced satiety, poor food choices and eating habits, and loss of control over eating (for a review see Loos and Yeo 2014). BMI-associated alleles also overlap with genes and pathways implicated in the central nervous system—particularly genes expressed in the hypothalamus and pituitary gland—that regulate appetite (Locke et al. 2015). Most BMI-associated loci appear to have their largest impact early in life or during adolescence although a few loci, which have also been associated with type 2 diabetes or coronary artery disease, exhibit stronger effects in older adults (Winkler et al. 2015).

Established loci from the largest BMI GWAS to date (N ~340,000) account for only 2.4% of the variation in BMI (Locke et al. 2015). Explanations for the small proportion of

heritability explained by genetic association studies include underpowered discovery sample sizes, measurement error due to incomplete linkage between the measured and causal alleles, undiscovered rare or low-frequency variants with larger effects, epistasis, or G × E interaction. In particular, lifestyle and social context have been found to fuel the onset and persistence of BMI, including evidence of FTO by environment interactions of exercise on the attenuation of BMI (Kilpeläinen et al. 2011), possibly mediated by DNA methylation (Almén et al. 2012; Bell et al. 2010). In the social sciences, gene-by-social environment interaction studies have linked G × E effects on BMI to lifetime SES, social norms, historical period, and institutional policies (e.g. Boardman et al. 2012; Guo et al. 2015; Liu and Guo 2015). An important insight from this line of research is the pervasive role the social environment may play in provoking underlying genetic risk. However, with the exception of historical period (i.e. birth cohort) all the above studies deploy endogenous measures of environment, calling into question whether FTO or other genetic determinants of BMI are truly moderated by cross-sectional environmental contexts. Further research that elucidates specific socioeconomic stressors that aggravate polygenic risk for BMI is needed to better understand the biological processes that are involved in the regulation of body weight over the life course.

Job Loss and Health

The corrosive effects of job loss on health and health behaviors have been documented extensively in the social science literature. For older workers in particular, the scarring effects from job loss are severe. Research finds job loss at older ages is associated with longer periods of unemployment than any other age group (Johnson and Park 2011), insufficient retirement savings and lower levels of household wealth (Munnell and Sass 2009), higher rates of depression and anxiety (Bender and Jivan 2004; Bonsang and Klein 2012; Gallo et al. 2006), and

a sharp increase in the need for medical care due to heightened stress levels and gaps in health insurance coverage (Sudano Jr and Baker 2003; Tu and Liebhaber 2009). Further, when reemployed, older workers suffer significant wage penalties and lower levels of employer-offered pension and health insurance (Brand 2004; Farber 2004; Jacobson et al. 1993). All these factors could trigger chronic stress and lead to adverse changes in health behaviors, both of which aggressively deteriorate health (Hammarström and Janlert 1994; Laitinen et al. 2002). With respect to eating behavior, chronic life stress in particular seems to be associated with a greater preference for energy- and nutrient-dense foods that are high in sugar and fat, and evidence from longitudinal studies suggests that chronic life stress may be causally linked to weight gain (e.g. Torres and Nowson 2007).

To investigate the health effects of job loss, past studies have used exogenous shocks to employment such as a plant or business closure to control for reverse causality, or the possibility that sicker people are more likely to be unemployed (Deb et al. 2011; Falba et al. 2005; Gallo et al., 2006; Gallo et al. 2004; Gallo et al. 2000; Gallo et al. 2001; Salm 2009; Strully 2009). However, results from these studies have been mixed. Using the HRS, Salm (2009) finds no causal effect of exogenous job loss on various measures of physical and mental health, whereas Gallo et al. (2006) find involuntary job loss is associated with increased depressive symptoms, but only for individuals with net worth below the median. Gallo et al. (2004) find job loss increases the risk of stroke but not myocardial infarction.

Other studies have found a positive association between involuntary job loss and harmful health behaviors like smoking (Falba et al. 2005; Marcus 2014), excessive alcohol consumption (Deb et al. 2011; Gallo et al. 2001; Marcus 2014), and weight gain (Deb et al. 2011; Marcus 2014). Using data from the German Socio-Economic Panel Study (SOEP), Marcus (2014) finds

involuntary job loss increases the probability of smoking initiation by three percentage points on average and also finds small (but statistically significant) changes in BMI (around 0.1 kg/m^2). Using finite mixture models Deb et al. (2011) find substantial heterogeneity in the effect of business closures on BMI and drinking behavior; the escalation of unhealthy behaviors was found to be concentrated among workers who were already pursuing unhealthy behaviors pre-job loss, indicating the effects of job loss may be especially problematic for high-risk individuals. However, while these studies have modeled differential trajectories in health after unemployment by prior health behaviors, this study is the first to examine whether weight gain after an unexpected job loss is modified by polygenic risk.

Data and Methods

The Health and Retirement Study

Data are from the Health and Retirement Study (HRS)—a nationally representative, longitudinal dataset of individuals over the age of 50 and their spouses that began in 1992.[3] The HRS interviews approximately 20,000 participants every two years from the time of their entry into the survey until their death. Every six years a new cohort of participants is added to keep the sample nationally representative of the US population over 50. To maximize sample size, we compile data from 11 waves (1992-2012) of the HRS. The HRS collects detailed demographic and socio-economic data on its participants, including information on changes in BMI, labor force participation, unemployment, physical and mental health, health related behaviors, income,

[3] The Health and Retirement Study (HRS; accession number 0925-0670) is sponsored by the National Institute on Aging (grant numbers NIA U01AG009740, RC2AG036495, and RC4AG039029) and is conducted by the University of Michigan. Additional funding support for genotyping and analysis were provided by NIH/NICHD R01 HD060726.

and wealth. In 2009, the HRS genotyped 12,507 participants who provided DNA samples in 2006 and 2008. Our final data set compiles genotype data from the HRS Genotype Data Version 1 File (2006-2008 samples), information on job loss and smoking behavior from the HRS 1992-2012 Public Use Core Files, and socio-demographic data from the RAND HRS Data File (v.O) (see Table 1 for a list of all variables used in the analysis).[4]

After quality control (QC) analysis on the genotype data, 78,319 observations on 9,186 individuals remained in the HRS sample.[5] Since we are using results from a GWAS on individuals of European ancestry to construct our polygenic score (PGS – formerly known in the literature as a genetic risk score [GRS] or polygenic risk score [PRS]), we exclude observations on individuals who report being black (N=9,196), American Indian, Alaskan Native, Asian, or Pacific Islander (N=3,192), or who report being white but of Hispanic origin (N=5,058).[6] This leaves us with 60,873 observations over time on 7,038 individuals. Additionally, since research has shown partial or full retirement may have positive effects on health (e.g. Coe and Zamarro 2011; Neuman 2008), we exclude observations on working respondents if they were over the Social Security Early Eligibility Age (EEA) of 62 (4,680); similarly, because the HRS is only representative of the US population 50 plus, we exclude individuals if they were below age 50 at baseline (N=1,221). Of the remaining 54,972 observations, 7,608 have missing baseline information or were not interviewed at baseline, 32,975 were not working for pay, 42 were missing information on work status, 5,101 were self-employed, 88 were missing information on

[4] The RAND HRS Data file is an easy to use longitudinal data set based on the HRS data. It was developed in RAND with funding from the National Institute on Aging and the Social Security Administration.

[5] See "BMI Polygenic Risk Score (PGS)" section for QC specifics.

[6] This number also includes one individual with three observations who was missing information on Hispanic origin.

self-employment status, 1,055 were missing information on reason for job loss, 170 were missing information on BMI, and 2,172 observations were missing information on other key covariates (i.e. census region lived in, smoking status, health insurance status, household income, or household wealth). After propensity score matching, an additional 40 observations were not on common support and 57 were not assigned a weight. The final analytic sample (Table 5, Column 5) consists of 5,664 observations on 2,150 full and part time workers ages 50-60 at baseline that were working for pay at the time of the interview and were not self-employed. The sample can include multiple observations for the same individual over time.

Treatment and Control Groups

For each remaining observation, we use information from two waves—before and after treatment. Before treatment $(t-1)$, all respondents were working for pay. At the following interview two years later (t), respondents in our treatment group report they are no longer working for their previous-wave employer. These respondents were asked why they left their employer. Possible answers included 'business closed', 'laid off/let go', 'poor health/disabled', 'quit' or other reasons.[7] Respondents could report up to three reasons. Our definition of exogenous job loss includes 235 observations (4.15% of the total sample) that report being laid off due to a business closure. Following Salm (2009), we exclude workers who in addition stated they quit or left for health reasons but include workers who also stated they were laid off or let go. For the control group, we use individuals who report working with the same employer the entire time they are in the sample—i.e. we do not include individuals in the control group if they ever quit their job or were laid off for any reason between ages 50 and 62.

[7] Other reasons include family care, better job, retired, family moved, sold business (own), strike, divorce/separation, transportation/distance to work, or early retirement incentive/offer.

Table 1. Variable definitions

Dependent variable	
BMI	Body mass index in kg/m^2
Independent variables	
Business closure	1=business closed between waves; 0=still working for previous wave employer
BMI PGS	Body mass index polygenic risk score, standardized
Overweight	1=BMI>=25; 0=BMI<25
Female gender	1=Female; 0=Male
Age	Age in years
Married	1=Married/partnered; 0=Divorced/separated/widowed/single
Highest degree obtained	Binary (0/1) variables for no degree or high school degree. The omitted category is associate's/bachelor's/professional degree.
Region dummies	Binary (0/1) variables for Census region of residence: Northeast (New England and Mid Atlantic); Midwest (EN Central and WN Central); South (S Atlantic, ES Central, and WS Central). The omitted category is West (Mountain and Pacific).
Household income (log)	Log of total (respondent + spouse) household income in 2010 dollars. Includes earnings, household capital income, income from all pensions and annuities, income from social security disability and supplemental social security income; income from social security retirement, spouse or widow benefits, income form unemployment or workers compensation, income from veteran's benefits, welfare and food stamps, alimony, other income, and lump sums from insurance, pension and inheritance.
Household wealth ($100k)	Total household income in 2010 dollars divided by 100,000 for scalar consistency. It is the sum of the value of primary residence, net value of real estate (not including primary residence), net value of vehicles, net value of businesses, net value of stocks, mutual funds, and investment trusts, value of checking, savings or money market accounts, value of CD, government savings bonds, and T-bills, net value of bonds and bond funds, and the net value of all other savings, less the value of all mortgages/land contracts (primary residence), value of other home loans (primary residence), and the value of any other debt.
Firm size[a]	Binary (0/1) variables for firm size categories: Less than or equal to 4 employees; 5-14 employees; 15-24 employees; 25-99 employees; 100-499 employees. The omitted category is firm size greater than or equal to 500 employees.
Part time	1=works part time; 0=does not work part time.
Industry[a]	Binary (0/1) variables for industry categories: agriculture, fishing, or farming; construction or mining; manufacturing; trade; public services; finance, insurance, or real estate; public administration. The omitted category is misc. services.
Occupational status[a,b]	Binary (0/1) variables for blue collar and service workers. The omitted category is white collar workers.
Job tenure[a]	Current job tenure in years.
Health status	1=excellent or very good self-reported health; 0=good, fair, or poor self-reported health.
Health insurance	1=covered by a federal or employer-sponsored health insurance program; 0=otherwise.
Exercise	1=exercises vigorously three or more times per week; 0=otherwise.
Ever smoke cigarettes	1=smoked 100 or more cigarettes in their lifetime; 0=otherwise.
Cigarettes per day[a]	Total number of cigarettes smoked per day, excluding pipes or cigars. Variable is set equal to zero if respondent does not smoke.
Drinks alcohol	1=drinks alcoholic beverages; 0=does not drink alcoholic beverages.
Doctor diagnosed psychiatric issue[a]	1=reports doctor diagnosed emotional or psychiatric problems; 0=otherwise.
Survey year	Binary (0/1) variables for 1994-2012. The omitted year is 1992.

[a] Variables with additional category for missing values. [b] Occupation groups are based on Meyer and Osborne three digit harmonized census occupation codes (Meyer and Osborne 2005).

BMI Polygenic Score (PGS)

We calculate a linear PGS for the HRS sample based on a genome-wide association (GWA) meta-analysis conducted by the Genetic Investigation of Anthropometric Traits (GIANT) Consortium on a total of 249,796 individuals of European ancestry (Speliotes et al. 2010). Specifically, we construct a genome-wide composite PGS composed of weighted effects of specific single nucleotide polymorphisms (SNPs) for BMI using original genotype (i.e. not imputed) data from the HRS Genotype Data Version 1 (2006-2008 sample) File.[8] SNPs in the HRS genetic database were matched to SNPs with reported results in the GWAS. In the HRS genetic data set, 838,490 SNPs were available to construct the BMI score in the second-generation PLINK software (Chang et al. 2015; Purcell and Chang 2015). For each of these SNPs, a loading was calculated as the number of BMI-associated alleles multiplied by the effect-size estimated in the original GWAS. Loadings are then summed across the SNP set to construct the polygenic score. Since pruning for linkage disequilibrium reduced performance of the score, our score includes all SNPs that passed quality control filters. Or, the PGS is a weighted average across the number of SNPs (n) of the number of reference alleles x (zero, one, or two) at that SNP multiplied by the effect size for that SNP (β):

$$PGS_i = \sum_{j=1}^{n} \beta_j x_{ij} \qquad (1)$$

To increase the power of its predictive capacity, we do not impose a GWAS p-value threshold or cut-off for SNPs included in our PGS (Dudbridge 2013). Instead, SNPs with relatively large p-values or small effects are down weighted in the composite score. The PGS is

[8] Genotyping was performed on the HRS sample using the Illumina Human Omni-2.5 Quad beadchip (HumanOmni2.5-4v1 array). The median call-rate for the 2006-2008 samples is 99.7%.

standardized to have a mean of zero and a standard deviation of one for the population of white, non-Hispanic workers (i.e. those of European ancestry) in our analytic sample.

The BMI PGS is predictive of cross-sectional measures of BMI in non-interactive main effects models for both the entire sample of white non-Hispanic respondents, and in our analytic sample (see Table 2). In addition, since we are estimating changes in weight after a job loss, we also estimate whether the PGS is associated with BMI in a longitudinal framework by regressing BMI on the BMI PGS and BMI at baseline, or in $t-1$. We find the PGS is a positive and significant predictor of changes in weight in the HRS European ancestry sample, but not in our analytic sample. However, a further breakdown by treatment status in Table 3 reveals the score is associated with changes in weight in the sample of treated individuals who were not overweight prior to job loss.

To control for confounding by population stratification—or the non-random distribution of genes across populations—we use principal components. The principal components measure the uncorrelated variation or dimensions in the data, accounting for ethnic or racial differences in genetic structures within populations that could bias estimates due to confounding with important environmental variation. We calculate the principal components using PLINK from the entire sample of genotyped respondents in the HRS, and include the first four in our regression analysis—a dimensionality that has generally proven adequate in the literature (Price et al. 2006). Controlling for the first four principal components accounts for any systematic differences in ancestry that can cause spurious correlations while also maximizing the power that is needed to detect true associations.

Table 2. Main effect of BMI PGS on BMI and changes in BMI by sample

	Analytic sample		European ancestry	
	BMI Level	BMI Change	BMI Level	BMI Change
BMI PGS	0.388***	0.00171	0.414***	0.0339***
	(0.0724)	(0.0258)	(0.02240)	(0.00918)
BMI (t-1)		0.952***		0.943***
		(0.00481)		(0.00170)
N	5664	5664	52320	52320
R^2	0.00680	0.875	0.0057	0.855

Analytic sample consists of white, non-Hispanic male and female workers who were not self-employed and between the ages of 50-60 at t-1. All regressions include controls for population stratification in the genotype data.
†p<.10; * p<.05; ** p<.01; ***p<.001.

Table 3. Main effect of BMI PGS on BMI and changes in BMI by treatment status

	Business Closed				Still Working for the Same Employer			
	BMI Level		BMI Change		BMI Level		BMI Change	
BMI PGS	0.761*	0.652	0.0690	0.437†	0.370***	0.0207	-0.00142	-0.0124
	(0.352)	(0.486)	(0.143)	(0.242)	(0.0740)	(0.106)	(0.0262)	(0.0483)
BMI (t-1)			0.893***	0.854***			0.955***	0.944***
			(0.0258)	(0.0326)			(0.00489)	(0.00659)
Overweight (t-1)		7.161***		0.818*		7.126***		0.184*
		(0.619)		(0.392)		(0.118)		(0.0725)
BMI PGS × Overweight (t-1)		-0.227		-0.547†		0.0526		0.0103
		(0.591)		(0.295)		(0.124)		(0.0567)
N	235	235	235	235	5429	5429	5429	5429
R^2	0.0552	0.406	0.848	0.853	0.00637	0.408	0.876	0.876

Analytic sample consists of white, non-Hispanic male and female workers who were not self-employed and between the ages of 50-60 at t-1. All regressions include controls for population stratification in the genotype data. Overweight status: BMI>=25.
†p<.10; * p<.05; ** p<.01; ***p<.001.

Empirical Strategy

To investigate whether the effect of an involuntary job loss on BMI is moderated by genotype, this study uses differences-in-differences (DD) estimation combined with nonparametric kernel matching to estimate the average treatment effect on the treated (ATT) by genotype, or the G × E effect (Heckman et al. 1997; Marcus 2014). This approach compares individuals who have been laid off due to a business closure with a group of (nearly identical) individuals who are still working for their same employer. To construct a control group with a similar distribution of covariates as the treatment group, the kernel-based matching estimator uses a distance-weighted average of all propensity scores in the control group to construct a counterfactual outcome for each individual in the treatment group. These weights are applied to the DD regression model to obtain a balanced sample of treated and untreated individuals. The coefficients from the DD regression are used to estimate the ATT by genotype, or the effect of job loss on changes in BMI by genotype for individuals who were actually laid off. The same covariates used to estimate the propensity score, or the probability of treatment, are also used in the DD regression model. Thus, coefficients from the regression-adjusted semiparametric DD matching estimator are considered "doubly robust" because the estimator will be consistent as long as either the regression model or the propensity score model is correctly specified (Imbens 2004; Robins and Ritov 1997).

In addition, the regression-adjusted DD matching estimator is considered more robust than a traditional DD estimator because it accounts for both selection on observables and selection on unobservables with time invariant effects. Or, in other words, the model allows for systematic differences between treatment and control groups even after conditioning on observables (Smith and Todd 2005). A traditional DD setting assumes that after conditioning on

observed characteristics, the BMI of individuals in the treatment group would have evolved similarly over time to the BMI of individuals in the control group if they had never been laid off. If i' denotes an individual in the control group with the same characteristics as individual i in the treatment group, this assumption can be expressed in formal notation as

$$E[BMI_{it} - BMI_{it-1}|X, BC = 0] = E[BMI_{i't} - BMI_{i't-1}|X, BC = 0] \quad (2)$$

Which states that if a business closure (BC) did not occur, the change in BMI between t and $t-1$ evolves similarly between the treatment and control groups after conditioning on observables X. While conditioning on genotype and a rich set of covariates minimizes the possibility of violating this assumption, other systematic differences between the treated and control groups may remain even after conditioning on observables. Such differences may arise, for example, if an individual selects into an industry that is more likely to experience a business closure because of unmeasured characteristics like ability or childhood conditions, or because of unmeasured differences in local labor markets where participants or nonparticipants reside. To minimize potential confounding from unobservable characteristics, we relax this assumption and use the weights from propensity score matching to reduce unmeasured differences between the treatment and control groups that could bias estimates:

$$E[BMI_{it} - BMI_{it-1}|W(X), BC = 0] = E[BMI_{i't} - BMI_{i't-1}|W(X), BC = 0] \quad (3)$$

Our empirical strategy can be broken down into three parts. First, we estimate the propensity score using a probit regression that regresses business closures on the PGS and a rich set of covariates (see Table 1). These covariates are both standard in the job loss literature and satisfy the conditional independence assumption outlined in Eq. 2—i.e. they influence job loss

and/or changes in BMI (Dehejia and Wahba 1999; Heckman et al. 1997). In addition, we only condition on observables that are unaffected by job loss (or the anticipation of it), or variables that are either fixed over time or measured in $t-1$ (Caliendo and Kopeinig 2008). To avoid losing observations with missing information on a covariate, we set missing values equal to zero and include an additional dichotomous variable that is equal to one if the observation is missing. As a result, matching is not only on observed values but also on the missing data pattern (Marcus 2014; Stuart 2010). Throughout, we restrict our analysis to the region of common support, or the subset of individuals in the control group that are comparable to individuals in the treatment group (Dehejia and Wahba 1999).[9]

Next, we use the estimates from the probit regression to compute the weights for the control group with kernel matching—a nonparametric matching estimator that uses the weighted averages of all observations on common support to construct the counterfactual outcome (Heckman et al. 1997; Smith and Todd 2005).[10] Specifically, the weight given to a non-treated individual j is in proportion to the closeness of their observables to treated individual i:

$$w(i,j) = \frac{K[(P_i - P_j)/b]}{\sum_{j \in BC=0} K[(P_i - P_j)/b]} \tag{4}$$

Where P is the propensity score for individual i or j in the treated or control group, respectively, $K[\cdot]$ is the kernel function, and b is the bandwidth parameter. The choice of bandwidth is thought to be more important than the choice of kernel function. High bandwidth values result in a smoother estimated density function, leading to a better fit and decreasing the

[9] Specifically, we drop treatment observations whose propensity score is greater than the maximum or less than the minimum propensity score of the controls.

[10] We use the program psmatch2 (Leuven and Sianesi 2003) in Stata 14 to compute $w(j)$.

variance between the estimated and actual density function. However, this may also bias estimates if underlying features of the distribution are obscured by a larger bandwidth. Following Heckman (1997), we use the Epanechnikov kernel function and a bandwidth of 0.06.[11] In addition, when computing the weights, we perform exact matching on survey year and overweight status in $t-1$ (e.g. Marcus 2014). This ensures 1) individuals who were laid off are matched with controls from the same macroeconomic time period, controlling for macroeconomic trends that might be highly correlated with job loss, and 2) treated individuals are grouped with non-treated individuals in the same pre-treatment BMI range, or individuals are matched based on whether or not they were above or below the Centers for Disease Control and Prevention (CDC) cut-off for overweight status (BMI>=25).

In the final step, we incorporate the weights from propensity score matching into the DD regression model. Our linear DD model takes on the following form:

$$E[BMI_t|W(X)] = \alpha BC_{it-1} + \gamma PGS_i + \delta BC_{it-1}\times PGS_i + \varphi BMI_{t-1} + X_{t-1}\beta \qquad (5)$$

Where BC is an indicator for job loss due to a business closing between waves $t-1$ and t for individual i and X is the vector of observable time variant and invariant characteristics measured at $t-1$ from propensity score matching, including the first four principal components for population stratification in the genotype data. Including the genotype fixed effect both controls for unobserved biological differences across individuals and captures any variance in treatment intensity by genotype. In addition, we include BMI_{t-1} to model changes in BMI or to control for BMI at baseline. The corresponding parameters from the regression equation can be used to estimate the conditional mean functions for treated and untreated individuals as follows

[11] Applying different bandwidths did not significantly change our estimates. Results are available from the authors upon request.

$$E[BMI_t|W(X), BC = 1, PGS = 1] = \alpha + \gamma + \delta + \varphi + X_{t-1}\beta \quad (6)$$

$$E[BMI_t|W(X), BC = 1, PGS = 0] = \alpha + \varphi + X_{t-1}\beta$$

$$E[BMI_t|W(X), BC = 0, PGS = 1] = \gamma + \varphi + X_{t-1}\beta$$

$$E[BMI_t|W(X), BC = 0, PGS = 0] = \varphi + X_{t-1}\beta$$

Where, since the PGS is standardized to have a mean of zero and a standard deviation of one, a PGS=0 or PGS=1 indicates individuals with an average or high risk PGS, respectively. From here, the ATT can be estimated by taking the difference in $E[BMI_t|W(X)]$ between treated and non-treated individuals

$$ATT_{PGS=1} = E[BMI_t|W(X), BC = 1] - E[BMI_t|W(X), BC = 0] = \alpha + \delta \quad (7)$$

$$ATT_{PGS=0} = E[BMI_t|W(X), BC = 1] - E[BMI_t - BMI_{t-1}|W(X), BC = 0] = \alpha$$

In addition, we also estimate models that include a three-way interaction term with overweight status in t-1. This differences-in-differences-in-differences (DDD) model takes into account the possibility that the impact of job loss and genotype on BMI varies by whether or not an individual was overweight (BMI>=25) prior to losing their job. Research has shown that particularly in the extremes of BMI distribution, there may be etiologically distinct subgroups (Harismendy et al. 2010). Although analyzing the full distribution is generally more powerful, when treatment heterogeneity is present, constructing separate slopes for different BMI subgroups may increase power (Pütter et al. 2011). For these models, we extend our strategy above to include an additional interaction term and calculate the ATT separately for underweight/normal weight individuals (BMI<25) and overweight/obese individuals (BMI>=25).

Finally, when estimating the variance of propensity score-based parameters, the literature is split on whether to account for uncertainty in the propensity score estimation (for a review see Stuart 2010). Typically, bootstrapping is used to estimate standard errors when they are difficult to compute analytically or when the theoretical distribution of the statistic is unknown. However, under general conditions studies have found using estimated rather than true propensity scores leads to an overestimation of the variance and thus more conservative estimates of the standard errors (Rubin and Stuart 2006; Rubin and Thomas 1996). Further, Abadie and Imbens (2006) show bootstrapping fails in the case of nearest-neighbor matching. Therefore, instead of bootstrapping, we follow Marcus (2014) and use robust standard errors from the weighted regressions.

Matching Quality and Descriptive Statistics

Table 4 shows the means and matching statistics for all covariates by the treatment group, control group, and matched control group. We compare the means of the treated and control groups before and after matching to assess the quality of the matching procedure. Before matching, differences between covariates are expected, however after matching the covariates should be balanced with little to no significant differences remaining. We include both the standardized bias and two-sample t-Tests for equality of the means to check for significant differences between covariates for both groups (Rosenbaum and Rubin 1985). The standardized bias compares the distance between the marginal distributions, or the difference in sample means between the treated ($\bar{X}_T$) and matched control ($\bar{X}_C$) subsamples as a percentage of the square root of the average of the sample variances in both groups for a covariate X:

$$SB_x = 100 \cdot \frac{\bar{X}_T - \bar{X}_C}{\sqrt{0.5 \cdot (\sigma_{XT}^2 - \sigma_{XC}^2)}} \quad (8)$$

Generally, a standardized bias at or below 5% (after matching) is seen as sufficient (Caliendo and Kopeinig 2008).

Before matching, individuals affected by a business closure have lower socioeconomic standing and worse health behaviors and outcomes than continuously employed individuals. Treated individuals were 17.8 percentage points less likely to have a college degree, are more likely to reside in the South, and have lower household income. Labor statistics show they are more likely to work part time, for smaller firms, in the agriculture/fishing/farming, construction/mining, trade, or miscellaneous service industries, are less likely to be white collar, and have lower job tenure than individuals in the control group. Treated individuals were also 23.7 percentage points less likely to have health insurance and are 9.3 percentage points more likely to be diagnosed with a psychiatric condition. They are 10.8 percentage points more likely to have ever been regular smokers, and among current smokers, smoke almost three cigarettes more per day than continuously employed individuals. In terms of weight status, there do not appear to be any significant differences between the two groups, however the BMI PGS is slightly higher for workers who lost their job before matching.

By and large, after matching the discrepancies between treated and control groups means, as measured by the t-Tests, largely disappear, and the standardized biases are at or below 5%. Notable exceptions include job tenure, trade and public administration industries, college education, household income, and access to health insurance. However, since we also adjust for these same covariates in our empirical model, any small remaining differences on these variables are further diminished via regression adjustment.

Table 4. Before treatment means of treated, controls, and matched controls

	Match status	Means		%bias	t-test	
		Treated	Control		t	p>t
Weight status						
BMI	U	27.740	27.641	1.800	0.310	0.756
	M		27.711	-0.800	-0.090	0.932
BMI PGS	U	0.112	-0.006	11.600	1.910	0.057
	M		0.077	6.600	0.730	0.465
Overweight	U	0.695	0.684	2.300	0.360	0.716
	M		0.698	0.000	0.000	1.000
Demographic						
Female	U	0.560	0.554	1.300	0.200	0.839
	M		0.562	-2.700	-0.290	0.771
Age	U	55.465	55.480	-0.500	-0.080	0.934
	M		55.556	-9.500	-1.040	0.300
Married	U	0.811	0.832	-5.600	-0.930	0.351
	M		0.834	-6.800	-0.730	0.465
Education						
No degree	U	0.105	0.062	15.700	2.880	0.004
	M		0.109	-1.000	-0.100	0.924
High school degree	U	0.669	0.534	27.800	4.380	0.000
	M		0.639	6.900	0.760	0.445
College degree	U	0.225	0.404	-39.100	-5.920	0.000
	M		0.252	-6.700	-0.780	0.434
Regional Census division						
Northeast	U	0.149	0.186	-9.800	-1.520	0.128
	M		0.154	-1.400	-0.160	0.871
Midwest	U	0.316	0.320	-0.800	-0.130	0.897
	M		0.330	3.200	0.340	0.734
South	U	0.415	0.328	17.900	2.960	0.003
	M		0.382	0.200	0.030	0.979
West	U	0.120	0.166	-13.100	-2.010	0.045
	M		0.134	-3.000	-0.340	0.736
Labor Market/Socioeconomic Status						
Household income (log)	U	10.953	11.353	-34.100	-7.140	0.000
	M		11.145	-10.100	-1.180	0.239
Household wealth ($100k)	U	3.684	3.510	2.100	0.340	0.737
	M		3.295	0.800	0.080	0.939
Firm size<=4	U	0.095	0.027	28.500	6.410	0.000
	M		0.072	9.100	0.840	0.400
Firm size 5-14	U	0.116	0.054	22.500	4.380	0.000
	M		0.128	2.800	0.250	0.802
Firm size 15-24	U	0.055	0.025	15.200	2.990	0.003
	M		0.052	3.700	0.340	0.737
Firm size 25-99	U	0.098	0.101	-1.000	-0.160	0.873
	M		0.109	1.800	0.190	0.852

Firm size 100-499	U	0.102	0.182	-23.200	-3.410	0.001
	M		0.128	-2.500	-0.290	0.774
Firm size>=500	U	0.331	0.599	-55.800	-8.880	0.000
	M		0.399	-2.500	-0.270	0.790
Missing firm size info	U	0.204	0.011	65.200	22.950	0.000
	M		0.111	-7.200	-0.760	0.446
Part time	U	0.178	0.120	16.300	2.860	0.004
	M		0.156	4.000	0.420	0.675
Industry						
Agriculture/Fishing/Farming	U	0.018	0.005	12.600	2.980	0.003
	M		0.008	4.300	0.480	0.628
Construction/Mining	U	0.065	0.031	16.000	3.120	0.002
	M		0.062	-4.900	-0.490	0.624
Manufacturing	U	0.211	0.184	6.900	1.140	0.254
	M		0.224	2.500	0.260	0.794
Trade	U	0.269	0.113	40.400	7.780	0.000
	M		0.227	10.700	1.040	0.299
Public Services	U	0.076	0.078	-0.500	-0.080	0.938
	M		0.078	1.200	0.120	0.902
Finance/Insurance/Real Estate	U	0.055	0.063	-3.600	-0.570	0.569
	M		0.053	2.700	0.290	0.769
Public Administration	U	0.004	0.072	-36.500	-4.390	0.000
	M		0.024	-10.400	-1.800	0.072
Misc. Services	U	0.236	0.445	-45.000	-6.820	0.000
	M		0.282	-4.800	-0.540	0.588
Missing industry info	U	0.065	0.010	29.600	8.230	0.000
	M		0.043	-7.000	-0.770	0.444
Occupation						
White Collar	U	0.615	0.708	-19.700	-3.300	0.001
	M		0.621	1.000	0.110	0.914
Blue Collar	U	0.240	0.204	8.600	1.430	0.151
	M		0.258	-0.500	-0.060	0.955
Service	U	0.087	0.082	1.900	0.310	0.757
	M		0.088	3.600	0.380	0.707
Missing occupation info	U	0.058	0.006	30.000	9.390	0.000
	M		0.034	-7.200	-0.830	0.406
Job tenure	U	7.946	15.943	-80.100	-12.350	0.000
	M		10.529	-19.400	-2.200	0.028
Missing tenure info	U	0.055	0.003	31.600	12.040	0.000
	M		0.042	-14.900	-1.580	0.115
Health status						
Health excellent/very good	U	0.611	0.649	-7.900	-1.300	0.194
	M		0.588	4.300	0.450	0.650
Health insurance	U	0.556	0.793	-52.100	-9.330	0.000
	M		0.644	-10.600	-1.070	0.286
Exercise vigorously 3+ times/week	U	0.371	0.356	3.100	0.510	0.610

	M		0.374	0.200	0.020	0.985
Missing exercise info	U	0.004	0.005	-1.400	-0.220	0.824
	M		0.004	0.500	0.060	0.953
Ever smoke cigarettes	U	0.636	0.528	22.100	3.530	0.000
	M		0.628	-0.600	-0.060	0.949
Cigarettes per day	U	5.840	3.079	29.300	5.410	0.000
	M		5.735	1.100	0.100	0.919
Missing cigarettes per day info	U	0.004	0.004	0.000	0.000	0.998
	M		0.002	4.300	0.520	0.606
Drinks alcohol	U	0.636	0.674	-7.900	-1.300	0.194
	M		0.603	4.600	0.490	0.624
Doctor diagnosed psychiatric issue	U	0.167	0.110	16.600	2.940	0.003
	M		0.159	-5.300	-0.550	0.580
Missing psychiatric info	U	0.004	0.003	0.900	0.160	0.876
	M		0.001	-2.200	-0.550	0.585
Survey year						
1994	U	0.069	0.152	-26.700	-3.780	0.000
	M		0.081	0.000	0.000	1.000
1996	U	0.120	0.124	-1.200	-0.190	0.846
	M		0.128	0.000	0.000	1.000
1998	U	0.175	0.100	21.700	3.950	0.000
	M		0.140	0.000	0.000	1.000
2000	U	0.153	0.120	9.500	1.610	0.107
	M		0.179	0.000	0.000	1.000
2002	U	0.175	0.091	24.900	4.640	0.000
	M		0.170	0.000	0.000	1.000
2004	U	0.058	0.067	-3.600	-0.570	0.571
	M		0.064	0.000	0.000	1.000
2006	U	0.069	0.113	-15.200	-2.260	0.024
	M		0.072	0.000	0.000	1.000
2008	U	0.062	0.102	-14.800	-2.180	0.029
	M		0.055	0.000	0.000	1.000
2010	U	0.095	0.077	6.300	1.060	0.287
	M		0.089	0.000	0.000	1.000
2012	U	0.025	0.054	-14.600	-2.070	0.039
	M		0.021	0.000	0.000	1.000
Principal components						
1	U	0.000	0.000	5.300	0.620	0.534
	M		0.000	2.200	0.400	0.688
2	U	0.000	0.000	2.900	0.340	0.737
	M		0.000	0.700	0.130	0.894
3	U	0.000	0.000	-3.300	-0.380	0.702
	M		0.000	-1.200	-0.220	0.823
4	U	0.000	0.000	-2.500	-0.300	0.766
	M		0.000	-0.700	-0.130	0.897

Notes: Analytic sample consists of white, non-Hispanic male and female workers who were not self-employed and between the ages of 50-60 in t-1.

Results

Differences-in-Differences (DD) Model

Table 5 shows the results from the DD model for the effect of a business closure on BMI across several different models and propensity score matching procedures. Specifications (1)-(2) do not control for baseline BMI, while specifications (3)-(5) model the change in BMI, or control for BMI in t-1. Specification (1) does not control for the BMI PGS while specification (2) shows the basic G × E results in the cross-section. Specification (4) performs exact matching on survey year and overweight status in t-1 and specification (5) additionally includes all conditioning variables from propensity score matching as controls in the regression analysis. We also report the number of individuals in the treatment group, the median standardized bias, and the percent of individuals off common support, or the share of individuals who are not considered in estimation due to inappropriate matches, for each specification.

The results in (1) are similar in magnitude and direction to the HRS results reported by Deb et al. (2011), which find no main effect from business closures on BMI. However, while matching on genotype and exact matching on survey year and baseline overweight status both substantially reduce median standardized bias, we find no evidence of a G × E effect in the DD model. After adjusting for all covariates in specification (5), we do find a slight protective effect for individuals who did not lose their job, indicating steady employment at older ages might actually curb weight gain among high-risk BMI genotypes. Specifically, the BMI PGS coefficient, which represents the effect of genotype on BMI for individuals who did not lose their job, is negative and significant at the 10% level.

Table 5. The effect of a business closure on BMI by genotype

	Cross-sectional (BMI)		Longitudinal (Change in BMI)		
	Without PGS	With PGS	Not in cells	In cells	Regression-adjusted
	(1)	(2)	(3)	(4)	(5)
Business closed	0.303	0.0516	-0.0259	0.0123	-0.0459
	(0.404)	(0.399)	(0.159)	(0.146)	(0.135)
BMI PGS		0.250	-0.000344	-0.0448	-0.106†
		(0.181)	(0.0713)	(0.0502)	(0.0543)
Business closed × BMI PGS		0.358	0.0236	0.111	0.164
		(0.351)	(0.143)	(0.150)	(0.144)
BMI (t-1)			0.915***	0.925***	0.894***
			(0.0286)	(0.0275)	(0.0385)
Overweight (t-1)					0.536*
					(0.277)
N	5758	5756	5756	5664	5664
Treated (50-60)	272	270	270	235	235
Off common support (%)	1.09	1.82	1.82	14.55	14.55
Median standardized bias	4.3	3.3	3.2	2.5	2.5

Notes: Analytic sample consists of white, non-Hispanic male and female workers who were not self-employed and between the ages of 50-60 at t-1. Coefficients in each column are from separate regressions. Specifications (1)-(2) do not control for baseline BMI, while specifications (3)-(5) control for BMI in t-1. Specification (4) performs exact matching on survey year and overweight status in t-1 and specification (5) additionally includes all conditioning variables from the propensity score matching in Table 1 as controls in the regression analysis. The 'off common support' row reports the share of treated individuals who are not considered in the estimation due to inappropriate matches. Individuals are considered overweight if their BMI in t-1 was greater than or equal to 25. Robust standard errors are in parentheses. †p<.10; * p<.05; ** p<.01; ***p<.001.

Heterogeneity Analysis by BMI Subgroup

There is no *a priori* reason to believe that individuals in different BMI subgroups will respond the same to the stress of job loss. Obese or overweight individuals may consume more food in response to stress, but it is also possible that chronic stress may trigger weight gain in otherwise normal weight, genetically-at-risk individuals. To investigate whether the G × E effect differs by BMI subgroup, we estimate DDD models that include a three-way interaction between the treatment variable (business closure), the BMI PGS, and a dichotomous indicator for whether or not an individual was overweight prior to losing their job. Here, the "Overweight" variable is set equal to one if an individual is at or above the CDC BMI cutoff of 25 in $t-1$, and zero otherwise. In addition, we assess the potential moderating roles of the pretreatment covariates

age, gender, prior health status, and socioeconomic status (i.e. education, occupation, and household wealth) within this framework.

The DDD results are reported in Table 6 by age group and sex and in Table 7 by socioeconomic status and self-reported health. To facilitate accurate comparisons, in addition to exact matching on survey year and overweight status, we also perform exact matching on each subgroup variable. In other words, we apply the same three-step procedure, but compute the weights separately for each combination of year, overweight status, and grouping variable.

Table 6. Effect of a business closure, BMI genotype, and overweight status on changes in BMI by age and sex

	Age (t-1)		Sex	
	50-60	50-63	Male	Female
	(1)	(2)	(3)	(4)
Business closure	-0.0723	-0.0781	0.408	-0.341
	(0.189)	(0.176)	(0.364)	(0.220)
PGS	-0.0835	-0.0850	-0.190	0.00518
	(0.0913)	(0.0768)	(0.140)	(0.111)
Business closure × PGS	0.458*	0.336	0.953**	0.207
	(0.215)	(0.204)	(0.332)	(0.218)
Overweight (t-1)	0.514†	0.569*	0.334	0.898*
	(0.281)	(0.246)	(0.271)	(0.425)
Business closure × Overweight (t-1)	0.0691	-0.0226	-0.419	0.325
	(0.263)	(0.242)	(0.421)	(0.401)
Overweight (t-1) × PGS	-0.0303	0.0502	-0.00847	-0.00796
	(0.113)	(0.103)	(0.163)	(0.148)
Business closure × Overweight (t-1) × PGS	-0.438	-0.428	-0.996**	-0.271
	(0.292)	(0.275)	(0.373)	(0.400)
BMI (t-1)	0.895***	0.902***	0.946***	0.839***
	(0.0376)	(0.0322)	(0.0309)	(0.0530)
N	5664	6707	2527	3137
R^2	0.875	0.873	0.905	0.869

The table presents the effect of a business closure on changes in body weight by age group in t-1 and sex. The analytic sample consists of white, non-Hispanic male and female workers who were not self-employed and between the ages of 50-60 at t-1 in columns (1) and (3)-(4) or between the ages of 50-63 at t-1 in column (2). Regressions include controls for population stratification in the genotype data as well as all controls listed in Table 1. Robust standard errors are in parentheses. †p<.10; * p<.05; ** p<.01; ***p<.001.

Table 7. Effect of a business closure, BMI genotype, and overweight status on changes in BMI by socioeconomic or demographic subgroup in t-1

	Education		Occupational Status		Marital Status		Health Status		Household Wealth	
	College	No college	White collar	Blue collar	Married/ Partnered	Not Married/ Partnered	Excellent/ Very Good	Good/Fair/ Poor	Above Median	Below Median
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Business closure	0.113	-0.0919	-0.355	-0.00316	-0.0245	0.951*	-0.305	0.455	0.116	-0.263
	(0.491)	(0.215)	(0.236)	(0.270)	(0.202)	(0.395)	(0.213)	(0.433)	(0.237)	(0.273)
PGS	-0.275	-0.0800	-0.196†	0.0176	-0.0490	0.265	0.129	-0.210	-0.00658	-0.161
	(0.186)	(0.111)	(0.106)	(0.126)	(0.0830)	(0.237)	(0.0946)	(0.128)	(0.106)	(0.145)
Business closure × PGS	0.827*	0.578†	0.770**	-0.457	0.268	0.405	0.303	0.742†	0.0445	1.072**
	(0.323)	(0.310)	(0.247)	(0.415)	(0.221)	(0.406)	(0.258)	(0.395)	(0.236)	(0.376)
Overweight (t-1)	0.551	0.385	0.528	0.371	0.656*	-0.0132	0.600	0.201	0.682	0.292
	(0.490)	(0.247)	(0.358)	(0.317)	(0.301)	(0.372)	(0.405)	(0.290)	(0.522)	(0.282)
Business closure × Overweight (t-1)	0.219	0.115	0.269	0.442	0.182	-1.375*	0.409	-0.450	-0.409	0.323
	(0.543)	(0.303)	(0.306)	(0.460)	(0.289)	(0.542)	(0.327)	(0.500)	(0.375)	(0.342)
Overweight (t-1) × PGS	0.313	-0.101	0.148	-0.0947	0.0109	-0.357	-0.157	0.0762	0.0134	-0.0243
	(0.228)	(0.134)	(0.138)	(0.194)	(0.112)	(0.303)	(0.128)	(0.163)	(0.158)	(0.169)
Business closure × Overweight (t-1) × PGS	-1.028*	-0.446	-0.758*	0.387	-0.431	0.405	-0.492	-0.243	0.0476	-0.995*
	(0.509)	(0.390)	(0.361)	(0.470)	(0.301)	(0.429)	(0.362)	(0.434)	(0.357)	(0.457)
BMI (t-1)	0.838***	0.919***	0.866***	0.920***	0.873***	0.993***	0.867***	0.931***	0.865***	0.921***
	(0.0660)	(0.0305)	(0.0518)	(0.0321)	(0.0404)	(0.0327)	(0.0619)	(0.0272)	(0.0818)	(0.0282)
N	1678	3353	3847	1412	4671	442	3399	1857	2399	2703
R^2	0.909	0.878	0.880	0.896	0.874	0.956	0.859	0.908	0.866	0.895

Notes: The table presents the effect of a business closure on changes in body weight by various subgroups. Analytic sample consists of white, non-Hispanic male and female workers who were not self-employed and between the ages of 50-60 at t-1. In addition to survey year and overweight status, exact matching is also performed on the subgroup variable. Regressions include controls for population stratification in the genotype data as well as the covariates listed in Table 1. Robust standard errors are in parentheses. †p<.10; * p<.05; ** p<.01; ***p<.001.

Following the logic outlined in Eq. 6, the coefficients reported in both tables represent the following marginal treatment effects:

Table 8. Interpretation of Marginal Treatment Effects from the DDD Model

Coefficient	Business closed	PGS	Overweight
Business closure	1	0	0
PGS	0	1	0
Business closure × PGS	1	1	0
Overweight	0	0	1
Business closure × Overweight (t-1)	1	0	1
Overweight (t-1) × PGS	0	1	0
Business closure × Overweight (t-1) × PGS	1	1	1

Notes: The PGS is standardized to have a mean of zero (PGS=0) and a standard deviation of one (PGS=1).

Where our main coefficients of interest are "Business closure × PGS" and "Business closure × Overweight (t-1) × PGS", which represent the marginal effect of a business closure for individuals with a high-risk genotype (PGS=1) who were either normal weight/underweight (BMI<25) or overweight/obese (BMI>=25), respectively.[12]

Results show significant heterogeneity by weight category for various demographic and socioeconomic subgroups. Overall, we find the G × E effect is significant for genetically-at-risk individuals who were *not* overweight prior to job loss. Specifically, we find stronger effects for normal weight individuals who are between ages 50-60, male, college educated, white collar, in poorer health, and who are below the median in household wealth. Notably, results in Table 6 reveal the effect on BMI is only significant for men and for individuals who are not eligible for early Social Security claiming. For every one standard deviation increase in the BMI PGS, the BMI of all individuals aged 50-61 prior to job loss increased by an average of 0.458 kg/m^2 after a business closure and by 0.953 kg/m^2 for men. This result is in line with multiple studies that

[12] Less than 1% of individuals in our analytic sample report being underweight, or have a BMI<18.5.

have linked unemployment to poorer health behaviors in men (for a review see Roelfs et al., 2011), as well as evidence from the Whitehall II study and other longitudinal studies that suggest chronic life stressors affect weight gain more in men than women (Kivimäki et al. 2006; Korkeila et al. 1998; Van Strien et al. 1985). Particularly among the older birth cohorts we observe in the HRS, work may be more crucial to the identity of men, eliciting a stronger stress response. The stress of displacement from a business closure may be further compounded if individuals have a harder time finding reemployment and/or are not yet eligible for retirement benefits.

Using the marginal treatment effects from these regressions, we extend the logic presented in Eq. 7 and estimate the ATT for individuals with a PGS=1 or PGS=2 by overweight status and subgroup (Table 9). We fail to find a significant ATT for all individuals aged 50-60, but do find a significant ATT for high-risk men. Men who were not overweight prior to job loss gain approximately 1.238 to 2.386 kg/m^2 after displacement. To contextualize this finding, for a hypothetical male who is 5'11''and weighs 170 lbs. prior to job loss (BMI=23.71), this is equivalent to a 9-17 lb. weight gain, which is enough to place them in the overweight category post job loss. We also find significant ATTs for college educated and white collar workers two standard deviations out in polygenic risk (PGS=2). This could in part reflect the loss of a major social role or a stronger social stigma from job loss among high SES workers. Particularly at older ages, individuals with a higher social standing may have greater employment commitment and stronger social networks culled over the lifetime of their career, exacerbating the emotional impact of job loss (e.g. Hayes and Nutman 1981; Turner 1995).

Table 9. Treatment effects by overweight status and genotype for various demographic and socioeconomic subgroups

		Not Overweight		Overweight	
Subgroup (t-1)	N	PGS=1	PGS=2	PGS=1	PGS=2
Age group					
50-60	235	0.386	0.844	0.0165	0.0363
		(0.326)	(0.518)	(0.245)	(0.403)
50-63	293	0.258	0.594	-0.193	-0.286
		(0.323)	(0.511)	(0.233)	(0.378)
Sex					
Male	98	1.238*	2.386**	0.0436	0.0550
		(0.537)	(0.827)	(0.242)	(0.371)
Female	124	-0.286	-0.113	0.00798	0.00236
		(0.316)	(0.502)	(0.418)	(0.680)
Education					
College	46	0.940	1.768*	0.131	-0.0693
		(0.637)	(0.882)	(0.377)	(0.643)
No college	181	0.486	1.064	0.156	0.288
		(0.419)	(0.705)	(0.287)	(0.471)
Occupational status					
White collar	142	0.415	1.186*	-0.0736	-0.0616
		(0.383)	(0.600)	(0.332)	(0.549)
Blue collar	79	-0.461	-0.918	0.368	0.298
		(0.493)	(0.871)	(0.370)	(0.563)
Marital status					
Married/Partnered	187	0.243	0.511	-0.00559	-0.168
		(0.339)	(0.536)	(0.252)	(0.414)
Not Married/Partnered	34	1.356*	1.762†	0.387	1.197*
		(0.573)	(0.911)	(0.332)	(0.516)
Health Status					
Excellent/Very Good	136	-0.00222	0.300	-0.0846	-0.274
		(0.362)	(0.591)	(0.345)	(0.552)
Good/Fair/Poor	88	1.197	1.938†	0.504†	1.003*
		(0.731)	(1.092)	(0.279)	(0.463)
Household wealth					
Above median	96	0.161	0.205	-0.201	-0.109
		(0.392)	(0.603)	(0.325)	(0.542)
Below median	130	0.809†	1.881*	0.137	0.214
		(0.478)	(0.816)	(0.351)	(0.577)

Notes*:* The table reports the effect of job loss on BMI by subgroup. Each row contains the ATT for the subgroup by genotype and overweight status prior to job loss. The number of treated observations on common support for each subgroup is displayed in the first column. In addition to survey year and overweight status, exact matching is also performed on the subgroup variable. With the exception of the 50-63 year old age group, ATT is calculated for white, non-Hispanic male and female workers who were not self-employed and between the ages of 50-60 at t-1. Regressions include controls for population stratification in the genotype data as well as all covariates listed in Table 1. Robust standard errors are in parentheses. †p<.10; * p<.05; ** p<.01; ***p<.001.

Not surprisingly, we find individuals who are not married/partnered at the time of job displacement—both in the normal weight and overweight categories—are more likely to gain weight. Spouses often provide financial and emotional support, mitigating strain from a job loss. Furthermore, normal weight workers below the median in household wealth gain 0.809-1.881 kg/m^2 more than individuals who are not unemployed, a finding that resonates with Gallo et al. (2006) who find involuntary job loss in the HRS is associated with depression symptoms among individuals with below median net worth only. Finally, we also find some evidence that individuals in the lowest self-rated health categories (good, fair, or poor) are significantly more likely to gain weight after a job loss, perhaps in part because job loss aggravates pre-existing adverse health behaviors (Deb et al. 2011).

Endogenous Job Loss

As a counterpoint to our business closure estimates, we also present findings from job loss due to quitting, poor health, or lay offs/firings. In all three cases, job loss is either tied to worker health, or may be linked to worker characteristics or worker incompetence, potentially biasing findings. Particularly with the addition of genotype data, genes may be acting as proxies for other unobserved rGE or G × E phenomena. For all three specifications, we use the same DDD matching strategy outlined above, with the exception that an individual is placed in the respective treatment group if they report no longer working for their previous wave employer because they quit, were in poor health, or were laid off/fired from their job.

Table 10. Effect of job loss due to quitting, poor health, or a lay off on BMI

	Quit	Poor Health	Laid Off
	(1)	(2)	(3)
Treatment	-0.350*	0.525	0.276
	(0.175)	(0.382)	(0.294)
PGS	-0.0719	-0.0977	-0.133
	(0.0853)	(0.129)	(0.102)
Treatment × PGS	0.0103	-0.895*	-0.0945
	(0.162)	(0.446)	(0.364)
Overweight (t-1)	0.206	0.161	0.0603
	(0.230)	(0.339)	(0.246)
Treatment × Overweight (t-1)	0.166	-0.235	-0.0971
	(0.269)	(0.457)	(0.345)
Overweight (t-1) × PGS	0.0159	-0.00199	0.140
	(0.112)	(0.187)	(0.128)
Treatment × Overweight (t-1) × PGS	0.282	0.531	0.0717
	(0.266)	(0.566)	(0.398)
BMI (t-1)	0.923***	0.936***	0.967***
	(0.0272)	(0.0285)	(0.0259)
N	5337	4797	5559
R^2	0.880	0.890	0.857

Notes: The table presents the effect of endogenous job loss on changes in body weight. "Treatment" referes to the specific type of job loss in the corresponding column. Analytic sample consists of white, non-Hispanic male and female workers who were not self-employed and between the ages of 50-60 at t-1. Regressions include controls for population stratification in the genotype data as well as the covariates listed in Table 1. Robust standard errors are in parentheses. †p<.10; * p<.05; ** p<.01; ***p<.001..

The results presented in Table 10 paint a considerably different picture from the G × E model with business closures. We find no evidence of a G × E interaction for high-risk genotypes who quit or were laid off. There is evidence of a small but significant weight loss (0.350 kg/m2) for individuals with average polygenic risk who quit or left their job voluntarily. For individuals who report leaving their job due to poor health, we find evidence of a G × E interaction that goes in the opposite direction of individuals who were displaced from a business closure. A one standard deviation increase in genetic risk reduces BMI by 0.895 kg/m^2 for workers in poor health. This outcome is not surprising given individuals afflicted with chronic,

debilitating, or life-threatening illnesses at older ages generally experience significant weight loss. Overall, these findings underscore the complexity of the social and biological mechanisms surrounding job loss and the importance of addressing selection bias in the environmental exposure to accurately capture G × E effects in observational data.

Discussion

We find evidence that job displacement from a business closure at older ages exacerbates polygenic risk for weight gain in otherwise normal or healthy weight subgroups. Men aged 50-60 years one to two standard deviations out in polygenic risk gained 1.238-2.386 kg/m^2 more than comparable men who were not displaced from their jobs. This translates to a 9-17 pound weight gain for a hypothetical 5'11'' male weighing 170 pounds before job loss. Individuals who gained weight after a job loss were also more likely to be in worse health, single, and at the bottom half of the wealth distribution. In the long term, because weight gain is progressive and weight loss difficult to maintain, weight gain of this magnitude could have substantial long-term effects on cardiovascular health.

The high unemployment rate and tremendous job destruction experienced during the Great Recession have left an anemic labor market in its wake. More than five years after the end of the Great Recession, both mean and median duration of unemployment spells remain at unprecedented levels (Farber 2015). Older adults in particular have faced longer stretches of unemployment and steeper wage losses once reemployed. Half of unemployed adults aged 50 to 61 experienced more than nine months of job search during the Great Recession, compared to six months for workers aged 25 to 34; similarly, median monthly earnings declined 23 percent after an unemployment spell for reemployed workers aged 50 to 61, compared with just 11 percent for

workers aged 25 to 34 (Johnson and Butrica 2012). The substantial increase in the size of the older workforce and the severity of the recent recession makes understanding the effects of job loss on BMI crucial to deciphering current trends in cardiovascular health in the aging US population.

Limitations of these analyses should be mentioned. In general, there is significant complexity surrounding obesity and aging such that differences in BMI may not indicate an actual change in body fat. Higher BMI at midlife is a risk factor for age-related disease and early mortality, however at older ages it might be somewhat protective of mortality because age-related diseases and aging itself are wasting conditions that induce significant weight loss. Therefore, while incrementally higher BMI in midlife is more likely a measure of risk for disease, later in life it may actually signal the absence of disease. In addition, individuals generally lose muscle mass with increasing chronological age, meaning older individuals could maintain a constant BMI while simultaneously losing lean body mass and gaining a greater portion of adiposity (Kyle et al. 2003). However, if anything we would expect any increases in BMI from a job loss alongside these countervailing trends to bias coefficients downward, which may in part explain the null findings we report for females and other subgroups (e.g. workers over age 62) in our sample.

In addition, the relatively nominal findings we report for the entire population may in part reflect a greater culmination of environmental and lifestyle factors on adiposity in older adults that overwhelm any genetic effects. The genomic influence on BMI has been shown to both weaken over the life course and increase in magnitude since the current obesity epidemic began in the mid-1980s (Guo et al. 2015). While performing exact matching on survey year may in part account for differences in the obesogenic environment across cohorts, our sample size of

treated individuals is too small to explore more detailed cohort analysis. Thus, similar studies that are able to test the impact of job loss on genetic moderation of anthropomorphic traits in a larger sample of both younger and older populations are needed. In particular, identifying when in the life course job loss and genetic variants affect body weight the most may inform public health initiatives that target unemployed persons for more aggressive cardiovascular screening and interventions aimed at reducing long-term weight gain.

References

Abadie, A., & Imbens, G. W. (2006). Large sample properties of matching estimators for average treatment effects. *Econometrica, 74*(1), 235-267.

Almén, M. S., Jacobsson, J. A., Moschonis, G., Benedict, C., Chrousos, G. P., Fredriksson, R., & Schiöth, H. B. (2012). Genome wide analysis reveals association of a FTO gene variant with epigenetic changes. *Genomics, 99*(3), 132-137.

Bell, C. G., Finer, S., Lindgren, C. M., Wilson, G. A., Rakyan, Vardhman K., Teschendorff, Andrew E., . . . Prokopenko, I. (2010). Integrated genetic and epigenetic analysis identifies haplotype-specific methylation in the FTO type 2 diabetes and obesity susceptibility locus. *PloS One, 5*(11), e14040.

Bender, K. A., & Jivan, N. A. (2004). *What Makes Retirees Happy?* Center for Retirement Research at Boston College.

Bhaskaran, K., Douglas, I., Forbes, H., dos-Santos-Silva, I., Leon, D. A., & Smeeth, L. (2014). Body-mass index and risk of 22 specific cancers: a population-based cohort study of 5· 24 million UK adults. *The Lancet, 384*(9945), 755-765.

Boardman, J. D., Roettger, M. E., Domingue, B. W., McQueen, M. B., Haberstick, B. C., & Harris, K. M.. (2012). Gene–environment interactions related to body mass: School policies and social context as environmental moderators. *Journal of Theoretical Politics, 24*(3), 370-388.

Bonsang, E., & Klein, T. J. (2012). Retirement and subjective well-being. *Journal of Economic Behavior & Organization, 83*(3), 311-329.

Brand, J. E. (2004). *Enduring Effects of Job Displacement on Career Outcomes* (Doctoral Dissertation). Citeseer.

Burgard, S. A., Brand, J. E., & House, J. S. (2007). Toward a better estimation of the effect of job loss on health. *Journal of Health and Social Behavior, 48*(4), 369-384.

Caliendo, M., & Kopeinig, S.. (2008). Some practical guidance for the implementation of propensity score matching. *Journal of Economic Surveys, 22*(1), 31-72.

Chang, C. C., Chow, C. C., Tellier, L.C. Vattikuti, S., Purcell, S. M., & Lee, J. J. (2015). Second-generation PLINK: rising to the challenge of larger and richer datasets. *Gigascience, 4*(7).

Coe, N. B., & Zamarro, G. (2011). Retirement effects on health in Europe. *Journal of Health Economics, 30*(1), 77-86.

Conley, D. (2009). The promise and challenges of incorporating genetic data into longitudinal social science surveys and research. *Biodemography and Social Biology, 55*(2), 238-251.

Deb, P., Gallo, W. T., Ayyagari, P., Fletcher, J. M., & Sindelar, J. L. (2011). The effect of job loss on overweight and drinking. *Journal of Health Economics, 30*(2), 317-327.

Dehejia, R. H., & Wahba, S. (1999). Causal Effects in Nonexperimental Studies: Reevaluating the Evaluation of Training Programs. *Journal of the American statistical Association, 94*(448), 1053-1062. doi: 10.2307/2669919

Dudbridge, F. (2013). Power and predictive accuracy of polygenic risk scores. *PLoS Genet, 9*(3), e1003348.

Falba, T., Teng, H., Sindelar, J. L., & Gallo, W. T. (2005). The effect of involuntary job loss on smoking intensity and relapse. *Addiction, 100*(9), 1330-1339.

Farber, H. S. (2004). Job loss in the United States, 1981-2001. *Research in Labor Economics, 23*, 69-117.

Farber, H. S. (2015). Job loss in the Great Recession and its aftermath: US evidence from the displaced workers survey: National Bureau of Economic Research.

Fletcher, J. M., & Conley, D. (2013). The Challenge of Causal Inference in Gene–Environment Interaction Research: Leveraging Research Designs From the Social Sciences. *American Journal of Public Health, 103*(S1), S42-S45.

Gallo, W. T., Bradley, E. H., Dubin, J. A., Jones, R. N., Falba, T. A., Teng, H., & Kasl, S. V. (2006). The persistence of depressive symptoms in older workers who experience involuntary job loss: results from the health and retirement survey. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 61*(4), S221-S228.

Gallo, W. T., Bradley, E. H., Falba, T. A., Dubin, J. A., Cramer, L. D., Bogardus, S. T., & Kasl, S. V. (2004). Involuntary job loss as a risk factor for subsequent myocardial infarction and stroke: findings from the Health and Retirement Survey. *American Journal of Industrial Medicine, 45*(5), 408-416.

Gallo, W. T., Bradley, E. H., Siegel, M., & Kasl, S. V. (2000). Health effects of involuntary job loss among older workers findings from the health and retirement survey. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 55*(3), S131-S140.

Gallo, W. T., Bradley, E. H., Siegel, M., & Kasl, S. V. (2001). The Impact of Involuntary Job Loss on Subsequent Alcohol Consumption by Older Workers Findings From the Health and Retirement Survey. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 56*(1), S3-S9.

Guo, G., Liu, H., Wang, L., Shen, H., & Hu, W. (2015). The Genome-Wide Influence on Human BMI Depends on Physical Activity, Life Course, and Historical Period. *Demography, 52*(5), 1651-1670.

Hammarström, A., & Janlert, U. (1994). Unemployment and change of tobacco habits: a study of young people from 16 to 21 years of age. *Addiction, 89*(12), 1691-1696.

Harismendy, O., Bansal, V., Bhatia, G., Nakano, M., Scott, M., Wang, X., . . . Murray, S. S. (2010). Population sequencing of two endocannabinoid metabolic genes identifies rare and

common regulatory variants associated with extreme obesity and metabolite level. *Genome Biol, 11*(11), R118.

Hayes, J., & Nutman, P. (1981). *Understanding the unemployed: The Psychological Effects of Unemployment* (Vol. 223): Routledge.

Heckman, J. J., Ichimura, H., & Todd, P. E. (1997). Matching as an econometric evaluation estimator: Evidence from evaluating a job training programme. *The Review of Economic Studies, 64*(4), 605-654.

Jacobson, L. S., LaLonde, R. Z., & Sullivan, D. G. (1993). Earnings losses of displaced workers. *The American Economic Review*, 685-709.

Johnson, R. W. (2012). *Older Workers, Retirement, and the Great Recession.* New York: Russell Sage Foundation.

Johnson, R. W, & Butrica, B. A. (2012). Age Disparities in Unemployment and Reemployment During the Great recession and Recovery. Unemployment and Recovery Project Brief, 3.

Johnson, R. W, & Park, J. S. (2011). Can Unemployed Older Workers Find Work? *Urban Institute: Older Americans' Economic Security, 25*.

Jusot, F., Khlat, M., Rochereau, T., & Serme, C. (2008). Job loss from poor health, smoking and obesity: a national prospective survey in France. *Journal of Epidemiology and Community Health, 62*(4), 332-337.

Kilpeläinen, T. O., Qi, L., Brage, S., Sharp, S. J., Sonestedt, E., Demerath, E., . . . Sandholt, C. H. (2011). Physical activity attenuates the influence of FTO variants on obesity risk: a meta-analysis of 218,166 adults and 19,268 children. *PLoS Med, 8*(11), e1001116.

Kivimäki, M., Head, J., Ferrie, J. E., Shipley, M. J., Brunner, E., Vahtera, J., & Marmot, M. G. (2006). Work stress, weight gain and weight loss: evidence for bidirectional effects of job strain on body mass index in the Whitehall II study. *International Journal of Obesity, 30*(6), 982-987.

Korkeila, M., Kaprio, J., Rissanen, A., Koskenvuo, M., & Sörensen, T. A. (1998). Predictors of major weight gain in adult Finns: stress, life satisfaction and personality traits. *International Journal of Obesity, 22*(10), 949-957.

Kyle, U. G., Schutz, Y., Dupertuis, Y. M., & Pichard, C. (2003). Body composition interpretation: contributions of the fat-free mass index and the body fat mass index. *Nutrition, 19*(7), 597-604.

Laitinen, J., Ek, E., & Sovio, U. (2002). Stress-related eating and drinking behavior and body mass index and predictors of this behavior. *Preventive Medicine, 34*(1), 29-39.

Lewis, C. E., McTigue, K. M., Burke, L. E., Poirier, P., Eckel, R. H., Howard, B. V., . . . Pi-Sunyer, F. X.. (2009). Mortality, health outcomes, and body mass index in the overweight range a science advisory from the American Heart Association. *Circulation, 119*(25), 3263-3271.

Liu, H., & Guo, G.. (2015). Lifetime Socioeconomic Status, Historical Context, and Genetic Inheritance in Shaping Body Mass in Middle and Late Adulthood. *American Sociological Review, 80*(4), 705-737.

Locke, A. E., Kahali, B., Berndt, S. I., Justice, A. E., Pers, T. H., Day, F. R., . . . Yang, J. (2015). Genetic studies of body mass index yield new insights for obesity biology. *Nature, 518*(7538), 197-206.

Loos, R., & Yeo, G. (2014). The bigger picture of FTO [mdash] the first GWAS-identified obesity gene. *Nature Reviews Endocrinology, 10*(1), 51-61.

Marcus, J. (2014). Does job loss make you smoke and gain weight? *Economica, 81*(324), 626-648.

Meyer, P. B., & Osborne, A. M. (2005). *Proposed Category System for 1960-2000 Census Occupations*: US Department of Labor, Bureau of Labor Statistics, Office of Productivity and Technology.

Munnell, A. H., & Sass, S. A. (2009). *Working Longer: The solution to the Retirement Income Challenge*: Brookings Institution Press.

Neuman, K. (2008). Quit your job and get healthier? The effect of retirement on health. *Journal of Labor Research, 29*(2), 177-201.

Ogden, C. L., Carroll, M. D., Kit, B. K., & Flegal, K. M. (2012). Prevalence of obesity in the United States, 2009-2010. *NCHS Data Brief*(82), 1-8.

Price, A. L., Patterson, N. J., Plenge, R. M., Weinblatt, M. E., Shadick, N. A., & Reich, D. (2006). Principal components analysis corrects for stratification in genome-wide association studies. *Nature Genetics, 38*(8), 904-909.

Purcell, S., & Chang, C. (2015). PLINK 1.9. *URL https://www. cog-genomics. org/plink2*.

Pütter, C., Pechlivanis, S., Nöthen, M. M., Jöckel, K. H., Wichmann, H. E., & Scherag, A. (2011). Missing heritability in the tails of quantitative traits? A simulation study on the impact of slightly altered true genetic models. *Human Heredity, 72*(3), 173-181.

Roelfs, D. J., Shor, E., Davidson, K. W., & Schwartz, J. E. (2011). Losing life and livelihood: a systematic review and meta-analysis of unemployment and all-cause mortality. *Social Science & Medicine, 72*(6), 840-854.

Rosenbaum, P. R., & Rubin, D. B. (1985). Constructing a control group using multivariate matched sampling methods that incorporate the propensity score. *The American Statistician, 39*(1), 33-38.

Rubin, D. B., & Stuart, E. A. (2006). Affinely invariant matching methods with discriminant mixtures of proportional ellipsoidally symmetric distributions. *The Annals of Statistics*, 1814-1826.

Rubin, D. B., & Thomas, N. (1996). Matching using estimated propensity scores: relating theory to practice. *Biometrics*, 249-264.

Salm, M. (2009). Does job loss cause ill health? *Health Economics, 18*(9), 1075-1089.

Schmitz, L., & Conley, D. (2015). Modeling Gene−Environment Interactions With Quasi−Natural Experiments. *Journal of Personality*. Advance online publication. DOI: 10.1111/jopy.12227.

Shungin, D., Winkler, T. W., Croteau-Chonka, D. C., Ferreira, T., Locke, A. E., Mägi, R., . . . Justice, A. E. (2015). New genetic loci link adipose and insulin biology to body fat distribution. *Nature, 518*(7538), 187-196.

Smith, J. A., & Todd, P. E. (2005). Does matching overcome LaLonde's critique of nonexperimental estimators? *Journal of Econometrics, 125*(1), 305-353.

Speliotes, E. K., Willer, C. J., Berndt, S. I., Monda, K. L., Thorleifsson, G. J., Anne U., . . . Mägi, R. (2010). Association analyses of 249,796 individuals reveal 18 new loci associated with body mass index. *Nature Genetics, 42*(11), 937-948.

Strully, K. W. (2009). Job loss and health in the US labor market. *Demography, 46*(2), 221-246.

Stuart, E. A. (2010). Matching methods for causal inference: A review and a look forward. *Statistical science: a review journal of the Institute of Mathematical Statistics, 25*(1), 1.

Sudano J. J., & Baker, D. W. (2003). Intermittent lack of health insurance coverage and use of preventive services. *American Journal of Public Health, 93*(1), 130-137.

Taylor, A. E., Ebrahim, S., Ben-Shlomo, Y., Martin, R. M., Whincup, P. H., Yarnell, J. W., . . . Lawlor, D. A. (2010). Comparison of the associations of body mass index and measures of central adiposity and fat mass with coronary heart disease, diabetes, and all-cause mortality: a study using data from 4 UK cohorts. *The American Journal of Clinical Nutrition, 91*(3), 547-556.

Torres, S. J., & Nowson, C. A. (2007). Relationship between stress, eating behavior, and obesity. *Nutrition, 23*(11), 887-894.

Tu, H. T., & Liebhaber, A. B. (2009). Rough Passage: Affordable Health Coverage for Near-Elderly Americans: Center for Studying Health System Change.

Turner, J. B. (1995). Economic context and the health effects of unemployment. *Journal of Health and Social Behavior*, 213-229.

Van Strien, T., Rookus, M. A., Bergers, G. P., Frijters, J. E., & Defares, P. B. (1985). Life events, emotional eating and change in body mass index. *International Journal of Obesity, 10*(1), 29-35.

Winkler, T. W., Justice, A. E., Graff, M., Barata, L., Feitosa, M. F., Chu, S., . . . Kilpeläinen, T. O. (2015). The influence of age and sex on genetic associations with adult body size and shape: a large-scale genome-wide interaction study. *PLoS Genet, 11*(10), e1005378.