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Socioeconomic Modifiers of Genetic and Environmental Influences on Body Mass Index in Adult Twins

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Objective: Individual measures of socioeconomic status (SES) suppress genetic variance in body mass index (BMI). Our objective was to examine the influence of both individual-level (i.e., educational attainment, household income) and macrolevel (i.e., neighborhood socioeconomic advantage) SES indicators on genetic contributions to BMI. ***Method:*** The study used education level data from 4,162 monozygotic (MZ) and 1,900 dizygotic (DZ) same-sex twin pairs (64% female), income level data from 3,498 MZ and 1,534 DZ pairs (65% female), and neighborhood-level socioeconomic deprivation data from 2,327 MZ and 948 DZ pairs (65% female). Covariates included age ($M = 40.4 \pm 17.5$ years), sex, and ethnicity. The cotwin control model was used to evaluate the mechanisms through which SES influences BMI (e.g., through genetic vs. environmental pathways), and a gene-by-environment interaction model was used to test whether residual variance in BMI, after controlling for the main effects of SES, was moderated by socioeconomic measures. ***Results:*** SES significantly predicted BMI. The association was noncausal, however, and instead was driven primarily through a common underlying genetic background that tended to grow less influential as SES increased. After controlling for the main effect of SES, both genetic and nonshared environmental variance decreased with increasing SES. ***Conclusions:*** The impact of individual and macrolevel SES on BMI extends beyond its main effects. The influence of genes on BMI is moderated by individual and macrolevel measures of SES, such that when SES is higher, genetic factors become less influential.

Keywords: body mass index, behavior genetics, socioeconomic status, twin study

Obesity is a serious medical condition that currently affects more than 25% of children and 35% of adults in the United States (Ogden, Carroll, Kit, & Flegal, 2013, 2014). The rate of obesity has been steadily rising—prevalence rates have increased approximately 8% per decade since 1980 (Flegal, Carroll, Kit, & Ogden, 2012). In 2008, the Centers for Disease Control and Prevention (CDC) estimated that medical costs associated with obesity were around $147 billion (Trogdon, Finkelstein, Feagan, & Cohen, 2012). Given its association with preventable mortality, substantial costs to society, and associated stigma, obesity poses a major public health challenge whose contributing factors are important to investigate and understand.

Classical twin studies show that two thirds of the variability in BMI is attributable to genetic factors (Elks et al., 2012; Franz et al., 2007; Ravussin & Bogardus, 2000). Many genes and gene variants (e.g., FTO [fat mass and obesity associated gene], MC4R [melanocortin 4 receptor]) have been associated with variance in body mass index (BMI) or body weight (Loos, 2012). Regardless of the approach used, the proportion of variance in BMI accounted for by known variants is small. For example, FTO has the strongest effect on obesity susceptibility, and each FTO risk allele increases BMI by 0.39kg m^2 (Loos, 2012). Genes do not fully account for the variation in BMI within families and across generations; indeed, studies show that while the rearing environment has little influence on BMI in adulthood, the nonshared environment has considerable influence (Franz et al., 2007; Silventoinen & Kaprio, 2009).

One of the most prevalent factors associated with obesity is socioeconomic status (SES). In a comprehensive review, obesity was found to be six times more prevalent at a lower SES compared with higher SES (McLaren, 2007). However, most studies examining the association between SES and BMI have focused on individual-level SES indicators such as education and income (Della Bella & Lucchini, 2014; Johnson & Krueger, 2005a; Johnson, Kyvik, Skytthe, Deary, & Sørensen, 2011).

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Following the sociological argument that "place" has important effects on health because of its role as constitutor and container of social and physical resources, psychologists have begun examining the health impact of contextual factors in addition to individual ones (Cummins, Curtis, Diez-Roux, & Macintyre, 2007). There are multiple mechanisms through which neighborhoods might influence health outcomes, from availability of resources, health services, and infrastructure, to cultural attitudes. Neighborhood deprivation, consistently associated with mortality (Pearson, Apparicio, & Riva, 2013; Ross, Oliver, & Villeneuve, 2013), is one such factor. Research has consistently found effects of neighborhood deprivation on BMI (El-Sayed, Scarborough, & Galea, 2012; Matheson, Moineddin, & Glazier, 2008; Powell-Wiley et al., 2015), and neighborhood SES has also been associated with obesity risk (Black & Macinko, 2008; El-Sayed et al., 2012; Glass, Rasmussen, & Schwartz, 2006). Macrolevel social factors such as neighborhood-level socioeconomic deprivation are becoming increasingly relevant to research investigating environmental determinants of health.

Although it is informative to investigate the main effects of SES on the BMI phenotype, it is equally important to examine the nonadditive effects of SES on the BMI genotype. Other quantitative genetic studies have investigated interactions between SES and genetic variance in BMI. Genetic variance in BMI decreased with increasing income in a sample of U.S. twins (Johnson & Krueger, 2005b), and with increasing educational attainment in a Danish twin sample (Johnson et al., 2011). The current study intends to replicate and expand previous findings that SES suppresses genetic variance in BMI using a sample of adult U.S. twins.

Our first aim is to examine the influence of individual-level SES indicators (i.e., educational attainment and household income) on genetic contributions to BMI. In line with previous research, we predict that SES and BMI share an underlying genotype which will be more significant in poorer environments. As an important contribution to current research, we also explore whether a macrolevel SES indicator (i.e., census-based area deprivation index, indicative of neighborhood socioeconomic advantage; Singh, 2003) demonstrates congruent effects on genetic variance in BMI. We predict that neighborhood socioeconomic advantage will have the same relationship with BMI as individual level SES factors. It is very important to understand through which mechanisms SES and BMI are correlated, and using neighborhood socioeconomic advantage in addition to individual-level SES indicators provides a clearer picture of the role of environmental context in the risk for and development of obesity in the U.S.

Method

Sample

The University of Washington Twin Registry (UWTR) is a cross-sectional community-based sample of adult twins reared together; construction methods are described in detail elsewhere (Afari et al., 2006; Strachan et al., 2013). Twins completed surveys that included items on sociodemographics, health, and lifestyle behaviors. Twins were classified as identical (monozygotic; MZ) or fraternal (dizygotic; DZ) using standard questions about childhood similarity, which determine zygosity with greater than 90% accuracy when compared with DNA-based methods (Eisen, Neuman, Goldberg, Rice, & True, 1989; Spitz et al., 1996; Torgersen, 1979). Written informed consent was provided as approved by the university's institutional review board. Complete education level information was available for 4,162 monozygotic (MZ) and 1,900 dizygotic (DZ) same-sex twin pairs (64% female); income level data for 3,498 MZ and 1,534 DZ (65% female) pairs; and neighborhood-level socioeconomic deprivation for 2,327 MZ and 948 DZ pairs (65% female). Overall, the sample was young (40.4 ± 17.5 years, *range* = 18 to 78, 1st quartile = 25.1, 3rd quartile = 54.8) and predominantly white (87% Caucasian, 2% African American, 3% Asian American, 1% Native American, 7% other).

Measures

Body mass index (BMI). Self-reported height and weight was used to calculate BMI (kg/m^2) using the CDC-based formula for BMI ([weight in lbs/height in $inches^2$]*703); 2.7% of the sample were classified as underweight (BMI <18.5), 49.0% normal weight (BMI ranging from 18.5–24.9), 29.7% overweight (BMI ranging from 25–29.9), and 18.6% obese (BMI >30). Among 200 twin pairs from the UWTR, we found that self-reported BMI was highly correlated, $r = .98$, $p < .01$ with directly measured BMI, indicating a high degree of construct validity in our sample.

Socioeconomic status (SES). We used three indices of socioeconomic status to moderate variance in BMI: household income, education level, and neighborhood-level socioeconomic deprivation.

Education level. Respondents indicated their level of education: 1 = *never attended school/only Kindergarten only*; 2 = *Grades 1–8*; 3 = *Grades 9–11*; 4 = *Grade 12/High School diploma/GED*; 5 = *some college*; 6 = *Associate's Degree/Vocational or Trade School Degree*; 7 = *bachelor's degree*; 8 = *graduate or professional degree*. The sample was highly educated; the median education level attained was an Associate's degree or vocational/trade school degree (1st quartile = some college, 3rd quartile = graduate or professional degree), and 42% of the sample earned at least a bachelor's degree.

Household income. Twins indicated their total household income using the following scale: 1 = *less than $20,000*; 2 = *$20,000 to $29,999*; 3 = *$30,000 to $39,999*; 4 = *$40,000 to $49,999*; 5 = *$50,000 to $59,999*; 6 = *$60,000 to $69,999*; 7 = *$70,000 to $79,999*; 8 = *more than $80,000*. The median household income was between $60K–70K (1st quartile = $30–40K, 3rd quartile ≥ $80K).

Neighborhood-level socioeconomic advantage. An area deprivation score was created using census tract data for twins' residential area (Singh, 2003). Seventeen total neighborhood deprivation factors contribute to area deprivation: percentage of the population with fewer than 9 years and with 12 or more years of education, median family income, income disparity, occupational composition, unemployment rate, family poverty rate, percentage of the population below 150% of the poverty rate, single-parent household rate, home ownership rate, median home value, median gross rent, median monthly mortgage, and household crowding. These indices are standardized then weighted to create an area deprivation score, where lower scores indicate greater socioeconomic deprivation (i.e., lower SES). The area deprivation score is typically scaled such that greater scores indicate lower area socio-

economic status. To keep the results consistent across analyses, we rescaled the area deprivation score to make higher scores correspond to higher SES.

Statistical Analyses

All analyses were conducted using latent variable path analysis using the computer program Mplus v. 7.0 (Muthén & Muthén, 2013) and maximum likelihood estimation. Analyses controlled for linear effects of age, gender, and ethnicity (white/nonwhite). We used likelihood ratio tests to compare nested models.

Univariate biometric decomposition. Although it was not the primary goal of our analysis, we began by using the classical twin model to decompose the variance of body mass index and the three socioeconomic status indicators into three components: additive genetic (A) variance, shared environmental (C) variance, and nonshared environmental (E) variance. The A variance components, which represent the additive effect of an individual's genes, correlate $r = 1.0$ between MZ twins (who share 100% of their genetic sequence) and $r = .5$ between DZ twins (who share on average 50% of their segregating genes). The C variance components correlate at 1.0 regardless of degree of genetic relatedness, because it represents environmental experiences that make members of the same family more alike. The E variance components, which represent environmental experiences unique to the individual, do not correlate between twins.

Causal pathways versus gene-environment correlation. Examining the association between socioeconomic status and body mass index within pairs of MZ and DZ twins raised in the same family provides the closest approximation of the causal effect of SES on BMI short of random assignment to socioeconomic deprivation. Assessing this relationship within twin pairs allows us to control for the effects of many measured and unmeasured confounds that vary between families, such as underlying genetic or family level environmental backgrounds that SES and BMI may share.

The bivariate twin model is essentially a regression model in which the outcome (BMI) is regressed on the A, C, and E terms of the predictor. The outcome also comprises residual variation not explained by the predictor that can also be partitioned into A, C, and E components. A causal relationship between SES and BMI is supported when the association is observed both between twin pairs (pairs who earn less, are less educated, or live in deprived neighborhoods on average have higher BMIs) and within twin pairs (the pair member who earns less, is less highly educated, or lives in the more deprived neighborhood has a higher BMI than his or her cotwin earning more, achieving a higher level of education, or living in the less deprived neighborhood). The within-pair association is the most valid measure of the causal effect of SES on BMI. Of course, if the SES–BMI association is not observed within families, a noncausal process that operates through genetic pathways can be inferred. This process is referred to as *gene–environment correlation* (*r*GE). Shared environmental factors (e.g., socioeconomic deprivation during childhood) may also be inducing this correlation. We chose to use *r*GE in our example, however, because BMI has no shared environmental variance.

The twin design does not control for *all* possible confounds of a causal relationship, but it does control for all those that are shared by pairs of twins who were raised together, measured or unmeasured. Because of the quasi-experimental nature of the cotwin control design, we assert that the twin design allows us to establish a *quasi-causal effect* of SES on BMI (Turkheimer & Harden, 2013).

Gene-by-environment (G × E) interaction. We are also able to test for moderation of the ACE variance components of BMI by SES, a form of G × E interaction, by extending the model described above (Purcell, 2002). The three regression parameters relating the socioeconomic indicator to BMI and the three residual variances of BMI can all be modified by the SES, as illustrated in Figure 1. For each of the modified paths, SES is the moderating variable; the b_0 terms are the values of the ACE variances where

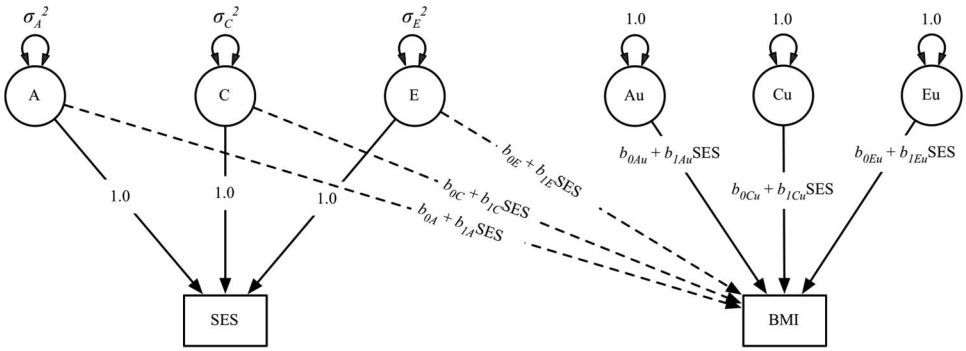

Figure 1. Path diagram of the fully saturated model fit to the data (Model 3; only one twin shown for clarity). Successive models were fit by fixing parameters to zero and conducting likelihood ratio tests whether adding parameters resulted in a significant improvement in model fit. The A, C, and E latent variables (represented with circles) are the additive genetic, shared environmental and nonshared environmental variance components of SES. The Au and Eu latent variables represent residual additive genetic and nonshared environmental variance in BMI. In this model, the main effect of SES on BMI (captured in the dotted single-headed paths from the A and E components of SES to BMI) is permitted to vary with level of SES. Similarly, the variance in BMI that remains after controlling for the main effect of SES (double-headed paths from Au, Cu, and Eu to BMI) also varies as a function of SES.

SES = 0; and the b_1 terms represent the rate of increase or decrease in a given variance component as a function of SES.

For individual-level moderators that can differ between twins from the same family (such as the socioeconomic indicators used in this study), the correlation between genes and environment (*r*GE) must be accounted for when testing for G × E effects to reduce the inflated false positive rate that results from failure to do so (van der Sluis, Posthuma, & Dolan, 2012). To account for *r*GE, the regressions of BMI on the ACE components of SES are also allowed to vary as a function of SES (i.e., the effect that SES has on BMI can depend on level of SES)—this procedure accounts for changes in total ACE variances in BMI that are instead attributable to the main effects of SES on BMI being nonstatic across levels of SES. We present a path diagram of the fully saturated model fit to the data in Figure 1.

Results

Descriptive Statistics & Univariate Biometric Analyses

Descriptive statistics are presented in Table 1, and twin correlations and standardized biometric variance components for BMI and the SES indicators are presented in Table 2. BMI was influenced primarily by additive genetic factors (75%), showed no shared environmental influences (0%), and modest influence from the nonshared environment (25%). BMI is by its nature not normally distributed, so follow-up analyses were conducted using the natural logarithm of BMI. These yielded results consistent with those presented in this report, suggesting that our results are not an artifact of right skew in the distribution of BMI. Education was moderately influenced by genetic (33%) and nonshared environmental (24%) factors, with a larger contribution from the shared environment (43%). Household income showed moderate contributions from A (28%) and C (25%) with a large nonshared component (46%). Neighborhood-level socioeconomic deprivation showed the least influence from genetic factors (17%), showed moderate shared environmental influence (35%), and large nonshared environmental influence (47%).

Table 1
Descriptive Statistics and Frequencies for Body Mass Index and Indicators of Socioeconomic Status

Body mass index	Statistic
M	25.840
SD	5.584
Range	13.730–71.913
1st quartile	21.946
3rd quartile	28.339
Education level	**Frequency**
8th grade or less	.5%
Grades 9 through 11	2.9%
HS graduate or GED	16.6%
Some college	26.3%
Associate's degree	10.7%
Technical or vocational school	1.1%
Bachelor's degree	25.1%
Graduate or professional degree	16.9%
Household income	**Frequency**
< $20,000	14.5%
$20,000 to $29,999	8.6%
$30,000 to $39,999	9.1%
$40,000 to $49,999	8.6%
$50,000 to $59,999	8.2%
$60,000 to $69,999	7.6%
$70,000 to $79,999	7.0%
≥ $80,000	36.3%
Area deprivation	**Statistic**
M	−.002
SD	.878
Range	−7.513 to 2.307
1st quartile	−.491
3rd quartile	.573

Note. As noted in the section where BMI is described, 2.7% of the sample classified as underweight (BMI < 18.5), 49.0% normal weight (BMI ranging from 18.5–24.9), 29.7% overweight (BMI ranging from 25–29.9), and 18.6% obese (BMI > 30).

Causal Pathways Versus rGE

The phenotypic regression of BMI on education level showed a significant negative relationship ($b = -0.171$, $p = .001$). That is, individuals with a graduate or professional degree had a BMI of 0.780 kg/m^2 less than individuals with just a high school degree, and 0.156 kg/m^2 less than college graduates. Household income had a similar negative effect on BMI ($b = -0.065$, $p = .001$), as did neighborhood-level socioeconomic advantage ($b = -0.293$, $p < .001$). We then tested whether these results were consistent with a causal hypothesis by fitting the bivariate quasi-causal model. The results are given in the first column of Table 3, labeled *Model 1: Quasi-Causal*. The quasi-causal pathway for the regression of BMI on education level was reduced to zero ($b_{0E} = 0.062$, $p = .402$), and a significant common genetic background to education level and BMI was present ($b_{0A} = -0.875$, $p < .001$), indicating that the genes that contribute to educational attainment are also the same genes influencing BMI. The association between income and BMI was similarly noncausal ($b_{0E} = 0.021$, $p = .363$) and also showed evidence of significant gene-environment correlation ($b_{0A} = -0.578$, $p < .001$). Neighborhood-level socioeconomic advantage showed the same noncausal, genetically induced association with BMI ($b_{0E} = 0.036$, $p = .663$; $b_{0A} = -3.641$, $p = .001$).

These phenotypic and within-family effects are illustrated in Figure 2. In Figure 2a, we show pair differences in BMI as a function of pair differences in educational attainment within randomly paired individuals (phenotypic difference; dotted line) and within MZ twin pairs (solid line). Comparison of these lines suggests that differences in education level do not predict differences in BMI within families, only between them. If the protective effect of educational attainment on BMI was causal, the slopes of these lines would closely approximate one another. In Figure 2b, we identified twin pairs concordant for lower household income (annual salary ≤ the median sample income of $70K; light gray), twin pairs concordant for higher household income (annual salary > $70K; dark gray), and twin pairs discordant for household income (i.e., one pair earns more than the median income of $70K and one earns less). Overall, there is a main effect of household

Table 2
Twin Intraclass Correlations and Standardized ACE Components for Body Mass Index and Indicators of Socioeconomic Status

Item	Body mass index	Education level	Household income	Area deprivation
Twin correlations				
MZ	.754 (.006)	.749 (.009)	.537 (.011)	.525 (.015)
DZ	.378 (.018)	.579 (.020)	.395 (.020)	.438 (.024)
ACE estimates				
h2	.752 (.031)	.340 (.040)	.283 (.044)	.173 (.053)
c2	.002 (.030)	.409 (.039)	.254 (.040)	.352 (.048)
e2	.246 (.006)	.251 (.009)	.463 (.011)	.475 (.015)

income on BMI such that higher income is associated with lower BMIs on average (this is evident comparing the height of two outermost bars). Examining the inner bars, however, demonstrates that the BMIs of discordant MZ twins are essentially identical regardless of their income level, again consistent with genetic selection (or *r*GE). As expected, the effect of household income on BMI is less attenuated in DZ twins who share on average only 50% of their genes. Figure 2c demonstrates the main effects of area deprivation on BMI, and consistent with our observations for education and income, no effects exist within members of MZ twins. Taken together, it appears that although there is an overall protective effect of individual-level and macrolevel measures of SES on BMI, this effect is driven primarily by genetic influences that are common to both SES and BMI.

G × E Interaction

For each predictor, we next fit a model which allowed for differences in the A and E variance components of BMI as a function of SES (solid single-headed paths from the latent variables Au, Cu, and Eu to BMI in Figure 1), controlling for age and gender. We did not include C in the model because there was no evidence of shared environmental influences on BMI in our univariate model. For each outcome, adding these additional parameters significantly improved model fit ($p < .001$ for each predictor; *Model 2* in Table 3), suggesting that variance in BMI depends on level of SES. Allowing the main effects of SES on BMI (the dotted paths from the A, C, and E components of SES to BMI in Figure 1) to be modified by different levels of SES—which tests whether heteroscedasticity of variance in BMI with respect to level of SES explains any change in the total variance of BMI—improved model fit for education level ($p < .001$) and income ($p < .001$), but not area deprivation ($p = .493$; *Model 3* in Table 3).

The best-fitting models (denoted with a † in Table 3) suggest that phenotypic variance in BMI decreases with increasing socioeconomic status. Residual genetic variance decreased by 0.075 standard deviation units ($p < .001$) for each additional level of education achieved; residual nonshared environmental variance decreased by 0.031 standard deviation units ($p < .001$). Residual A variance in BMI decreases by 0.032 standard deviation units for each additional $10,000 earned ($p < .001$); residual E variance decreased by 0.035 standard deviation units ($p < .001$). As the level of neighborhood-level socioeconomic advantage increased, residual A variance decreased (0.057 standard deviation units for every additional unit of area deprivation; $p = .003$) as did residual E variance (0.129 standard deviation units for each additional unit of area deprivation; $p < .001$).

These model results are illustrated in the stacked variance plots in Figure 3. The black regions represent residual additive genetic variance in BMI decreasing as a function of increasing SES, and the gray regions represent the same relation for nonshared environmental variance. Also evident is that total residual variance in BMI decreases as a function of socioeconomic advantage. This heteroscedasticity is further illustrated in Figure 4, which shows box plots overlaid with violin plots (which show the probability density of the data) of BMI by quartile of SES. Several characteristics of these plots are worth mentioning. First, the median BMI tends to decrease with increasing SES, illustrating the main effect of SES on BMI at the phenotypic level. Second, the distribution of BMI becomes less platykurtic at higher levels of socioeconomic advantage, demonstrating that overall variance in BMI is more constrained at higher SES levels. This effect appears to be driven primarily by the presence of fewer individuals in the upper tail of the BMI distribution at higher SES levels. Third, the proportion of the sample falling in the overweight or obese range is higher at lower SES levels, such that more than 50% of the sample tends to be overweight or obese at SES levels that are below the population median.

Discussion

The current study uses a large twin sample from the U.S. to examine the effects of individual- and macrolevel socioeconomic status on levels of BMI and variance in BMI. Our results show that there is a phenotypic association between these various socioeconomic indicators (education, income, and neighborhood deprivation) and BMI. In other words, we found that in the general population (i.e., unrelated individuals), lower education or income is associated with higher BMI, which is consistent with prior research as well (Arendt, 2005; Neuman, Kawachi, Gortmaker, & Subramanian, 2013). When examining this effect within families (i.e., twin pairs), however, it becomes nonsignificant, suggesting that there is not a phenotypic causal relation between SES measures and BMI, but rather that these phenotypes share an underlying genotype. That is, the genes contributing to an individual attaining higher SES also predict lower BMI (an example of gene-environment correlation). This finding, too, is in line with past research (Johnson & Krueger, 2005a; Johnson et al., 2011; Osler, McGue, Lund, & Christensen, 2008; Webbink, Martin, & Visscher, 2010).

We also detected gene-by-environment interaction in the relationship between socioeconomic indicators and BMI. In G × E analyses in which the measure of the environment (i.e., the socioeconomic indicators) vary at the individual as opposed to the pair level, there are two components to the interaction, one in the regressions of the outcome on the environmental indices, and the other in the residual variance of the outcome once the effects of the environmental indicator have been accounted for. Regarding the former, we found that the genetic associations between education and income, but not area deprivation, and BMI were weaker in environments with greater SES. Put another way, it appears that genetic variance related to both SES and BMI is more strongly expressed in poorer environments.

Table 3
Parameter Estimates and Model Fit Statistics for G × SES Models

Variable	Parameter	Model 1: Quasi-causal	Model 2: Moderation of residual variance	Model 3: Moderation of main effects†
Education	Main effect of education on BMI			
	b_{0A}	**−.758 (.145)**	**−.783 (.144)**	**1.579 (.510)**
	b_{1A}	—	—	**−.364 (.079)**
	b_{0E}	.045 (.058)	.071 (.058)	−.658 (.332)
	b_{1E}	—	—	.111 (.049)
	Effect of education on residual ACE components of BMI			
	b_{0Au}	**2.220 (.043)**	**2.502 (.065)**	**2.588 (.069)**
	b_{1Au}	—	**−.053 (.008)**	**−.067 (.009)**
	b_{0Eu}	**1.293 (.034)**	**1.460 (.057)**	**1.451 (.056)**
	b_{1Eu}	—	**−.025 (.007)**	**−.025 (.007)**
	Model fit			
	−2LL	78981.985	78897.412	78880.980
	Δ−2LL (Δ*df*)	—	84.573 (+2)	16.432 (+2)
	p	—	<.001	<.001
Household income	Main effect of income on BMI			
	b_{0A}	**−.578 (.119)**	**−.623 (.126)**	−.053 (.200)
	b_{1A}	—	—	**−.125 (.041)**
	b_{0E}	.021 (.023)	.013 (.023)	.023 (.083)
	b_{1E}	—	—	−.006 (.016)
	Effect of income on residual ACE components of BMI			
	b_{0Au}	**2.193 (.035)**	**2.286 (.039)**	**2.310 (.040)**
	b_{1Au}	—	**−.026 (.004)**	**−.032 (.005)**
	b_{0Eu}	**1.408 (.026)**	**1.546 (.032)**	**1.537 (.032)**
	b_{1Eu}	—	**−.035 (.004)**	**−.035 (.004)**
	Model fit			
	−2LL	140012.024	139834.908	139819.667
	Δ−2LL (Δ*df*)	—	177.116 (+2)	15.241 (+2)
	p	—	<.001	<.001
Area deprivation	Main effect of area deprivation on BMI			
	b_{0A}	**−3.641 (1.140)**	**−3.875 (1.259)**	**−3.919 (1.268)**
	b_{1A}	—	—	−.417 (.384)
	b_{0E}	.036 (.084)	.052 (.087)	.051 (.089)
	b_{1E}	—	—	.094 (.113)
	Effect of area deprivation on residual ACE components of BMI			
	b_{0Au}	**2.142 (.046)**	**2.128 (.048)**	**2.124 (.049)**
	b_{1Au}	—	**−.047 (.016)**	**−.057 (.019)**
	b_{0Eu}	**1.347 (.030)**	**1.345 (.030)**	**1.344 (.030)**
	b_{1Eu}	—	**−.129 (.015)**	**−.129 (.015)**
	Model fit			
	−2LL	74144.051	74024.367	74023.147
	Δ−2LL (Δ*df*)	—	119.684 (+2)	1.220 (+2)
	p	—	<.001	.543

Note. Standard errors are presented within parentheses. **Bold** face denotes statistically significant ($p < .05$) parameter estimates. −2LL represents the −2 × log-likelihood of the model.
† Best-fitting model.

With regard to the variance in BMI remaining after controlling for the main effects of SES, we observed that both A and E variance decreased with increasing SES. It appears, therefore, that at high levels of SES the phenotypic variance of BMI is attenuated: the genetic and nonshared environmental variances are reduced to a roughly equal degree, so the percentages of the residual variance attributed to A and E do not change substantially. Indeed, testing a proportional model (Kremen et al., 2005) of this interaction indicated that the changes in A and E variance as a function of SES could be constrained to be equal, suggesting that these changes are proportional and that the primary effect of SES is on total variance in BMI.

Our results support prior research (Johnson & Krueger, 2005a; Johnson et al., 2011) showing that genetic variance in BMI decreases as a function of increasing SES, but we note important contributions that our report makes to the existing literature. Whereas previous reports have all been on the level of the individual, we extend these findings to include the area deprivation, which, as a measure of neighborhood-level socioeconomic deprivation, is a macrolevel indicator of SES. Neighborhoods are not simply geographical locations in which one resides, they also provide the context and limitations within which one develops, forms relationships, and makes life choices. Neighborhoods determine, for example, how many parks and outdoor spaces are avail-

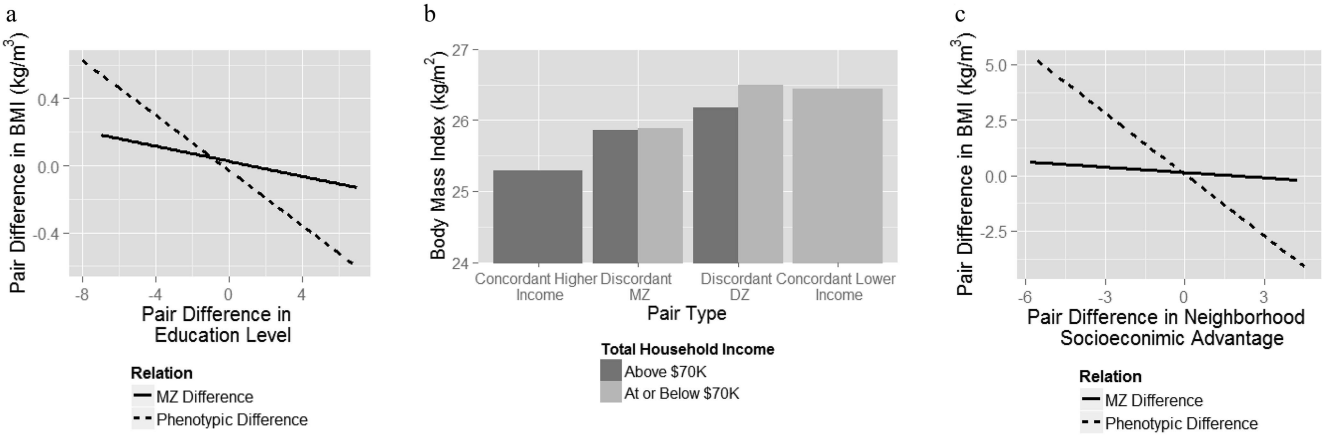

Figure 2. Illustrative analyses of the main effects of education level (a), household income (b), and area deprivation (c) on body mass index. Figure 2a and 2c show pair differences in BMI as a function of pair differences in SES. The phenotypic effect of SES on BMI, equivalent to a population regression, is represented by the dashed line. The solid line represents the same relation within pairs of MZ twins, and shows the nonshared environmental effect of SES on BMI. Figure 2b shows mean BMI as a function of family income in various pair types. Comparison of the outermost bars (mean BMI of twins concordant for higher income vs. that of twins concordant for lower income) shows the phenotypic effect of income on BMI. The inner bars show this same comparison within MZ and DZ pairs discordant for household income level.

able in proximity to the residence and how many healthy food stores are within walking distance (Leslie, Cerin, & Kremer, 2010; Morland, Wing, Diez Roux, & Poole, 2002), both of which could impact BMI above and beyond individual-level factors.

The impact of individual and neighborhood SES on BMI variability has practical implications for the screening and treatment of obesity-related diseases. For example, the American Diabetes Association recommends screening overweight children (BMI >85th percentile) based on a number of preestablished criteria and sociodemographics, such as age, sex, medical family history, race or ethnicity-specific risk, and signs of insulin resistance (Barness, Opitz, & Gilbert-Barness, 2007). Our results contribute to a growing body of research that implicates low SES in increasing the genetic risk for high BMI, and support current clinical care measures requiring that individuals who are found to be at risk based on family history go through an assessment of living environment as a means of contributing to early detection and alleviation of obesity. In such an assessment, the neighborhood-level socioeconomic advantage should be taken into account in addition to individual SES. Our results show that measures targeting individual-level factors such as family SES would not fully address the issue, because these types of factors are not alone in impacting BMI variability. Public policy needs to take a broader perspective and integrate the macrolevel living environment in

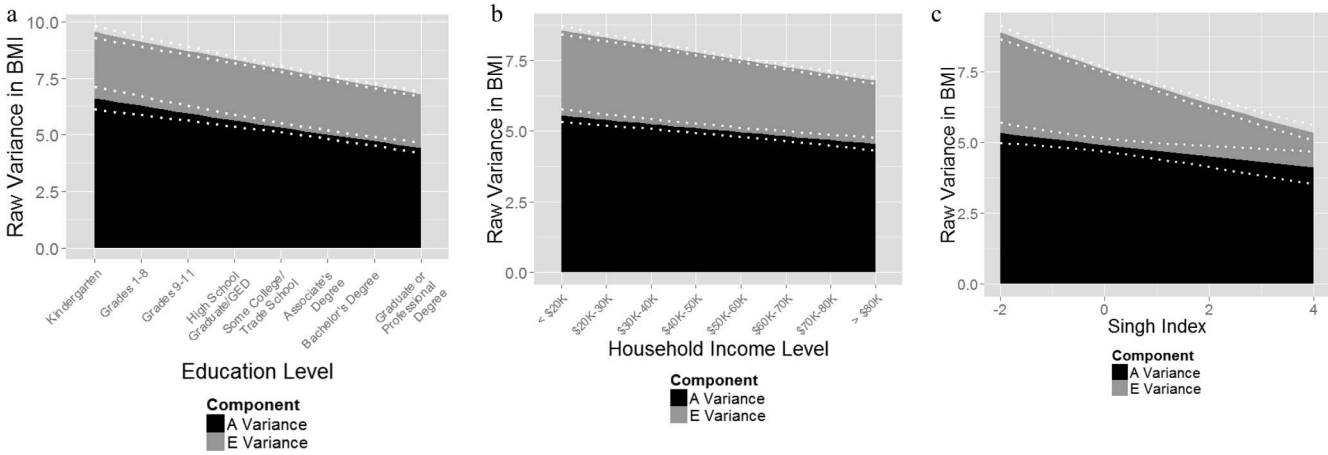

Figure 3. Residual variance in BMI as a function of individual- and neighborhood-level socioeconomic status. The stacked variance plots illustrate how the A, E, and total residual variance in BMI decreases with increasing education (a), income (b), and area deprivation (c). The dotted white lines represent the 95% confidence intervals around the change in variance.

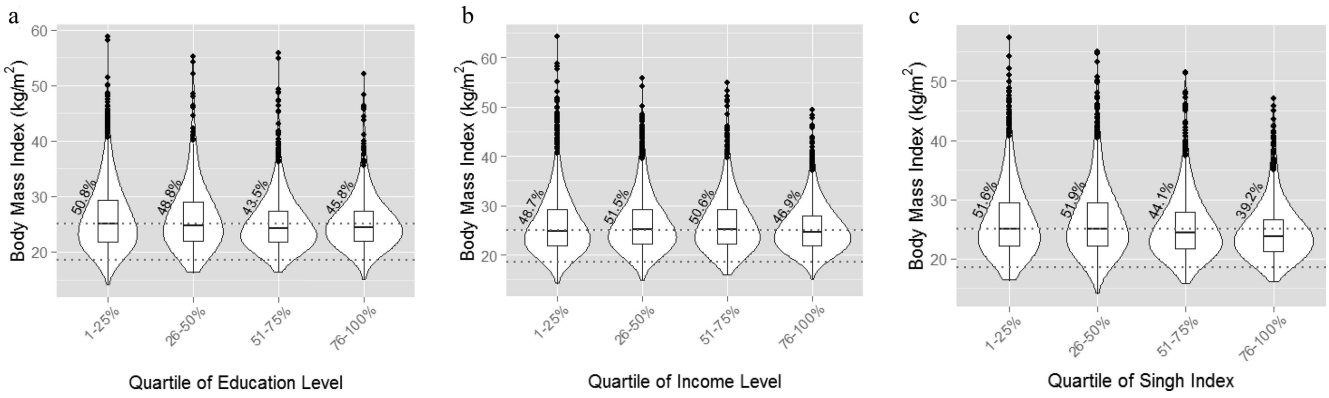

Figure 4. Box plots overlaid with violin plots, body mass index as a function of quartile of education level (a), household income (b), and area deprivation (c). Normal weight (18.5 kg/m^3 to 25 kg/m^3) falls between the two dotted red lines. The sample was divided according to quartiles of individual- and neighborhood-level SES. The boxplots of the distribution of BMI grow narrower as a function of increasing SES, demonstrating that the 25th and 75th percentiles cover a narrower range of BMI at greater levels of socioeconomic advantage. Similarly, the violin plots (which provide information about the probability density of the subsamples) grow shorter and wider with increasing SES, further reflecting decreased variance in BMI. The percentage next to each violin plot is the proportion of the subsample that is overweight or obese (BMI $\geq$25 kg/m^3), and tends to decrease as SES increases.

implementing measures that would contribute to a long-term reduction in obesity (Gearhardt et al., 2012).

Despite the use of twin models to aid in the analysis of causal and noncausal associations between SES and BMI, the correlational and cross-sectional nature of our data limits our ability to draw true causal conclusions. Although the genetically informed methodology we are using is an ideal way to draw *quasi-causal* conclusions based on the available data (Turkheimer & Harden, 2013), future studies should replicate our findings and that of other researchers (Johnson & Krueger, 2005a; Johnson et al., 2011; Osler et al., 2008; Webbink et al., 2010) using a longitudinal design. Our sample lacked ethnic diversity, so our findings generalize primarily to Caucasian populations. Similarly, although our sample is representative of the U.S. in terms of educational attainment ("Educational Attainment in the United States: 2013," 2013), it is slightly above national averages in terms of income (our sample's median was \$60K–\$70K, whereas median household income in the U.S. in 2013 was \$50K [DeNavas-Walt & Proctor, 2014]), and below national averages in terms of BMI (69% of the adult U.S. population is considered to be overweight or obese [National Institute of Diabetes and Digestive and Kidney Diseases, 2014], whereas in our sample it was 48.3%). Future research might consider examining populations which more closely resemble national averages for ethnic composition and SES variables. Lastly, we did not examine sex differences, a potentially important distinction in this context. Past G $\times$ E studies suggest, however, that the interactive effect of SES on residual variance in BMI shows similar patterns of results in men and women (Johnson et al., 2011).

In all of our models, genetic and nonshared environmental residual variance of BMI was moderated by differences in levels of education, income, and neighborhood-level socioeconomic advantage. Substantively, this is interpreted as an interaction between the socioeconomic environment and the magnitudes of genetic and nonshared environmental components of individual differences in BMI. That is, the influence of SES on BMI extends beyond its main effects, facilitating or restricting the expression of individual differences that are related to genetic and nonshared environmental factors. Low SES is, if not a direct cause of high BMI, an important influence on the forces that regulate body weight, independent of genetic and environmental variables that vary between twin pairs reared together. The effects of neighborhood-level SES factors on BMI variance are similar to those of individual-level variables, meaning that the environment in which we live, even the aspects of it not directly modifiable by ourselves, can moderate our genetic propensity for BMI. The pathways through which SES impacts body weight are very complex and cannot be described by simply asserting that poor people are heavier.

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Correction to Hatzenbuehler, Slopen, and McLaughlin (2014)

In the article "Stressful Life Events, Sexual Orientation, and Cardiometabolic Risk Among Young Adults in the United States" by Mark L. Hatzenbuehler, Natalie Slopen, and Katie A. McLaughlin (*Health Psychology,* 2014, Vol. 33, No. 10, 1185–1194, http://dx.doi.org/10.1037/hea0000126), the name of author Katie A. McLaughlin was misspelled as Kate A. McLaughlin. The online version of this article has been corrected.

http://dx.doi.org/10.1037/hea0000345