# What Money Can (and Can't) Buy: A Behavior Genetic Analysis of the Physical and Mental Health Benefits of Socioeconomic Advantage in Modern America

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## Abstract

Socioeconomic status (SES) has long been observed to predict health and psychosocial functioning: The wealthy tend to be happier, healthier, and live longer than their poorer counterparts. Using contemporary samples of adolescent and adult American twin pairs raised in the same household (the National Longitudinal Study of Adolescent Health and the Washington State Twin Registry), this dissertation takes two approaches to investigating the SES–health gradient. First, individual- and family-level socioeconomic indicators (*e.g.*, income, educational attainment, family income during childhood) as well as community-level measures of socioeconomic advantage or inequality (*e.g.*, Area Deprivation Index, Gini Index) were used to systematically evaluate the effect of SES—controlling for genetic confounds that often lead to biased or spurious findings—on mean levels of mental and physical health and health behaviors. Second, the influence of SES indicators on genetic and environmental risk for the same range of health outcomes was evaluated to understand whether SES makes some individuals more or less susceptible to expressing a particular health phenotype. We hypothesized that the SES–health gradient is largely an artifact of gene-environment correlation, and the data decisively supported this prediction. We also hypothesized that genetic diathesis for negative health outcomes or behaviors would be greatest in the least advantaged environments. Instead, we observed that *environmental risk* for poor health is exacerbated by socioeconomic deprivation. Our results suggest that in 21$^{st}$ century America, money does not buy better health and well-being at the population level. On the other hand, reducing the socioeconomic burden of those at the lowest tier of the socioeconomic spectrum will yield better health for those at greatest environmental risk for poor health.

## Dedication

This dissertation is dedicated to the socioeconomically deprived and to the public servants who tirelessly advocate for them.

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$$X = \begin{bmatrix} A & U & X & R & C \\ T & Y & S & V & M \\ N & X & X & Y & H \\ H & O & O & E & U \\ K & X & X & X & ! \end{bmatrix} \qquad Y = \begin{bmatrix} 0 & 0 & 1 & 0 & 0 \\ 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 \\ 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 \end{bmatrix}$$

Evaluate: X'Y

# Chapter 1: Introduction

## Overview

Socioeconomic disparity and health disparity are increasingly becoming synonymous terms in 21st century America. History has long observed this socioeconomic status (SES)–health gradient (*e.g.*, Chapin, 1924; Schereschewsky, Warren, & Sydenstricker; 1916; Smith, Carroll, Rankin, & Rowan, 1992): The wealthy are healthier physically and psychologically and live longer than their poorer counterparts (*e.g.*, Adler et al., 1994). The SES–health gradient follows a dose-response trend (Adler et al., 1994; Lorant et al., 2003; Marmot et al., 1991) and exists despite national wealth (Adler et al., 1994; Antonovsky, 1967), transcends political systems (Adler & Snibbe, 2003), is independent of method of healthcare delivery (Adler & Snibbe, 2003), and persists despite improvements to overall health associated with modern medicine (Mackenbach et al., 2003; van Doorslaer and Koolman, 2004; Kunst et al., 2005; Shkolnikov et al., 2011). More recently, income inequality has also gained attention as a significant predictor of health. Population-level life expectancy is shorter in societies with greater differences between the rich and poor (Wilkinson, 1992; Wilkinson & Pickett, 2008), and the United States ranks among developed countries as the world's most inequitable societies with respect to financial resources (Central Intelligence Agency, n.d.).

This body of research has not gone unrecognized by the United States Government and public policy makers. Among the primary objectives of the U.S. Department of Health and Human Services' *Healthy People 2020* initiative is to "achieve health equity, eliminate disparities, and improve the health of all groups" (Office of

Disease Prevention and Health Promotion, 2010). The Affordable Care Act, signed into law in 2010 and expanding health insurance coverage to all American citizens, is expected to redistribute income to the bottom 20% of earners (Aaron & Burtless, 2014), thereby reducing economic disparity between the rich and the poor and its associated health inequities. Of course, this well-established, long observed relationship is not without its shortcomings. Whether the SES–health gradient represents a causal process is a topic of much debate.

**Socioeconomic Status: Definition & Measurement**

Broadly defined, socioeconomic status refers to "the placement of persons, families, households and census tracts or other aggregates with respect to the capacity to create or consume goods that are valued in our society" (Miech & Hauser, 2001, p. 75). As this definition implies, SES is not limited to the individual- and family-levels, but can be measured at higher levels of organization as well (*e.g.*, neighborhood- or census tract levels). Indicators of socioeconomic status fall under two broad categories, compositional and contextual measures of SES, each addressing different possible mechanisms through which the SES–health gradient operates.

***Compositional SES Measures.*** Compositional measures of SES typically focus on individual- and family-level socioeconomic conditions. Compositional SES is commonly measured using income, wealth, educational attainment, occupation, and employment. Importantly, these SES indicators are non-interchangeable components of a multidimensional construct, each potentially affecting health differently (Pampel, Krueger, & Denney, 2010) and possessing a unique set of strengths and limitations (Shavers, 2007). For example, education, which strongly influences health behaviors

(Pampel et al., 2010), is readily measured and tends to be stable after young adulthood, but is imperfectly related to economic return (Shavers, 2007). Current income, which may be particularly important for men's health in later life (Luo & Waite, 2005), is similarly easy to measure, but is relatively age-dependent and tends to be less stable than other measures of SES (Shavers, 2007).

***Contextual SES Measures.*** Contextual measures of SES assess economic conditions affecting all individuals sharing a particular social environment (Kaplan, 1999). Commonly used indicators of contextual SES include the Area Deprivation Index, a composite measure of area-level socioeconomic disadvantage (Singh, 2003; Kind et al., 2014), and the Gini Index, a measure of income distribution that is widely used as a proxy for income inequality (Blakely, Lochner, & Kawachi, 2002; Cowell, 1977). Such contextual measures are useful for understanding the important role of the social environment in mental and physical health research, but they, too, are not without their shortcomings. The accuracy and validity of contextual measures depend on the time elapsed since data collection and compositional changes within the geographic area of interest (Blakely, Kennedy, Glass, & Kawachi, 2000; Shavers, 2007). In addition, the strength of the association of contextual factors with health depends on level of aggregation. For example, income inequality measured in larger catchment areas is a better predictor of mortality than income inequality based on smaller catchments (Wilkinson & Pickett, 2008).

***Comparing Compositional and Contextual SES.*** Contextual measures of SES are weakly to moderately correlated with compositional measures of SES (Demissie, Hanley, Menzies, Joseph, & Ernst, 2000; Greenwald, Polissar, Borgatta, & McCorkle,

1994; Marra, Lynd, Harvard, & Grubisic, 2011) and tend to show less robust associations with health than do compositional SES measures (Pickett & Pearl, 2001). Contextual SES also predicts health independent of compositional SES (Kondo et al., 2009).

***Subjective Versus Objective Socioeconomic Position.*** There is some evidence that subjective SES, or social standing, is more strongly correlated with health than objectively measured SES or social status (Adler, Epel, Castellazzo, & Ickovics, 2000; Ostrove, Adler, Kuppermann, & Washington, 2000; Singh-Manoux, Marmot, & Adler, 2005), and predicts health independent of objectively measured SES (Schnittker & McLeod, 2005). This dovetails with research suggesting that relative deprivation generally explains the SES–health gradient better than an absolute deprivation or a deprivation threshold model (Adler et al., 1994).

**Socioeconomic Status and Mental Health**

Low socioeconomic status is a risk factor for mental health problems, including internalizing (*e.g.*, emotional problems), externalizing (*e.g.*, attention-deficit/hyperactivity disorder, delinquent behavior), and greater expression of maladaptive personality traits (*e.g.*, neuroticism). In children (Bøe, Øverland, Lundervold, & Hysing, 2012; Gilman, Kawachi, Fitzmaurice, & Buka, 2002), adolescents (Goodman, 1999; Lemstra et al., 2008b), and adults (Ansseau et al., 2007; Green & Benzeval, 2013; Lorant et al., 2003; Zimmerman & Katon, 2005), lower SES is associated with increased risk for depression or depressive symptoms and suicide, and this relationship strengthens with age (Miech & Shanahan, 2000). Socioeconomic status also accounts for a large proportion of the disease burden for depression (26-40% depending on the SES indicator) among adolescents (Goodman, Slap, & Huang, 2003).

Anxiety symptoms (Ansseau et al., 2007; Lemstra et al., 2008b; Green & Benzeval, 2013; South & Krueger, 2011; Tambs et al., 2012) and neuroticism characteristics (Jonassaint, Siegler, Barefoot, Edwards, & Williams, 2011; South & Krueger, 2011) are similarly associated with lower SES across the lifespan. Socioeconomic factors (particularly parental education) are also implicated in externalizing problems in adolescents (Amone-P'Olak et al., 2009; Bøe et al., 2012; Huisman et al., 2010), including aggression (Amone-P'Olak et al., 2009), delinquency or misconduct (Amone-P'Olak et al., 2009; Bøe et al., 2012), and symptoms of attention-deficit/hyperactivity disorder (Amone-P'Olak et al., 2009; Bøe et al., 2012; National Center for Health Statistics, 2012). There is also evidence that lower SES impedes the gradual increase in mental health that occurs during mid- to later adulthood (Williams, Cunich, & Byles, 2013).

Most of the existing research on the SES-mental health link has used compositional measures of SES; those using contextual measures typically yield similar results. Lower community SES (indicated by median income, education level, and occupational status within a given ZIP code) is associated with more depressive symptoms (Muramatsu, 2003) and greater risk for mental disability and psychiatric hospitalization, especially among low- and middle income groups (Hudson, 2005). In a study of individuals with borderline, avoidant, schizotypal, or obsessive-compulsive personality disorders, neighborhood-level SES was associated with more severe personality pathology and lower levels of functioning and social adjustment, but only among those at highest individual-level socioeconomic risk (Walsh et al., 2012). This relationship held even while controlling for compositional SES factors. Poverty and income inequality are also associated with violent crime, especially homicide and assault

(Hsieh & Pugh, 1993).

**Socioeconomic Status and Physical Health**

Low socioeconomic status is also a risk factor for a multitude of physical health problems across the lifespan. The SES–health gradient is present across a wide spectrum of diseases that carry elevated burden for morbidity and premature mortality (Adler et al., 1994; Adler & Ostrove, 1999; Eibner & Evans, 2005; Goodman et al., 2003). Low SES is associated with lower self-rated health (Eibner & Evans, 2005; Goodman, 1999) and increased risk for upper and lower gastrointestinal symptoms (Bytzer et al., 2001), asthma (Almqvist, Pershagen, & Wickman, 2005; Basagaña et al., 2004; Cesaroni, Farchi, Davoli, Forastiere, & Perucci, 2003), and rhinitis, arthritis and other musculoskeletal conditions (Bengtsson et al., 2005; Cunningham & Kelsey, 1984), and cardiovascular disease (for a review, see Clark, Desmeules, Luo, Duncan, & Wielgosz, 2009). Low socioeconomic status is also related to higher body mass index (BMI) and obesity (Eibner & Evans, 2005) and greater incidence of diabetes (Lee et al., 2011; Stringhini et al., 2013) and diabetes-related complications or mortality (Saydah & Lochner, 2010; Secrest et al., 2011) as well as higher rates of rehospitalization in patients with congestive heart failure, pneumonia, and myocardial infarction (Kind et al., 2014). Poorer self-rated health (Goodman, 1999) and obesity (Ogden, Lamb, Carroll, & Flegal, 2010) during childhood and adolescence is associated with lower parental education and family income. In a natural experiment of the SES–health gradient, one study showed that individuals coming from U.S. states that had compulsory school laws show better self-rated health, less cardiovascular disease, fewer weight problems, and lower mortality

rates (Fletcher, 2015; Lleras-Muney, 2005). Lower SES also hastens the gradual decline in health that begins in middle adulthood (Williams, Cunich, & Byles, 2013).

Socioeconomic status affects functioning of the immune system as well. Lower SES is associated with compromised cell-mediated immunity, such as decreased white blood cell count (Chen et al., 2006) and increased antibody response to latent cytomegalovirus (Dowd & Aiello, 2009; Dowd, Haan, Blythe, Moore, & Aiello, 2008) and herpes simplex virus type 1 (Dowd et al., 2008) infections. Inflammatory responses are similarly affected by socioeconomic factors. Lower SES is associated with higher production of cytokines implicated in asthma (Chen et al., 2006) and higher C-reactive protein levels (Alley et al., 2006; Deverts, Cohen, Kalra, & Matthews, 2012).

Research using contextual measures of SES conforms to that using compositional measures. Income inequality predicts increased mortality and poorer health as many as 15 years later (Blakely et al., 2000; Blakely, Lochner, & Kawachi, 2002) and independently of individual- or family-level income (Kondo et al., 2009; Subramanian, Kawachi, & Nemmedy, 2001; van Deurzen, van Oorschot, van Ingen, 2014). An early study of income inequality and mortality suggested this correlation may be as high as –0.86 (Wilkinson, 1992; but see Beckfield, 2004 and Wilkinson & Pickett, 2006). Neighborhood socioeconomic deprivation is similarly independently associated with lower self-rated health (Stafford & Marmot, 2003), higher waist-to-hip ratio (Stafford & Marmot, 2003), and poorer sleep (Watson et al., 2015). Research suggests that low neighborhood- or area-level SES is associated with increased risk of tuberculosis infection (Cantwell, McKenna, McCrae, & Onorato, 1997; Lopez de Fede, Stewart,

Harris, & Mayfield-Smith, 2008; Oren, Koepsell, Leroux, & Mayer, 2012), coronary heart disease (Diez-Roux et al., 1997; Sundquist, Malmström, & Johansson, 2004), cardiovascular disease and death (Diez-Roux, Borrell, Haan, Jackson, & Schultz, 2004), diabetes (particularly non-insulin dependent or type 2 diabetes; Connolly, Unwin, Sherriff, Bilous, & Kelly, 2000; Evans, Newton, Ruta, MacDonald, & Morris, 2000; Haynes et al., 2006; Rabi et al., 2006), asthma (Basagaña et al., 2004; Cesaroni et al., 2003), and mortality (Singh, 2003).

**Socioeconomic Status and Health Behaviors**

Socioeconomic deprivation increases the likelihood of taking health risks or engaging in fewer healthy behaviors or activities (Eibner & Evans, 2005; Hanson & Chen, 2007). Research and theory suggest that few detrimental health behaviors tend to be related to high SES (Bronfenbrenner & Ceci, 1994; Hanson & Chen, 2007), although in terms of substance use, the SES–health behavior association profile may depend on the type of substance examined, how substance use is defined, and the age of the sample. For example, low SES is related to greater alcohol, marijuana, and cocaine use in late childhood and early adolescence (Lemstra et al., 2008b; Goodman & Huang, 2002), but higher parental income and education are related to increased use of these substances in young adulthood (Humensky, 2010). Higher SES is commonly related to increased substance use, but problematic use tends to be more characteristic of individuals of lower socioeconomic status. Adolescents from affluent families are more likely to experiment with or engage in low-level cannabis use, but less likely to report frequent or problematic use (Hanson & Chen, 2007; Legleye, Beck, Khlat, Peretti-Watel, & Chau, 2012). Similarly, higher income young adults consume alcohol more frequently, but do so in

lesser quantities compared with their lower-SES counterparts (Casswell, Pledger, & Hooper, 2003). In adults, lower SES is associated with greater alcohol use and abuse and more alcohol-related problems (van Oers, Bongers, van de Goor, & Garretsen, 1999). Unlike other categories of substance use, cigarette use appears to be robustly related to lower SES (Eibner & Evans, 2005; Goodman & Huang, 2002; Hiscock, Bauld, Amos, Filder, & Munafò, 2012; Patrick, Wightman, Schoeni, & Schulenberg, 2012). Physical activity is similarly inversely related to individual-level socioeconomic status (Eibner & Evans, 2005; Janssen, Boyce, Simpson, & Pickett, 2006), although research using contextual measures of SES (discussed below) predominate over compositional measures.

Studies using contextual measures of SES tend to mirror those using compositional measures. More frequent moderate substance use is observed in higher-SES neighborhoods, whereas problematic use is more prevalent in poorer neighborhoods (Galea, Ahern, Tracy, Rudenstine, & Vlahov, 2007). Neighborhood socioeconomic deprivation and income inequality are associated with increased risk for heavy drinking, an association that strengthens with age (Fone, Farewell, White, Lyons, & Dunstan, 2013; Karriker-Jaffe, Roberts, & Bond, 2013). Income inequality is also implicated in more frequent marijuana use (Galea, Ahern, Tracy, & Vlahov, 2007), and drug overdose deaths are more common in less equitable neighborhoods (Galea et al., 2003). Cigarette smoking, on the other hand, appears to be consistently related to neighborhood socioeconomic deprivation (Shohaimi et al., 2003). Compared with people living in higher SES neighborhoods, people living in low SES neighborhoods tend to have better access to activity-promoting facilities but are less likely to utilize them (Giles-Conrti &

Donovan, 2002). In adolescents, physical activity decreases and sedentary behaviors (*e.g.*, watching television or playing computer or video games) increase with increasing area-level socioeconomic deprivation (Brodersen, Steptoe, Boniface, & Wardle, 2007). Living in a poverty area is similarly associated with less physical activity, but also a greater decline in physical activity over time compared with living in a non-poverty area (Yen & Kaplan, 1998). Some research suggests differential effects of income- and education-based neighborhood SES: poorer neighborhood residents walked more, whereas more educated neighborhood residents also walked more (Ross, 2000).

**Navigating Causality in SES-Health Gradient Research**

There is little doubt that socioeconomically advantaged individuals enjoy better health than their disadvantaged counterparts, but how can we know whether this relationship is causal? It is plausible that socioeconomic resources buffer against poor health outcomes or potentiate other factors that protect against poor health. It is equally plausible, however, that superior physical or mental health contributes to socioeconomic successes, or that background factors contribute to both socioeconomic success and good health. Short of random assignment into socioeconomic strata, such selection hypotheses are impossible to rule out.

Causal effects of socioeconomic status on health are often assumed after statistically adjusting for factors known to be associated with both SES and health (*e.g.*, health care coverage or utilization). Traditional social science studies, however, are limited to controlling for measured covariates, typically only environmental ones. Natural experiments of the SES–health gradient, which compare health outcomes of individuals experiencing external events or factors such as random increases in income or

shifts in education policy, have largely supported a causal link between these phenotypes. Evans and colleagues (Evans, Wolfe, & Adler, 2012) provide an excellent review of such experiments, but we offer a short summary here.

Studies of the impact of compulsory schooling policies on health outcomes suggest that greater educational attainment is associated with higher self-rated health (Adams, 2002; Arendt, 2005; Oreopoulos, 2006), healthier body mass index (Arendt, 2005), greater functional ability (Adams, 2002; Oreopoulos, 2006), and lower mortality rates (Lleras-Muney, 2005). Educational opportunities are similarly related to smoking behavior (Currie & Moretti, 2003; de Walque, 2007; Grimand & Parent, 2007) and infant health (Currie & Moretti, 2003). Sudden increases in income is related to better general health (Case, 2004; Lindahl, 2005), lower risk of being overweight or obese (Lindahl, 2005; Wolfe, Jakubowski, Haveman, & Courey, 2012), lower risk of externalizing disorders in children (Costello, Compton, Keeler, & Angold, 2003), less smoking and heavy drinking (Wolfe et al., 2012), and lower mortality rates (Lindahl, 2005). Expansion of federal aid programs such as food stamp provisions have yielded similar results for outcomes related to infant health (Almond, Hoynes, & Schanzenbach, 2011) and the physical and mental health of women (Evans & Garthwaite, 2014). A cash transfer program also showed benefits for height and weight in a sample of Mexican youth (Fawley & Juvenal, 2010). Other natural experimental studies, however, have observed negligible to no effects (Clark & Royer, 2013; Currie & Moretti, 2008; Fawley & Juvenal, 2010; Frijters, Haisken-DeNew, & Shields, 2005; Meer, Miller, & Rosen, 2003), and some have observed effects in the opposite direction for income (Adda, von Gaudecker, & Banks, 2009; Snyder & Evans, 2006).

A different sort of natural experiment, genetically informed research designs, control for genetic selection effects and many *unmeasured* environmental selection effects as well. Genetically informed research designs, and twin and sibling studies in particular, offer traditional correlational studies an additional layer of control for parsing selection from causation. By using sibling pairs of varying degrees of genetic relatedness who have been reared together, it is possible to examine a phenotypic—or observed—relationship *after* taking into account genetic and shared environmental confounds (Turkheimer & Harden, 2014; Turkheimer & Waldron, 2000). For example, any observed difference between identical twins discordant for an environmental exposure (*e.g.*, education level, neighborhood-level socioeconomic advantage) cannot be attributable to genetic or shared environmental selection (D'Onofrio et al., 2005; Kendler et al., 1993), and therefore must be the result of environmental factors not shared between twins.

To illustrate, consider an identical twin pair and a fraternal twin pair, each with members occupying different socioeconomic strata. Suppose these twins inherited genetic characteristics that decreased their risk of poor health and increased their likelihood of pursuing higher education or living in a wealthier neighborhood. Despite observing a relationship between SES and health at the population level, we would find no relationship when comparing the identical twins (*i.e.*, the more educated member of the pair would not be less healthy than her less educated co-twin, because the relation is genetically-mediated[1]).

Suppose now that growing up these twin pairs were exposed to environmental

[1] Genetically-mediated pathways are often referred to as gene-environment correlation, or *r*GE.

factors—such as socioeconomic status of the family of origin or neighborhood characteristics—which are related to both health and socioeconomic success. A population-level relationship would again be observed, but in this instance it would be caused by familial experiences shared between twins. In this case, we would find no differences in health associated with SES when comparing either the identical or the fraternal twins, because the relation is mediated by the environment shared between members of sibling pairs.

Finally, suppose that socioeconomic status *is* causally related to health. In this case, non-shared experience (either SES or something correlated with SES within sibling pairs) accounts for differences in outcomes even after controlling for genetic and environmental confounds shared by members of the same family. We would find that the socioeconomically advantaged identical and fraternal co-twins enjoyed better health than their (relatively) socioeconomically disadvantaged counterparts (although differences may be larger for the fraternal than the identical pair if genetic selection partially accounted for the association).

In pointing to the benefits of genetically informed designs, we note that observing a significant phenotypic association after controlling for genetic and shared environmental confounds is consistent with a causal relationship. It is not, however, probative, because the possibility always exists (in the absence of random assignment) that other nonshared environmental factors are responsible for the observed effect (*e.g.*, one twin may be married, which is correlated with both higher household income as well as better overall health). Although such possibilities exist, by controlling for all possible genetic and shared environmental confounds—measured or *unmeasured*—twin studies

provide a rigorous test of whether an observed effect is due to selection.

**Socioeconomic Status as a Potentiator of Genetic Risk for Poor Health**

Genetically informed studies are useful not only for evaluating the pathways through which socioeconomic status influences level of health (*i.e.*, genetic, shared environmental, or nonshared environmental), but also provide a different perspective on the SES–health gradient, one that cannot be addressed using traditional correlational studies. Genetically informed studies can assess how genetic and environmental variance in health can be impacted by socioeconomic factors. Perhaps most interesting is the effect of SES on genetic variance in health, which (depending on the effect of SES on level of health and the direction of change in variance) can be interpreted as restricting or potentiating genetic risk for poorer health. This is a form of genotype-by-environment interaction (G×E interaction).

Distinguishing between selection and causation processes in genotype × socioeconomic status interaction research of the SES–health gradient is not as straightforward as genetically informed analysis of the influence of SES on level of health, however. The key is in interpreting how variance in health changes with SES *in the context of how SES influences level of health.* Johnson (2007) outlined social selection and social causation interpretations of G×E interaction in a seminal report on the SES–health gradient from a genetically informed perspective. According to Johnson (2007), social selection predominates when genetic variance and *r*GE are high at the same end of the environmental range, whereas social causation is implicated when genetic variance is high where *r*GE is low. "The primary marker of a social causation process is that it involves moderation of genetic and/or environmental variance *unique* to

the trait rather than genetic and/or environmental variance *common* to the trait of the social cause" (Johnson, 2007, p. 433). Johnson also discussed causal interpretations in the context of the stress–diathesis and related models (Bronfenbrenner & Ceci, 1994; Shanahan & Hofer, 2005). The stress-diathesis model (Bronfenbrenner & Ceci, 1994) suggests that environmental stress potentiates expression of dysfunctional traits for which a latent predisposition exists, and is relevant when genetic or environmental variance expands in poor or more stressful environments (Johnson, 2007). The stress-diathesis model also posits that environmental potentiation of positive or functional traits will occur in more advantaged environments (Bronfenbrenner & Ceci, 1994). Shanahan & Hofer (2005) offer additional, more mechanistic causal interpretations of G×E processes. *Social context as compensation* is an interpretation that is relevant when positive environmental factors suppress the genetic risk for expressing a dysfunctional trait. *Social context as social control* involves the social or institutional constraints on behavior that translates to buffering genetic diathesis by guiding behavior or limiting choices. Finally, *social context as enhancement* is present when positive environmental factors potentiate expression of positive or adaptive traits.

**Genetically Informed Research on the SES-Health Gradient**

Reducing the health gap between the wealthy and the poor may not be as simple as balancing socioeconomic advantage. It is a commonplace finding in behavior genetics research that the SES–health gradient does not replicate within pairs of individuals who are genetically identical but have differential exposure in terms of socioeconomic advantage. Instead the relation appears to be due to a genetic background common to both SES and health; that is, the association between SES and many health outcomes is

not driven by socioeconomic disadvantage and is in fact spurious. Complicating the picture further, SES appears have an observable effect on genetic risk for certain health issues independent of its effect on level of health, with low SES exacerbating genetic risk for certain health outcomes. What follows is a systematic, comprehensive review of the behavior genetics literature on the SES–health gradient, organized by topic and publication date.

***Mental Health.***

**Internalizing Symptomatology.** Several genetically informed studies have investigated the link between socioeconomic status and internalizing symptoms or disorders. These studies have primarily used compositional measures of SES. In the first of these studies, Osler, McGue, and Christensen (2007) examined the relation between social class (determined based on type of employment, vocational education, and number of employee subordinates) and depressive symptoms in 664 identical (monozygotic, or MZ) and 602 fraternal (dizygotic, or DZ) pairs of middle-aged Danish twins. At the phenotypic level, there were no differences in depressive symptoms as a function of social class in women, whereas depression scores among men were statistically significantly higher in the lower social class. Within pairs of MZ twins discordant for social class, however, the differences in depressive symptom scores were substantially reduced and not statistically distinguishable from zero. Pair differences were slightly larger (but nonsignificant) within discordant DZ twin pairs, suggesting genetic mediation of this association.

South and Krueger (2011) investigated the association between household income and variance in internalizing psychopathology in 367 MZ and 352 DZ middle-aged

American twin pairs. Internalizing psychopathology was a latent factor indicated by DSM-III (American Psychiatric Association, 1987) symptom counts for depression, generalized anxiety, panic attacks, and a scale score for a Five Factor Model-based (Lachman & Weaver, 1997) neuroticism measure. The phenotypic correlation between income and internalizing (–0.06) was statistically significant. Additive genetic influences[2] accounted for 37% of the variance in internalizing psychopathology and nonshared environmental factors[3] accounted for 63%; the shared environment[4] did not contribute to variation in internalizing. Variance in household income was attributable to additive genetic (21%), shared environmental (14%), and nonshared environmental (65%) influences. In the unmoderated model (*i.e.*, when variance in internalizing was not permitted to vary with household income), the genetic correlation between household income was substantially higher than the nonshared environmental correlation (0.33 versus –0.05) and accounted for approximately 74% of the total phenotypic correlation (the report does not include information about the statistical significance of these parameter estimates). An omnibus test suggested that household income significantly moderated variance in internalizing psychopathology, although this effect appears to have been driven primarily by decreasing E variance with increasing household income. In this moderated model, the nonshared environmental correlation between income and

[2] Additive genetic influences, abbreviated A, are the cumulative effects of the genome. Additive genetic influences represents a type of between-family effect (*i.e.*, serves to make members of the same family more alike, but makes families different from one another).

[3] Nonshared environmental influences, abbreviated E, are all environmental experiences that are not shared by members of a sibling pair. The nonshared environment is a within-family effect (*i.e.*, serves to make members of the same family different from one another).

[4] Shared environmental influences, abbreviated C, are all environmental experiences that are shared by members of a sibling pair. The shared environment represents a type of between-family effect (*i.e.*, serves to make members of the same family more alike, but makes families different from one another).

internalizing was reduced to near zero while the genetic correlation became more robust and increased with increasing household income. The authors also tested whether the residual variance of the internalizing indicators varied as a function of household income, and found no evidence for such heteroscedasticity. This research does not support a causal association between income and internalizing disorders, nor does it support a genotype-by-environment interaction. Rather, this research suggests that this relationship is genetically mediated, and that *heritability* (but not genetic variance) of internalizing spectrum disorders increases with increasing household income.

Tambs and colleagues (2012) examined the relation between education level and anxiety disorder diagnosis in 1,325 MZ and 2,014 DZ young adult Norwegian twin pairs. The phenotypic correlation between education level and anxiety disorder diagnosis was –0.30. A bivariate correlated factor model suggested that additive genetic effects account for 46% of the variation in anxiety and nonshared environmental factors accounted for the remaining (54%) of the variance; no variance was attributable to shared environmental factors. For education level, additive genetic factors accounted for 59%, the shared environment accounted for 18%, and the nonshared environment accounted for 23% of the variance. The nonshared environmental correlation between the genetic components of education and anxiety disorder diagnosis was not statistically significant. The genetic correlation, on the other hand, was statistically significant and accounted for over 80% of the association between education and anxiety disorder diagnosis. These results suggest that the association between anxiety disorders and education is non-causal, and is instead genetically mediated.

Most recently, Behrman, Xiong, & Zhang (2015) studied the effect of total years

of schooling on experience of negative affect (frequency of experiencing sadness, fear, indignation, and disgust) in a sample of 914 pairs of identical Chinese adult twins. The fixed effect of schooling on emotional experience was statistically significant and equaled a 0.035 unit decrease in frequency of negative affect per additional year of schooling. Within twins, however, this effect was nonsignificant and reduced to 0.003, consistent with a non-causal hypothesis and suggesting mediation by genetic or shared environmental effects common to both schooling and experiencing negative affect.

A single report exists investigating contextual effects on internalizing from a genetically informed perspective. Strachan, Duncan, Horn, and Turkheimer (2017) used 3,155 MZ and 1,275 DZ adult twins from the State of Washington to explore how neighborhood socioeconomic deprivation affects level of and variance in depression. Depression, indicated using a latent factor comprised of three items, was heritable (22%) and showed some influence from shared environmental factors (14%) but was heavily influenced by nonshared environmental factors (64%). Neighborhood deprivation, measured using the Singh Index (Singh, 2003), also showed modest genetic (16%) and shared environmental influence (33%) and substantial nonshared environmental influence (51%). Neighborhood deprivation was associated with depression at the phenotypic level, such that each additional unit increase in the Singh Index predicted a 0.02 increase in latent depression score. After controlling for between-family effects, this association was no longer statistically significant and was reduced by 67% (0.007); the genetic regression was substantially larger (0.14). Variance in depression increased with decreasing neighborhood-level SES, which was driven by increases in genetic variance. These results support genetic mediation of the association between neighborhood-level

SES and depression, and follow most closely with a social selection explanation of the impact of contextual SES on genetic diathesis for depression.

**Externalizing Symptomatology.** A review of the literature yielded a single genetically informed study of the association between socioeconomic status and externalizing behavior. Tuvblad, Grann, & Lichtenstein (2006) investigated the modifying effects of family- (parental education and occupation) and neighborhood socioeconomic status (ethnic diversity, education level, unemployment level, net income, and crime rates) on genetic and environmental variance in antisocial behavior (including property, drug, and violent offenses) among 849 MZ and 1,284 DZ Swedish twin pairs aged 16-17. Phenotypic correlations suggested an inverse relation between SES indicators and antisocial behavior, and the correlations were generally small, ranging from –0.05 to –0.06 for family-level indicators and –0.02 to –0.11 for neighborhood-level indicators. The biometric components of antisocial behavior differed by gender. In girls, additive genetic factors accounted for 59% of the variance in antisocial behavior, the shared environment accounted for 17%, and the nonshared environment accounted for 24%. In boys, additive genetic factors accounted for just 6% of the variance in antisocial behavior, whereas the shared environment accounted for 52% and the nonshared environment accounted for 42%. Parental occupational status significantly moderated the ACE variance components of antisocial behavior; A variance increased from low to high parental occupational status, whereas C and E variances decreased. Parental occupational status did not moderate variance in antisocial behavior among girls, and parental education had no moderating effects in either gender. Variance in antisocial behavior was similarly independent of neighborhood educational status. Ethnic diversity and

crime rate, used as proxies for neighborhood-level socioeconomic status, significantly moderated variance in antisocial behavior in girls but not in boys. Increasing neighborhood ethnic diversity was associated with increasing A and E variance in antisocial behavior and decreasing C variance. A similar pattern was observed for neighborhood crime rate, although only C variance changed significantly with crime rate.

To demonstrate the impact of neighborhood socioeconomic status on variance in antisocial behavior more globally, the researchers generated a composite score from neighborhood ethnic diversity, education level, and crime rate. Neighborhood disadvantage made no statistically significant difference to variance in antisocial behavior among girls, but was associated with decreased heritability, increased standardized C variance, and decreased standardized E variance. This finding is somewhat contradictory to results using the individual measures of neighborhood SES, in which the effects were present only in females. This may reflect the importance of examining both standardized and unstandardized ACE variances in genotype-by-environment interaction studies (see the section titled *Standardized vs. Unstandardized Variance Components in Health* in Chapter 11 for more information on this concept). Overall, however, the results of this study were consistent with the stress-diathesis model: additive genetic variance is greater in more advantaged home or neighborhood environments, and shared environmental variance has a greater role in less advantaged environments.

***Physical Health.***

**General Health.** Numerous researchers have examined the association between indicators of socioeconomic status and global health measures. In one of the earliest studies, Lichtenstein and colleagues (Lichtenstein, Harris, Pedersen, & McClern, 1992)

used 166 pairs of Swedish MZ twin pairs reared together, 99 MZ twin pairs reared apart, 221 DZ twin pairs reared together, and 238 DZ twin pairs reared apart to investigate the relationship between socioeconomic status (material resources, perceived standard of living, education, and occupational status) and self-rated health. Twins were classified as reared apart if separated from their twin prior to age 10. Phenotypic correlations between self-rated health and material resources, perceived standard of living, education, and occupational status were statistically significant (material resources = 0.10, perceived standard of living = 0.14, education = 0.15, and occupational status = 0.12). All phenotypes demonstrated influence from genetic and environmental factors (material resources: 23% A, 10% C, 56% E, and 11% rearing environment; perceived standard of living = 14% A, 14% C, 69% E, and 3% rearing environment; education = 24% A, 19% C, 37% E, and 20% rearing environment; occupational status = 44% A, 0% C, 41% E, and 14% rearing environment; self-rated health = 13% A, 11% C, 75% E, and 0% rearing environment). The genetic correlation (*r*A) between material resources and self-rated health was greater in magnitude compared with the shared environmental correlation (*r*C) and the nonshared environmental correlation (*r*E)[5] and accounted for a substantially larger proportion of the total phenotypic correlation (*r*A = 0.88 and accounted for 71% of the total phenotypic correlation, *r*C = –0.38 and 17%, *r*E = –0.04 and 11%). With the exception of education, the other indicators of SES followed a similar pattern (perceived standard of living: *r*A = 0.77 and 73%, *r*C = 0.27 and 22%, *r*E = –0.01 and 5%; education: *r*A = 0.21 and 26%, *r*C = 0.26 and 27%, *r*E = .13 and 47%; occupational level:

[5] The authors provided no information about the statistical significance of these parameter estimates.

*r*A = 0.43 and 33%, *r*C = 1.00 and 45%[6], *r*E = –0.08 and 22%). These results suggest that additive genetic and shared environmental factors tend to mediate the association between socioeconomic status and self-rated chronic illnesses and health.

Krieger and colleagues (Krieger, Chen, Coull, & Selby, 2005) used 178 MZ and 130 DZ adult female American twin pairs to examine the association between socioeconomic position (occupational class and educational attainment) and blood pressure, cholesterol levels, post-load glucose levels and self-rated health.[7] Compared with their working class co-twin, MZ and DZ twins in a non-working class occupation showed no statistically significant differences in post-load glucose levels (MZ = –3.80 mg/dL, DZ = –2.54 mg/dL). MZ twins, but not DZ twins, showed statistically significant within-pair differences on systolic blood pressure (MZ = 4.54 mm Hg, DZ = –0.98 mm Hg), diastolic blood pressure (MZ = 3.80 mm Hg, DZ = 0.33 mm Hg), and cholesterol levels (MZ = 7.82 mg/dL, DZ = –9.37 mg/dL). In addition, the working class twin was statistically significantly more likely to meet criteria for high blood pressure ($\kappa_{MZ}$ = 0.29, $\kappa_{DZ}$ = –0.10; note the opposite effect in DZ twins) and less likely to rate their health as fair or poor ($\kappa_{MZ}$ = –0.14, $\kappa_{DZ}$ = 0.43; again, note the opposite effect in DZ twins). Compared with their higher educated co-twin, MZ and DZ twins who had less than a college degree showed no differences in any of the health outcomes measured with the exception that DZ twins showed a statistically significant difference in glucose levels

[6] Although the authors reported that occupational status contained no variance attributable to the shared environment, parameter estimates are provided for this pathway, thus, we computed the contribution of *r*C to the total phenotypic correlation between occupational status and self-rated health.

[7] The authors did not calculate this association at the population level, so it is impossible to know whether examining this relation within twin pairs affected the magnitude of the association. Furthermore, differences between DZ pairs showed no consistent pattern, making it difficult to conclude whether genetic influences were at play in this association.

(MZ = –10.30 mg/dL, DZ = 15.03 mg/dL). Less educated twins were no more likely to meet criteria for high blood pressure ($\kappa_{MZ}$ = –0.01, $\kappa_{DZ}$ = 0.00), but were more likely to rate their health as fair or poor ($\kappa_{MZ}$ = 0.65, $\kappa_{DZ}$ = 0.61). The inconsistencies in the results for MZ and DZ twins in combination with failure to compute the phenotypic association between occupational status and educational attainment and the various indices of health used in this study makes it difficult to deduce the relative contributions of genetic and environmental influences on this association. Nevertheless, there is some evidence to suggest the presence of genetic, shared environmental, and nonshared environmental factors in the correlation between occupation and education and health.

Svedberg, Bardage, Sandin, and Pedersen (2006) used 2,510 MZ and 3,459 DZ adult Swedish twins to investigate the relationship between unemployment and self-rated health 25-30 years later in life. The odds of poor self-rated health was significantly related to previous unemployment (OR = 1.24), but this association was substantially reduced and no longer statistically significant when examined within pairs of MZ twins (OR = 0.76), suggesting genetic or shared environmental mediation of the influence of previous unemployment on self-rated health.

Osler et al. (2007) used 664 MZ and 602 DZ pairs of middle-aged Danish twins to study the relation between social class (determined based on type of employment, vocational education, and number of employee subordinates) and self-rated health. At the phenotypic level, being in the lower social class was statistically significantly related to greater likelihood of rating one's health as fair or poor. Within pairs of MZ twins discordant for social class, however, the differences in odds of endorsing fair to poor health were substantially reduced and not statistically distinguishable from zero,

suggesting mediation of this association by genetic or shared environmental influences.

Fujiwara and Kawachi (2009) examined the relationship between education and self-rated health using 351 MZ and 338 DZ middle-aged adult American twin pairs. At the phenotypic level, education was positively associated with statistically significantly better self-rated global health ($\beta$ = 0.06 in males and 0.07 in females) and self-rated physical health ($\beta$ = 0.08 in both males and females). Within twin pairs, these associations were reduced to nonsignificance (global health: $\beta_{MZ}$ = 0.11 and $\beta_{DZ}$ = 0.17 in men, $\beta_{MZ}$ = 0.08 and $\beta_{DZ}$ = –0.34 in women; physical health: $\beta_{MZ}$ = 0.07 and $\beta_{DZ}$ = 0.07 in men, $\beta_{MZ}$ = 0.00 and $\beta_{DZ}$ = 0.01 in women). There is some evidence to suggest that nonshared environmental influences may affect the association between education and self-rated global health, but genetic confounding is clearly present.

Johnson and colleagues (2010) used 21,522 same-sex adult Danish twin pairs (41% MZ) to examine the influence of educational attainment on health and variance in health. Health was measured using the Short Form Health Survey (Ware, Kosinsky, & Keller, 1996), which assesses overall health and functional limitations related to pain or health conditions. Health correlated with education at 0.13 in women and 0.15 in men[8]. In men, the genetic correlation between education and health increased with increasing educational attainment (0.21 at two standard deviations below the mean education level of the sample, compared with 0.75 at two standard deviation units above the mean education level). In women, the shared environmental correlation between education and health was a constant –0.26 across the range of educational attainment. In both men and

[8] The authors provided no information about the statistical significance of most of their parameter estimates, making interpretation of the study results somewhat difficult.

women, the nonshared environmental correlation between education and health was essentially zero. These results suggest that family factors, not socioeconomic status, are contributing to individual differences in health. With respect to variance in health, the researchers observed that total variance in health decreased as a function of education, and was driven primarily by decreasing A variance[9]. The authors interpreted these results as suggesting that low socioeconomic status compromised individuals' ability to "manage their health in ways that would have minimized the kinds of genetic vulnerabilities to health problems to which people of all levels of education were subject" (p. 412).

Behrman and colleagues (2011) used 5,294 MZ and 11,234 DZ adult Danish twin pairs to examine the impact of years of schooling on number of days hospitalized per year. At the phenotypic level, each additional step increase in schooling was significantly related to 0.056 fewer days hospitalized each year. Within pairs of MZ and DZ twins, this coefficient was substantially reduced and nonsignificant. Schooling predicted 0.005 more days of hospitalization in MZ twins and 0.008 fewer days of hospitalization in DZ twins. The DZ twins estimate was more robust than the MZ twin estimate, suggesting that the association between schooling and hospitalization in this sample is genetically mediated.

Gerdtham and colleagues (Gerdtham, Lundborg, Lyttkens, & Nystedt, 2012) examined the influence of income, education, and labor market status on self-rated health using 4,079 MZ and 11,357 DZ adult Swedish twin pairs. At the population level, income (averaged across 10 years) was positively associated with health, such that each additional unit increase of log-transformed income was associated with a 0.029 increase

[9] No information about the statistical significance of this test was provided, nor were parameter estimates.

in self-rated health. Educational attainment (0.006) and being self-employed (0.031) were similarly positively associated with self-rated health, whereas being employed part-time (–0.031), unemployed (–0.029), or economically inactive (–0.253) was negatively related to health status. Within pairs of MZ and DZ twins, income (MZ = 0.013, DZ = 0.022) and being self-employed (MZ = 0.031, DZ = 0.020) continued to be statistically significantly related to better health, whereas education level was no longer significantly related to health (MZ = 0.003, DZ = 0.003). Notably, the effects of income and education were substantially reduced within twin pairs, suggesting the presence of between-family confounds. Further, the effect of income was larger in DZ pairs than in MZ pairs, which is consistent with a genetically-mediated association. Like income and being self-employed, other labor market statuses also continued to be related to poorer health within twin pairs, and these associations often strengthened in magnitude (employed part-time: MZ = –0.038, DZ = –0.041; unemployed: MZ = –0.070, DZ = –0.044; economically inactive: MZ = –0.224, DZ = –0.265). These results suggest that the relation between education and self-rated health is mediated entirely by family-level confounds, and that the association between income and self-rated health is partially mediated by genetic factors. The increase in magnitude of certain labor market effects within twin pairs was also consistent with family-level confounds working in the direction opposite to the observed direction of the effect; that is, genetic or shared environmental influences acting on labor market status and health were positively correlated.

Lundborg (2013) used 347 MZ middle-aged adult American twin pairs to study the association between educational attainment and global self-rated health. There was a

statistically significant phenotypic association between years of schooling and self-rated global health, in which each additional year of education was associated with a 0.068 unit increase in self-rated health. Within MZ twins, this association remained approximately equal in magnitude (–0.067) but was no longer statistically distinguishable from zero. Taken together, although these results support confounding from family influences, they do not rule out causal effects of education on self-rated health.

Amin, Behrman, and Spector (2013) used 741 female MZ adult twin pairs from the United Kingdom to examine the relationship between schooling and cardiovascular fitness (measured by shortness of breath while walking). At the phenotypic level, schooling had a marginally significant effect on cardiovascular fitness (a unit increase in schooling equaled a 0.016 unit decrease in the shortness of breath measure). Within pairs of MZ twins, however, the effect of schooling on cardiovascular fitness was not substantially reduced (–0.012) but was not statistically significant. These results suggest that the association between years of schooling and cardiovascular fitness is confounded by genetic or shared environmental factors common to both phenotypes.

Behrman, Xiong, and Zhang (2015) investigated the relationship between schooling and self-rated health using 914 MZ adult Chinese twin pairs. Years of schooling had a marginally significant protective effect on health, such that each additional year of schooling was associated with a 0.018 unit increase in self-rated health. Within pairs of MZ twins, this effect was essentially zero and not statistically significant, suggesting full mediation of the schooling-health relationship by genetic or shared environmental factors.

Amin, Behrman, and Koehler (2015) examined the association between

schooling and self-rated health using MZ twin pairs from three separate samples of American twins: 2,164 pairs from the Mid-Atlantic Twin Registry, 655 pairs from the Minnesota Twin Registry, and 951 pairs from the NAS-NRC Twin Registry. The phenotypic association between education and self-rated health was statistically significant and ranged between a 0.07–0.08 unit increase in self-rated health for each step in schooling. Examining this association within MZ twin pairs showed a significant reduction in magnitude (ranging from 0.02 to 0.05) and was no longer statistically significant, suggesting that the association between schooling and self-rated health is not causal but mediated by genetic or shared environmental confounds.

**Disease & Chronic Illness.** Behrman and Wolfe (1989) used 500 female Nicaraguan sibling pairs to investigate the association between schooling and four disease categories (medically preventable, therapeutically treatable, preventable by public policy, and parasitic). Schooling was related to a statistically significant reduced odds of having a therapeutically treatable disease (OR = 0.92), a disease preventable by public policy (OR = 0.93), or a parasitic disease (OR = 0.91) at the phenotypic level. Within pairs of sisters, these estimates were no longer statistically significant, although their magnitude did not change substantially (therapeutically treatable OR = 0.90, preventable by public policy OR = 0.92, parasitic OR = 0.89). This results support genetic or shared environmental confounding of the association between schooling and select disease categories.

Lichtenstein et al. (1992) investigated the relationship between socioeconomic status (material resources, perceived standard of living, education, and occupational status) and self-reported chronic illnesses in 166 pairs of Swedish MZ twins reared

together, 99 MZ twin pairs reared apart, 221 DZ twin pairs reared together, and 238 DZ twin pairs reared apart. Twins were classified as reared apart if separated from their twin prior to age 10. The phenotypic correlation between material resources and perceived standard of living and chronic illnesses were statistically significant at 0.12 and 0.14, respectively; education and occupational status were not correlated with self-reported chronic illnesses. All phenotypes demonstrated influence from genetic and environmental factors (material resources: 23% A, 10% C, 56% E, 11% rearing environment; perceived standard of living = 14% A, 14% C, 69% E, 3% rearing environment; education = 24% A, 19% C, 37% E, 20% rearing environment; occupational status = 44% A, 0% C, 41% E, 14% rearing environment; self-reported chronic illnesses = 44% A, 0% C, 56% E, 0% rearing environment). The genetic correlation between material resources and chronic illnesses was greater in magnitude compared with the nonshared environmental correlation[10] (0.29 compared with 0.09; the shared and rearing environmental correlations were set to zero since the chronic illnesses phenotype contained no variation attributable to these influences) and accounted for a greater proportion of the total phenotypic correlation (61% versus 39%). The genetic correlation between perceived standard of living and chronic illnesses was similarly larger in magnitude (0.40 versus 0.08 for *r*A and *r*E, respectively) and accounted for a large proportion of the total phenotypic correlation (72% versus 28%). These results suggest that additive genetic factors tend to mediate the association between socioeconomic status and self-reported chronic illnesses.

Johnson and Krueger (2005; see also Johnson, 2007) used 367 MZ and 352 DZ

[10] The authors provided no information about the statistical significance of these parameter estimates.

middle-aged adult American twin pairs to determine the relationship between household income and variance in number of health problems. Income was moderately heritable (29%) and showed substantial influence from the nonshared environment (71%); number of chronic illnesses was also moderately heritable (A = 27%, E = 73%). At the phenotypic level, higher household income was statistically significantly associated with fewer chronic illnesses. The genetic correlation between household income and chronic illnesses was substantially larger than the nonshared environmental correlation (–0.33 vs. 0.14) and was statistically significant (the nonshared environmental correlation was not). This genetic correlation accounted for 54% of the phenotypic correlation between household income and number of chronic illnesses, and increased with increasing household income. Variance in number of chronic illnesses decreased with increasing household income, driven by decreased genetic variance. This research suggests that the effect of household income on level of health is non-causal, driven instead by genetic factors common to both phenotypes. However, results support a causal hypothesis (specifically, social context as compensation) for the effect of household income on variance in health.

Osler and colleagues (2009) examined the association between adulthood social class (indicated by type of employment, number of employee subordinates, and vocational education) and physical limitations, dental status, and fatigue in 670 MZ and 624 DZ middle-aged adult Danish twin pairs. At the phenotypic level, each step decrease in social class was associated with statistically significantly greater odds of reporting physical limitations (odds ratio [OR] = 1.20), poor dental status (OR = 1.33), and fatigue (1.13). Within twin pairs, the association of social class with physical limitations

remained statistically significant (OR = 1.13), but its association with poor dental status (OR = 1.01) and fatigue (OR = 1.06) were nonsignificant and substantially reduced[11]. With the exception of the findings for physical limitations, these results support mediation of the association between social class and health by genetic and shared environmental influences common to both health and social class, rather than a causal hypothesis that social class directly impacts health.

Behrman et al. (2015) investigated the relationship between schooling and number of chronic illnesses using 914 MZ adult Chinese twin pairs. Years of schooling had a marginally significant protective effect on health, such that each additional year of schooling was associated with a 0.018 unit increase in self-rated health and 0.053 fewer chronic illnesses endorsed. Schooling was not related to being overweight. Within pairs of MZ twins, these effects were essentially zero and not statistically significant, suggesting full mediation of the schooling-health relationship by genetic or shared environmental factors.

**Body Mass Index & Obesity.** Teasdale, Sørensen, and Stunkard (1990) used 2,015 non-familial adoptees from Denmark to examine the association between social class and BMI in adulthood. Biological father's social class was negatively associated with BMI, an association that grew stronger with age. Adoptive father's social class was not statistically significantly associated with BMI, suggesting that genetic and shared environmental factors influence the social class–BMI relationship.

Silventoinen, Lähteenkorva, Koshenvuo, & Kaprio (2004) examined the

[11] The authors pooled MZ and DZ twins, making it impossible to differentiate between genetic and shared environmental pathways from social class to health outcomes.

association between educational attainment and BMI in 2,482 MZ and 5,113 DZ adult Finnish twin pairs. Each phenotype was influenced by genetic and environmental factors. Variance in education was comprised of 45% and 46% additive genetic influences in males and females, respectively; 36% and 39% shared environmental influences; and 19% and 15% nonshared environmental influences. Variance in BMI was comprised of 71% and 46% A in males and females, respectively; 0% and 23% C; and 29% and 31% E. At the phenotypic level, the correlation between education and BMI in both genders was –0.15. The genetic correlation in between education and BMI was statistically significant at –0.20 in men and –0.32 in women and accounted for 97% and 96% of the total phenotypic correlation in men and women, respectively. The nonshared environmental correlation in men was nonsignificant (–0.04 and accounted for 3% of the total phenotypic correlation), but was statistically significant in women (0.06 and accounted for 4% of the total phenotypic correlation). The results of this study support genetic mediation of the association between education and BMI.

Krieger and colleagues (Krieger, Chen, Coull, & Selby, 2005) used 178 MZ and 130 DZ adult female American twin pairs to examine the association between socioeconomic position (occupational class and educational attainment) and BMI and waist-to-hip ratio. The authors did not calculate these associations at the population level, so it is impossible to know whether examining this relation within twin pairs affected the magnitude of the association. Furthermore, differences between DZ pairs showed no consistent pattern, making it difficult to conclude whether genetic influences were at play in this association. Compared with their working class co-twin, MZ and DZ twins in a non-working class occupation showed no statistically significant differences in

both BMI (MZ = 0.61 kg/m$^2$, DZ = –0.64 kg/m$^2$) and waist-to-hip ratio (MZ = 0.00, DZ = –0.02). Compared with their higher educated co-twin, MZ and DZ twins who had less than a college degree showed no differences in BMI or waist-to-hip ratio. As noted above, the inconsistencies in the results for MZ and DZ twins in combination with failure to compute the phenotypic association between occupational status and educational attainment and the various indices of health used in this study makes it difficult to deduce the relative contributions of genetic and environmental influences on this association. Nevertheless, there is some evidence to suggest the presence of genetic, shared environmental, and nonshared environmental factors in the correlation between occupation and education and indices of adiposity.

Johnson and Krueger (2005; see also Johnson, 2007) studied the association between household income and body mass index using 367 MZ and 352 DZ middle-aged adult American twin pairs. Income was moderately heritable (29%) and showed substantial influence from the nonshared environment (71%); number of chronic illnesses was also moderately heritable (A = 27%, E = 73%); body mass index was substantially heritably (A = 76%, E = 24%). At the phenotypic level, higher household income was statistically significantly associated with lower BMI. The genetic association between household income and BMI was larger than the nonshared environmental correlation (–0.30 vs. 0.00), was statistically significant, and accounted for 100% of the phenotypic correlation between household income and BMI. Variance in BMI decreased with increasing household income, driven by decreased genetic variance. This research suggests that the effect of household income on level of BMI is non-causal, driven instead by genetic factors common to both phenotypes. However, results support a

causal hypothesis (specifically, social context as compensation) for the effect of household income on variance in BMI.

Osler et al. (2007) used 664 MZ and 602 DZ pairs of middle-aged Danish twins to study the relation between social class (determined based on type of employment, vocational education, and number of employee subordinates) and BMI. At the phenotypic level, being in the lower social class was statistically significantly related to higher BMI; within pairs of MZ twins discordant for social class, however, the differences in BMI as a function of social status were substantially reduced and not statistically distinguishable from zero. Pair differences in BMI were slightly larger (but similarly nonsignificant) within discordant DZ twin pairs, suggesting genetic mediation of this association.

Webbink, Martin, & Visscher (2010) used 3,808 MZ adult Australian twin pairs to investigate the association between educational attainment and BMI. Multiple body measurements spanning 13 years were used in the analysis. Cross-sectional phenotypic correlations between education and the odds of being overweight were (with one exception) statistically significant and ranged from 0.97–0.98 in men and 0.98–0.99 in women. Within male twin pairs, these estimates were not reduced in magnitude and tended to be statistically significant. Within female twin pairs, however, these odds ratios were nonsignificant and equaled 1.00. Similar results were found for the effect of educational attainment on BMI. No phenotypic effect was observed in women, but in men each additional unit in educational attainment was associated with a 0.021–0.026 $kg/m^2$ decrease in BMI. Within twin pairs, this effect remained statistically significant and even showed a moderate increase in magnitude (0.036 to 0.055 $kg/m^2$ decrease in

BMI). Overall, the results from this study support a causal protective relationship between education and BMI in men.

Johnson and colleagues (Johnson, Kyvik, Skytthe, Deary, & Sørensen, 2011) examined the effect of educational attainment on body mass index (BMI) using 21,522 same-sex adult Danish twin pairs (41% MZ). Educational attainment was 30% heritable, and showed substantial influences from the shared (38%) and nonshared (32%) environments. Body mass index was also substantially heritable (47%), and showed influences from the shared (26%) and nonshared (27%) environments. At the phenotypic level, BMI correlated with education at –0.13 in women and –0.15 in men. Within twins, however, the nonshared environmental correlation between education and BMI was nearly zero (ranging from –0.02 to 0.02). On the other hand, the genetic correlation was substantially larger, ranging from –0.17 to –0.08 and accounting for 42–70% of the total phenotypic correlation in women and 57–80% in men. The shared environmental correlation was also greater in magnitude compared with the nonshared environmental correlation, ranging between –0.96 to –0.14 and accounting for 23–55% of the total phenotypic correlation in women and 17–25% in men. Overall, these results suggest that between-family confounds account for the majority of the relationship between education and BMI. The authors observed decreased total variance in BMI as a function of increasing educational attainment, an effect that was driven primarily by decreases in shared environmental variation. Additive genetic and nonshared environmental variation also decreased as a function of increasing education in women. Of note, in both men and women, heritability increased with increasing educational attainment, but this was a function of total variance decreasing at a faster rate than genetic variance. These results

show some support for a genotype-by-interaction, whereby genetic vulnerabilities for high BMI are expressed more readily in environments characterized by low socioeconomic status.

Amin, Behrman, and Spector (2013) used 741 female MZ adult twin pairs from the United Kingdom to examine the relationship between schooling and BMI. At the phenotypic level, schooling had a statistically significant protective effect on BMI (a unit increase in schooling was associated with a 0.329 $kg/m^2$ decrease in BMI) and odds of being overweight (OR = 0.97). Within pairs of MZ twins, however, the effect of schooling on BMI was substantially reduced and was no longer statistically significant (–0.052). Likewise, the odds ratio of being overweight (OR = 1.01) was not statistically distinguishable from 1.00 within MZ twins. These results suggest that the association between years of schooling and BMI is confounded by genetic or shared environmental factors common to both phenotypes.

Lundborg (2013) used 347 MZ middle-aged adult American twin pairs to study the association between educational attainment and BMI. There was a statistically significant phenotypic association between years of schooling and BMI, such that each additional year of schooling was associated with a 0.27 $kg/m^2$ decrease in BMI. This association was reduced in magnitude and was no longer statistically significant within pairs of MZ twins, such that each additional year of schooling was associated with a 0.07 $kg/m^2$ decrease in BMI. These results suggest that family influences confound the relationship between years of education and BMI.

Amin, Behrman, and Koehler (2015) examined the association between schooling and BMI using MZ twin pairs from three separate samples of American twins: 2,164

pairs from the Mid-Atlantic Twin Registry, 655 pairs from the Minnesota Twin Registry, and 951 pairs from the NAS-NRC Twin Registry. The phenotypic association between schooling and BMI was nonsignificant in one sample but significant in the other two; for each step in schooling, BMI decreased by 0.28 $kg/m^2$. The odds of being overweight was also significantly related to schooling, with a step increase reducing the odds of being overweight (OR = 0.97–0.99). Examining these associations within MZ twin pairs, each was substantially reduced and no longer statistically significant (BMI = –0.05 to –0.03, overweight odds ratio = 0.99 to 1.00). These results suggest that the association between schooling and BMI and probability of being overweight is not causal but mediated by genetic or shared environmental confounds.

Dinescu, Horn, Duncan, and Turkheimer (2015) used 2,327 MZ and 948 DZ twin pairs from the State of Washington to investigate the association between individual-level (*i.e.*, income and education) and neighborhood-level socioeconomic status and body mass index. Indicators of socioeconomic status showed influence from genetic, shared environmental, and nonshared environmental factors (education: A = 34%, C = 41%, E = 25%; income: A = 28%, C = 25%, E = 46%; neighborhood deprivation: A = 17%, C = 35%, E = 48%); BMI was not influenced by shared environmental factors, but showed influence from genes and the nonshared environment (A = 75%, E = 25%). At the phenotypic level, BMI was statistically significantly associated with education ($b$ = –0.17), income ($b$ = –0.07), and neighborhood deprivation ($b$ = –0.30), such that each of these indicators of SES were protective against high BMI. The unstandardized regressions of BMI on the additive genetic component of each SES indicator were substantially larger than unstandardized regressions on the nonshared environmental

components and were statistically significant (income: $b_A$ = –0.62, $b_E$ = 0.13; education: $b_A$ = –0.78, $b_E$ = 0.07; neighborhood deprivation: $b_A$ = –3.88, $b_E$ = 0.05). These regression coefficients tended to increase with increasing SES. Variance in BMI also decreased with increasing SES, which was driven by decreases in both genetic and nonshared environmental variances (with nonshared environmental variance tending to decrease at a faster rate). Decreasing genetic variance unique to BMI in the context of increasing common variance (which is equivalent to a higher overall genetic correlation) is suggestive of a causal influence of SES on variance in BMI. However, the results also suggest genetic mediation of the effect of SES on level of BMI. The authors did not consider the influence of compositional and contextual measures of SES simultaneously, making it impossible to infer their effects independent of one another.

**Mortality.** Behrman and colleagues (2011) used 5,294 MZ and 11,234 DZ adult Danish twin pairs to examine the impact of years of schooling on mortality. At the phenotypic level, each additional step increase in schooling was significantly related to reduced probability of mortality (ranging –0.0085 to –0.0047 depending on gender and cohort age). Within pairs of MZ and DZ twins, these coefficients tended to be substantially reduced and nonsignificant. Probability of mortality as a function of schooling ranged from 0.0127 to –0.0015 in MZ twins and –0.0071 to –0.0006 in DZ twins. Estimates in DZ twins tended to be more robust than those in MZ twins, suggesting that the association between schooling and hospitalization and mortality in this sample is genetically mediated.

Madsen, Andersen, Christensen, Andersen, and Osler (2010) used 5,260 MZ and 11,088 DZ adult Danish twin pairs to investigate the relationship between educational

status and mortality. At the phenotypic level, the hazard ratio (HR) of death between 1980-2008 according to low educational status (7 or fewer years of education) was statistically significant (HR = 1.25). Within twin pairs, however, this association disappeared and was somewhat larger in DZ twins than in MZ twins ($HR_{MZ}$ = 1.08, $HR_{DZ}$ = 1.15), suggesting the presence of genetic-based mediation of the association between education and mortality in this sample.

***Health Behaviors***

**Substance Use.** Osler et al. (2007) used 664 MZ and 602 DZ pairs of middle-aged Danish twins to study the relation between social class (determined based on type of employment, vocational education, and number of employee subordinates) and smoking (current status) and alcohol use ("safe" alcohol use status, defined as 21 or fewer drinks per week in men and 14 or fewer in women). At the phenotypic level, being in the lower social class was statistically significantly related to a higher likelihood of being a current smoker but was not related to alcohol use. Within pairs of MZ twins discordant for social class, however, no differences in likelihood of being a current smoker were observed. Pair differences in smoking odds were slightly larger (and nonsignificant) within pairs of discordant male DZ twins, suggesting genetic mediation of the relation between social class and probability of smoking in men. In women, pair differences in odds of being a current smoker did not differ by zygosity, suggesting shared environmental mediation of this association.

Amin, Behrman, and Spector (2013) used 741 female MZ adult twin pairs from the United Kingdom to examine the relationship between schooling and smoking (lifetime and current) and alcohol consumption (units per week). At the phenotypic level,

each additional step in schooling was statistically significantly associated with greater odds of never smoking (OR = 1.04), reduced odds of smoking currently (OR = 0.98), and consuming 0.24 more units/week of alcohol. Within pairs of MZ twins, however, the effect of schooling on each phenotype was reduced and no longer statistically significant (lifetime smoking OR = 0.97, current smoking OR = 1.00, alcohol consumption = 0.19 fewer units/week). These results suggest that the association between years of schooling and smoking and alcohol consumption is confounded by genetic or shared environmental factors common to both phenotypes.

Lundborg (2013) used 347 MZ middle-aged adult American twin pairs to study the association between educational attainment and smoking[12]. There was a statistically significant phenotypic association between years of schooling and smoking (–0.046), but this association disappeared and was no longer statistically significant within pairs of MZ twins (–0.014), supporting confounding from family influences in the association between schooling and smoking.

Amin, Behrman, and Koehler (2015) examined the association between schooling and lifetime smoking and alcohol consumption (drinks per week) using MZ twin pairs from two separate samples of American twins: 2,164 pairs from the Mid-Atlantic Twin Registry and 951 pairs from the NAS-NRC Twin Registry. The phenotypic association between schooling and lifetime smoking was statistically significant and protective (odds of never smoking = 1.02–1.03). Like Amin et al. (2013), alcohol consumption was positively related to schooling, where each additional step in schooling was associated with 0.12 additional drinks per week. Examining these associations within MZ twin

[12] Authors did not indicate the units for their index of smoking.

pairs, neither was statistically significant (never smoking OR = 1.01, alcohol consumption = 0.35 fewer drinks/week). These results suggest that the association between schooling and substance use is not causal but mediated by genetic or shared environmental confounds.

Behrman et al. (2015) investigated the relationship between schooling and smoking (number of packs of cigarettes smoked per day) and alcohol use (number of days drinking per week) using 914 MZ adult Chinese twin pairs. Years of schooling was significantly associated with less smoking (0.017 fewer packs smoked per day) but not frequency of alcohol use. Within pairs of MZ twins, the protective effect of schooling on smoking remained statistically significant and roughly equal in magnitude, supporting a nonshared environmental (*i.e.*, causal) effect of years of schooling on reduced cigarette smoking.

Hamdi, Krueger, and South (2015) used 350 MZ and 322 DZ middle-aged adult American twin pairs to examine the association between income, education, and alcohol use (operationalized as amount, frequency, and problems related to alcohol use). Both income and education showed influence from genetic and environmental factors (income: A = 16%, C = 16%, E = 67%; education: A = 41%, C = 31%, E = 28%). Alcohol use showed influence primarily from genetic and nonshared environmental factors (drinking amount: A = 61%, C = 0%, E = 39%; drinking frequency: A = 55%, C = 2%, E = 43%; problematic alcohol use: A = 37%, C = 0%, E = 63%). At the phenotypic level, drinking amount was not related to education ($r$ = –0.01) but was statistically significantly related to income ($r$ = 0.06). Similarly, problematic use was related to income ($r$ = 0.06) but not education ($r$ = 0.03). Drinking frequency was associated with both income ($r$ = 0.12) and

education ($r$ = 0.08)[13]. Variance in drinking amount decreased with increasing income and education, effects that were driven by decreases in genetic variance. Variance in drinking frequency was not associated with income or education, nor was variance in problematic alcohol use. The results for drinking amount support the diathesis-stress model in which variance in drinking behavior expands in poorer or more stressful environments.

**Exercise.** Krieger and colleagues (2005) used 178 MZ and 130 DZ adult female American twin pairs to examine the association between socioeconomic position (occupational class and educational attainment) and physical activity (amount of time spent engaging in activities of various intensity at home, work, or during leisure time)[14]. Compared with their working class or more highly educated co-twin, MZ and DZ twins in a non-working class or with less than a college degree showed no differences in physical activity, suggesting the presence of genetic or shared environmental mediation of the correlation between occupation and education and physical activity.

Osler et al. (2007) used 664 MZ and 602 DZ pairs of middle-aged Danish twins to study the relation between social class (determined based on type of employment, vocational education, and number of employee subordinates) and physical activity (frequency of walking, running, and biking). At the phenotypic level, being in the lower social class was statistically significantly related to a lower physical activity score in both males and females. Within pairs of MZ twins discordant for social class, however, the

[13] The authors opted to not decompose these correlations into genetic and environmental components.
[14] The authors did not calculate these associations at the population level, so it is impossible to know whether examining this relation within twin pairs affected the magnitude of the association. Furthermore, differences between DZ pairs showed no consistent pattern, making it difficult to conclude whether genetic influences were at play in this association.

differences in physical activity scores as a function of social status was substantially reduced and not statistically distinguishable from zero. Pair differences in BMI were slightly larger (and nonsignificant in males but not in females) within discordant DZ twin pairs, suggesting genetic mediation of the relation between social class and physical activity levels.

Amin, Behrman, and Spector (2013) used 741 female MZ adult twin pairs from the United Kingdom to examine the relationship between schooling and exercise (defined as moderate or heavy exercise during leisure time during the past 12 months). At the phenotypic level, each additional step in schooling was statistically significantly associated with greater odds of exercising (OR = 1.02), but this association was reduced to nonsignificance (OR = 0.99) within pairs of MZ twins. These results suggest that the association between years of schooling and exercise behavior is confounded by genetic or shared environmental factors common to both phenotypes.

Lundborg (2013) used 347 MZ middle-aged adult American twin pairs to study the association between educational attainment and exercise (number of days engaging in vigorous physical activity, such as running or lifting heavy objects, during the past month). There was a statistically significant phenotypic association between years of schooling and exercise (each additional year of schooling was associated with 0.29 additional days of exercising). This association was no longer statistically significant within pairs of MZ twins, but was equal in magnitude. Although these results support confounding from family influences, they do not rule out causal effects of education on frequency of exercise.

Amin, Behrman, and Koehler (2015) examined the association between schooling

and exercise (operationalized as regular exercise at least once weekly) using 2,164 MZ adult American twin pairs. Schooling was phenotypically associated with exercise: Each step increase in schooling represented a 4% increase in odds of regularly exercising. Within MZ twin pairs, this association was no longer statistically significant (OR = 1.01), suggesting that the association between schooling and substance use is not causal but mediated by genetic or shared environmental confounds.

**Summary**

Socioeconomic status reliably predicts physical health, mental health, and health behaviors. This health–SES gradient is observed regardless of socioeconomic indicator, and holds true for both compositional (*e.g.*, individual- and family-level SES indicators such as income or educational attainment) and contextual (*e.g.*, neighborhood-level socioeconomic advantage, area-level income inequality) measures, generally independently of the other. Evidence from genetically informed studies suggests that this association is mediated by genetic pathways that are common to both SES and health or health behaviors, suggesting that a causal interpretation for the SES–health gradient (as it relates to level of health) may not be appropriate. Further complicating the picture is a small body of genotype-by-environment interaction research suggesting that more socioeconomically deprived environments may potentiate genetic risk for expressing dysfunctional traits, and these studies are mixed regarding whether this represents a causal versus a selection process. More research is needed to understand how socioeconomic status affects level of health *in the context of* how it simultaneously affects the influence of genetic and environmental factors on health. This dissertation research integrates these two perspectives using a broadscale, genetically informed

approach. It cultivates a clearer understanding of the SES–health gradient and postulates a new theory of the mechanism by which socioeconomic status impacts on health.

## Chapter 2: Dissertation Study Aims

Using contemporary samples of adolescent and adult American twin pairs raised in the same household (the National Longitudinal Study of Adolescent Health and the Washington State Twin Registry) and sophisticated quantitative genetic models (described in detail in Chapter 3), the proposed dissertation research takes a comprehensive approach to understanding the socioeconomic status–health gradient, in terms of both family- and individual-level measures of SES (*i.e.*, compositional SES measures) and community-level measures of SES or income inequality (*i.e.*, contextual SES measures). Accordingly, this dissertation has two specific aims:

1) **Determine the pathways through which socioeconomic status acts on level of health.** Consistent with prior research, we expected lower SES to predict poorer mental and physical health, greater participation in negative health behaviors, and less participation in positive health behaviors at the population level. Also informed by existing research, we predicted that these effects will be non-causal, and instead will act through shared environmental or (more likely) genetic pathways common to both phenotypes. We did not expect this pattern to differ according to compositional versus contextual measures of SES (although the little genetically informed research that exists on the effects of contextual SES on health and health behaviors suggests that contextual SES may operate through nonshared environmental pathways on health behaviors; see Watson et al., 2015).

2) **Examine whether socioeconomic status influences variance in health.** Informed by prior research and the stress-diathesis model, we expected variance in mental health, physical health, and negative health behaviors to decrease, and variance in

positive health behaviors to increase, as a function of increasing SES. We also hypothesized that SES will be acting primarily on genetic variance, potentiating genetic vulnerabilities for negative health outcomes or suppressing genetic expression of adaptive characteristics that may serve to combat negative health outcomes.

The samples, measures, and statistical methods we used in this dissertation research are detailed in Chapter 3. We make explicit throughout the dissertation which methods correspond to each study aim. We also present preliminary results in Chapter 4.

# Chapter 3: Analytic Strategy

## Samples

***National Longitudinal Study of Adolescent Health (Add Health).*** The National Longitudinal Study of Adolescent Health (Add Health; Harris, 2009) is a nationally representative sample of young adults in the United States who have been followed since adolescence. There are currently four complete waves of data, collected between 1994 and 2009; details of data collection and survey procedures are described elsewhere (Harris et al., 2009). The present study uses data collected from 2,085 sibling pairs (49.8% male, 50.2% female) from the Add Health genetic subsample (described below). These members were interviewed during Wave I (between April and December, 1995), Wave II (between April and August, 1996), Wave III (between August, 2001 and April, 2002), and Wave IV (between January, 2008 and February, 2009)[15]. This subsample is diverse, with 50.8% identifying as non-Hispanic white, 22.3% as African American, 6.2% as Asian American, and 20.7% as bi- or multiracial or other ethnicity. The mean age of respondents at Wave I was 16.10 years (*SD* = 1.70, *Range* = 11.58–21.25) and 28.92 years at Wave IV (*SD* = 1.73, *Range* = 24.42–34.33).

The Add Health genetic sample includes participants identified as members of monozygotic twin pairs (MZ; *n* = 255 pairs; 123 male pairs, 132 female pairs) and dizygotic twin pairs (DZ; *n* = 319 pairs; 120 male-male pairs, 199 female-female pairs). Zygosity was determined using four self-report items that are standard nonserological determinates of zygosity and have high validity when compared with DNA-determined

[15] Except where noted, we will use data collected during Wave I. Three outcomes of interest (neuroticism and immunological markers) were assessed during Wave III (retrospective ADHD) or Wave IV only (neuroticism and immunological markers).

zygosity (Loehlin & Nichols, 1976; Spitz et al., 1996). Pairs with undetermined zygosity were excluded from analyses.

***Washington State Twin Registry (WSTR).*** The Washington State Twin Registry (WSTR) is a 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 a survey with items on sociodemographics, health, and lifestyle behaviors. Standard questions about childhood similarity that determine zygosity with greater than 90% accuracy when compared with DNA-based methods were used to classify twins as identical (monozygotic; MZ) or fraternal (dizygotic; DZ) (Eisen et al., 1989; Spitz et al., 1996; Torgersen, 1979). Prior to 2008, twins' residential street addresses (used to identify census tract) were not available; therefore, we have area deprivation and income inequality information for a subsample of the total WSTR sample described above (1,089 MZ female pairs; 467 DZ female pairs; 534 MZ male pairs; 173 DZ male pairs). Overall, the sample was young (39.4 ± 17.6 years; range = 18–96; 25$^{th}$ percentile = 24.2; 75$^{th}$ percentile = 53.2), well-educated (93% with a high school degree and 40% with a Bachelor's degree or higher), and predominantly white (85% white, 2% African-American, 3% Asian, 1% Pacific Islander, 1% Hispanic, 1% Native American, 7% multi-ethnic).

**Measures**

***Socioeconomic status.*** Several indicators of individual-level, neighborhood-level, and family-of-origin-level socioeconomic status and income inequality were used in the dissertation analyses. Each is described in detail below according to the sample to which it belongs.

**Educational Attainment (Add Health).** At Add Health Wave I (1995), the female head of household (typically) indicated the highest level of education she and her partner attained to provide a measure of family-of-origin-level educational attainment: 0 = *never went to school*; 1 = *8th grade or less*; 2 = *did not graduate high school or attended a business/trade/vocational school instead of high school*; 3 = *high school graduate or GED*; 4 = *some college or business/trade/vocational school*; 5 = *graduated from a college or university*; 6 = *professional training beyond a 4-year college or university*. In instances where parents or guardians had discrepant education levels, the highest education level between the individuals was used as the measure of parental education. The median education level attained by parents in the Add Health sample was some college (1st quartile = high school graduate, 3rd quartile = college degree); 31% of households had a highest-educated parent who had earned at least a Bachelor's degree, and just 14% of households had a highest-educated parent who did not graduate high school.

**Educational Attainment (WSTR).** WSTR respondents indicated their highest achieved level of education to provide an individual-level measure of educational attainment: 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*; 7 = *Bachelor's Degree*; 8 = *graduate or professional degree*. The WSTR sample is highly educated; the median education level attained was an Associate's degree (1st quartile = some college, 3rd quartile = bachelor's degree), with 46% of the sample earning a Bachelor's Degree or higher.

**Household Income (Add Health).** At Add Health Wave I (1995), the female

head of household (typically) indicated the total 1994 pretax household income (range = $0 to $999K) to provide a measure of family-of-origin-level earnings. The median household income was $36K (1st quartile = $20K, 3rd quartile = $55K).

**Household Income (WSTR).** WSTR respondents reported their current total household income using the following scale to provide a measure of individual-level earnings: 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 $50K–60K (1st quartile = $30–40K, 3rd quartile ≥ $80K).

**Neighborhood-Level Socioeconomic Advantage (Add Health).** To obtain a measure of neighborhood-level socioeconomic advantage in Add Health, we created a deprivation index similar to that used by other researchers (see the description of the Area Deprivation Index in the following subsection). We used factor scores generated from maximum likelihood factor analysis using varimax rotation of census tract data (from the 1990 U.S. Census) on 15 indicators of SES (percentage of the population with fewer than 9 years and with 12 or more years of education, percentage of persons employed in white collar occupations, log of median family income, income disparity [log of 100 times the ration of households with <$15,000 income to number of households with ≥$75,000 income], log of median home value, log of median gross rent, log of median monthly mortgage, home ownership rate, unemployment rate, family poverty rate, percentage of the population below 185% of the poverty rate, single-parent household rate, percentage of households with a telephone, and log of percentage of households without complete plumbing). A similar process for creating deprivation scores has been used elsewhere

(*e.g.*, Ford & Dzewaltowski, 2011). Higher factor scores correspond to greater socioeconomic advantage (*i.e.*, higher SES).

**Neighborhood-Level Socioeconomic Advantage (WSTR).** The Area Deprivation Index (ADI; Singh, 2003; Kind et al., 2014; University of Wisconsin-Madison, School of Medicine and Public Health, Health and Innovation Program, 2014) was used as a measure of neighborhood-level socioeconomic advantage in the WSTR[16]. Census tract data (from the 2000 U.S. Census) on 17 indicators of SES (percentage of the population with fewer than 9 years and with 12 or more years of education, percentage of persons employed in white collar occupations, median family income, income disparity [log of 100 times the ration of households with <$10,000 income to number of households with ≥$50,000 income], median home value, median gross rent, median monthly mortgage, home ownership rate, unemployment rate, family poverty rate, percentage of the population below 150% of the poverty rate, single-parent household rate, percentage of households without a motor vehicle, percentage of households without a telephone, log of percentage of households without complete plumbing, and household crowding) for twins' residential area were weighted (by 0.0849, –0.0970, –0.0874, –0.0977, 0.0936, –0.0688, –0.0781, –0.0770, 0.0615, 0.0806, 0.0977, 0.1037, 0.0719, 0.0694, 0.0877, 0.0510, and 0.0556, respectively) and summed to generate the ADI score. Scores for all census tracts nationwide were standardized by arbitrarily setting the mean to 100 and the standard deviation to 20. Block identification numbers were not available for all twins; therefore, a mean ADI score for each census-tract was used as the measure

[16] It is important to note that, although this is a neighborhood-level indicator of SES, this indicator is an individual-level predictor of level and variance in health and health behaviors (*i.e.*, is not necessarily shared between members of a twin pair).

of twins' neighborhood-level socioeconomic advantage. Traditionally, higher scores on the ADI indicate greater socioeconomic deprivation (*i.e.*, lower SES) relative to U.S. averages, but in order to keep our results consistent across analyses, we opted to rescale the ADI to make higher scores correspond to higher SES (rescale function = $-1 * \text{ADI} + 2 * \overline{\text{ADI}}$).

**County-Level Income Inequality (WSTR).** The Gini coefficient was used to measure county-level income inequality in the WSTR. The Gini coefficient is derived from the Lorenz curve, which plots the cumulative proportion of income earned by the population as a function of the cumulative proportion of the population. The Gini coefficient ranges between zero (perfect income equality) and 1.0 (perfect inequality; Cowell, 1977), and is the most commonly used measure of income inequality (Blakely, Lochner, & Kawachi, 2002). The Gini coefficient used in this dissertation is based on 2000 U.S. Census data (Arizona State University, College of Liberal Arts and Sciences, GeoDa Center for Geospatial Analysis and Computation, 2014).

***Mental Health.*** Several indicators of mental health were used in the dissertation analyses. Each is described in detail below according to the sample to which it belongs.

**Internalizing Behavior (Add Health).** We measured internalizing in the Add Health sample using a depressive symptom count, which was assessed using 19 items from the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977). The original 20-item CES-D Scale is a valid measure of the frequency of depressive symptoms in young adult samples (Radloff, 1991). Respondents indicated the extent to which they experienced a cluster of depressive symptoms (*bothered by things*; *appetite*

*was poor*; *could not shake off blues*; *felt just as good as others*; *trouble keeping mind on things*; *felt depressed*; *too tired to do things*; *felt hopeful about the future*; *thought life had been a failure*; *enjoyed life*; *felt tearful*; *felt happy*; *talked less than usual*; *felt lonely*; *people were unfriendly to you*; *felt sad*; *people disliked me*; *hard to get started doing things*; and *felt life was not worth living*) during the past seven days: 0 = *never or rarely*; 1 = *sometimes*; 2 = *a lot of the time*; 3 = *most of the time or all of the time*. Cronbach's alpha and McDonald's omega (McDonald, 1999) for these items demonstrated adequate internal consistency and general factor saturation ($\alpha$ = 0.87, $\omega$ = 0.85). We created a latent construct composed of these items for the dissertation analyses.

**Internalizing Behavior (WSTR).** Internalizing in the WSTR was operationalized as experiencing symptoms of depression and anxiety. Depression was measured using three items from the Patient Health Questionnaire-9 (PHQ-9; Spitzer, Kroenke, & Williams, 1999). The original 9-item PHQ-9 Scale is a valid measure of depressive symptom severity in adult samples (Kroenke, Spitzer, & Williams, 2001). Respondents indicated how often they experienced a cluster of depressive symptoms (*little interest or pleasure in doing things; feeling down, depressed, hopeless; and feeling tired or having little energy*) during the past 4 weeks: 0 = *not at all*; 1 = *several days*; 2 = *more than half the days*; 3 = *nearly every day*. Anxiety was measured using six items from the Brief Symptom Inventory (BSI; Derogatis & Melisaratos, 1983). The anxiety subscale of the BSI is a valid measure of symptoms of anxiety in adults (Derogatis & Melisaratos, 1983). Respondents indicated the extent to which they experienced symptoms related to anxiety (*nervousness or shakiness inside; suddenly scared for no reason; feeling fearful; feeling tense or keyed up; spells of terror or panic; feeling so*

*restless you could not sit still*) during the past 4 weeks: 0 = *not at all*; 1 = *a little bit*; 2 = *moderately*; 3 = *quite a bit*; 4 = *extremely*.

The three PHQ-9 items demonstrated adequate internal reliability ($\alpha$ = 0.81) and high general factor saturation ($\omega$ = 0.89), suggesting that a creating a latent depression factor from these items is appropriate. Similarly, the six BSI items had good internal validity ($\alpha$ = 0.87) and adequate general factor saturation ($\omega$ = 0.88), supporting the creation of a latent anxiety factor comprising these items. Depression and anxiety tend to be highly correlated with one another, and researchers have proposed a tripartite model of negative affect (Clark & Watson, 1991); that is, anxiety and depression are widely accepted as indicators of a general level of affective distress, yet simultaneously represent two dimensions of negative affect that are characterized by different symptoms clusters. Consistent with this model, mean scores of the depression items and the anxiety items were correlated $r$ = 0.59 ($p < 0.001$) and all depression and anxiety items together showed adequate general factor saturation ($\omega$ = 0.81), suggesting the presence of a general internalizing factor. This led us to conduct a confirmatory factor analysis (CFA) in which all depression and anxiety items load onto a general internalizing factor as well as specific depression and anxiety factors (see Figure 3.1). This model fit the data well (RMSEA = 0.039, 95% CI = 0.033 to 0.046; CFI = 0.993, TLI = 0.986), and serves as the baseline model for WSTR internalizing behavior in our primary analyses.

**Neuroticism (Add Health).** Neuroticism was measured in the Add Health sample using 12 items from the International Personality Item Pool (IPIP; Goldberg, 1999; Goldberg et al., 2006). The IPIP is a public domain personality inventory that measure lower-level facets of the Five-Factor Model of personality (Costa & McCrae,

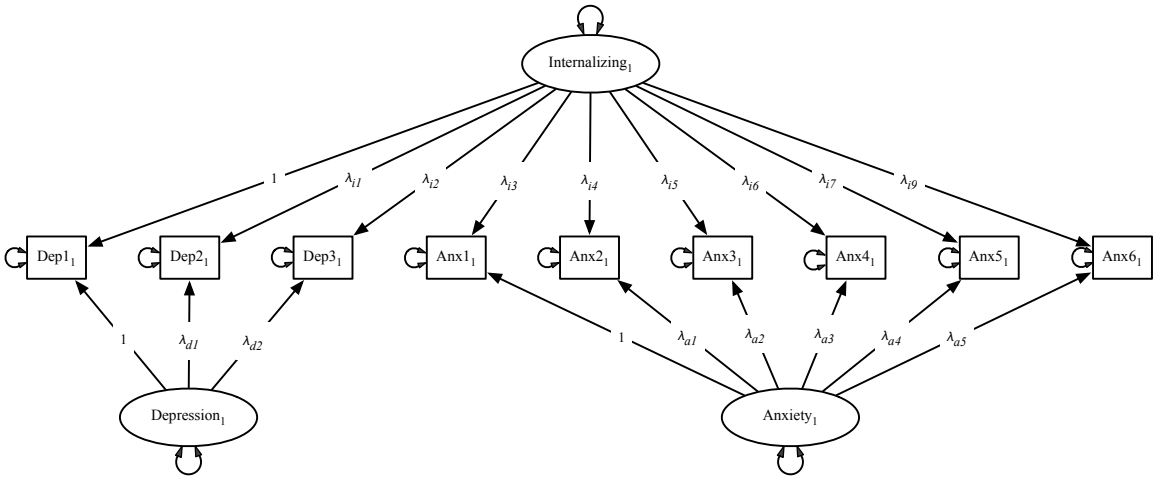


*Figure 3.1.* Path diagram of the CFA model fit to internalizing in the WSTR. All items load onto a general internalizing factor, items measuring depressive symptoms load onto a specific factor representing depression, and items measuring anxiety symptoms load onto a specific factor representing anxiety.

1992). At Wave IV, respondents indicated the extent to which they agreed with a number of statements about their personality and behavior (neuroticism items included *I have frequent mood swings; I worry about things; I get angry easily; I am relaxed most of the time; I am not easily bothered by things; I rarely get irritated; I get upset easily; I get stressed out easily; I lose my temper; I seldom feel blue; I don't worry about things that have already happened; I keep my cool*): 5 = *strongly agree*; 4 = *agree*; 3 = *neither agree nor disagree*; 2 = *disagree*; 1 = *strongly disagree*. These items demonstrated adequate internal reliability ($\alpha$ = 0.85) and adequate general factor saturation ($\omega$ = 0.81). Therefore, we used a latent factor to represent neuroticism in the dissertation analyses.

**Neuroticism (WSTR).** We measured neuroticism in the WSTR using 9 items from the IPIP (Goldberg, 1999; Goldberg et al., 2006). Respondents indicated how accurately a number of statements about personality and behavior described him- or herself (neuroticism items included *I get caught up in my problems; I am relaxed most of*

*the time; I get upset easily; I have frequent mood swings; I seldom feel blue; I am not embarrassed easily; I remain calm under pressure; I don't know why I do some of the things I do; I easily resist temptations*): 1 = *very inaccurate*; 2 = *moderately inaccurate*; 3 = *neither inaccurate nor accurate*; 4 = *moderately accurate*; 5 = *very accurate*. These items demonstrated adequate internal reliability ($\alpha$ = 0.77) and adequate general factor saturation ($\omega$ = 0.78). Therefore, we used a latent neuroticism factor to characterize this personality construct in the dissertation analyses.

**Externalizing (Add Health).** We used two measures to operationalize externalizing behavior in the Add Health sample: having symptoms characteristic of attention-deficit hyperactivity disorder (ADHD) and delinquent or antisocial behavior. At Wave III, Add Health participants retrospectively reported symptoms of ADHD experienced between the ages of 5 and 12 years using a DSM-IV (American Psychiatric Association, 2000) symptom checklist. Research suggests that ADHD follows a latent dimensional continuum rather than a categorical one (Lubke, Hudziak, Derks, van Bijsterveldt, & Boomsma, 2009), and that dimensional models of ADHD demonstrate stronger validity than categorical models of ADHD (Marcus & Barry, 2011). Thus, where possible, we treated ADHD as a continuum. In the Add Health sample, the 18 items comprising this ADHD scale covered the two primary domains of ADHD, inattentiveness and hyperactivity. The inattentiveness items demonstrated adequate internal reliability ($\alpha$ = 0.87) and adequate general factor saturation ($\omega$ = 0.86), as did the hyperactivity items ($\alpha$ = 0.81, $\omega$ = 0.86). Mean scores of the inattentiveness items and the hyperactivity items were correlated $r$ = 0.70 ($p < 0.001$) and all ADHD items together showed adequate general factor saturation ($\omega$ = 0.87), suggesting the presence of a

general ADHD factor. We then performed a CFA in which all inattentiveness and hyperactivity items loaded onto a general ADHD factor as well as specific inattentiveness and hyperactivity factors (similar to Figure 3.1). This model fit the data adequately (RMSEA = 0.058, 95% CI = 0.054 to 0.062; CFI = 0.934, TLI = 0.913), and serves as the baseline model for Add Health ADHD in our primary analyses.

Delinquent or antisocial behavior was assessed using 15 items tapping participation in delinquent or illegal behaviors during the 12 months prior to data collection: *vandalism; damage property; lie to parents about where or with whom the adolescent was; petty theft; physical fighting; hurting someone physically; running away from home; driving a car without the owner's permission; burglary; threaten another with a weapon; sell drugs; grand theft; gang fighting; buy/sell stolen goods; being unruly in public*. Respondents indicated the frequency with which they engaged in these behaviors: 0 = *never*; 1 = *1–2 times*; 2 = *3–4 times*; 3 = *5 or more times*. These items demonstrated adequate internal reliability ($\alpha$ = 0.87) and adequate general factor saturation ($\omega$ = 0.81). Therefore, we used a latent delinquency factor to characterize antisocial behavior in Add Health in the dissertation analyses.

**Externalizing (WSTR).** Externalizing behavior in the WSTR was operationalized as having ever been diagnosed with ADHD by a health care professional. Approximately 4.4% of respondents endorsed this item.

***Physical Health.*** Several indicators of physical health were used in the dissertation analyses. Each is described in detailed below according to the sample to which it belongs.

**General Health (Add Health).** Add Health respondents rated their general

health using the following scale: 5 = *excellent*; 4 = *very good*; 3 = *good*; 2 = *fair*; 1 = *poor*. This single-item measure of self-rated health is considered a "gold standard" of overall health and has been widely used in psychological research (Idler & Benyamini, 1997). It is established as a valid measure of mortality, and tends to be superior even to objective physician ratings in predicting mortality (Ferraro & Farmer, 1999; Idler & Benyamini, 1997).

**General Health (WSTR).** WSTR respondents rated their general health of the past four weeks using the following scale: 6 = *excellent*; 5 = *very good*; 4 = *good*; 3 = *fair*; 2 = *poor*; 1 = *very poor*. Overall, respondents rated their health as *good* to *very good* ($M = 4.86$, $SD = 0.88$).

**Health Conditions (WSTR).** WSTR respondents reported whether they had ever been diagnosed by a health care professional with 48 different medical conditions, including heart disease (*e.g.*, heart attack, angina, bypass surgery), hypercholesterolemia, hypertension, migraine headaches, and diabetes mellitus type 2. See Table 3.1 for a list of these health conditions and frequency of endorsement. We conducted a set of exploratory factor analyses, using scree plots and simple structure to determine the best-fitting solution and extracting factors using varimax rotation. Items with uniquenesses ≥ 90% or with no loadings ≥ 0.30 on any factor were eliminated, and this process was repeated until these criteria were met. The final solution included 15 items loading onto three factors. These items and their factor loadings are presented in Table 3.2.

Because we were most interested in overall health (as opposed to particular conditions), we also considered a general factor of health. Confirmatory factor analyses suggested that a Schmid-Leiman solution fit the data better (*RMSEA* = 0.040, 95%

Table 3.1. Health conditions assessed and endorsement rates in the WSTR.

| Health Condition | % Endorsing Diagnosis |
|---|---|
| Heart Disease* | 3.38 |
| High Cholesterol* | 20.21 |
| High Blood Pressure* | 17.65 |
| Type 2 Diabetes* | 4.06 |
| Hearing Loss* | 11.37 |
| Herniated Disc* | 7.82 |
| Arthritis* | 17.01 |
| Low Back Pain* | 30.26 |
| Bladder Infection* | 24.44 |
| Kidney Infection* | 5.92 |
| Chronic Tension Headaches* | 4.70 |
| Migraines* | 15.32 |
| Asthma* | 16.94 |
| Chronic Sinus Problems* | 7.83 |
| Seasonal Allergies* | 29.83 |
| Breast Cancer | 1.49 |
| Melanoma | 1.25 |
| Skin Cancer | 5.34 |
| Other Cancer | 3.45 |
| Stroke | 1.23 |
| Hypothyroidism | 7.46 |
| Type 1 Diabetes | 0.53 |
| Narcolepsy | 0.41 |
| Restless Leg Syndrome | 4.80 |
| Chronic Fatigue Syndrome | 1.54 |
| Meningitis | 1.04 |
| Multiple Sclerosis | 0.61 |
| Parkinson's Disease | 0.21 |
| Seizures | 2.45 |
| Bronchitis | 4.84 |
| COPD | 1.31 |
| Canker Sores | 15.85 |
| Cold Sores | 33.14 |
| GERD | 10.46 |
| Irritable Bowel Syndrome | 7.08 |
| Peptic Ulcer | 2.26 |
| Gum Disease | 8.95 |
| Temporomandibular Joint Disorder | 4.41 |
| Fibromyalgia | 2.34 |
| Lupus | 0.47 |
| Kidney Disease | 0.83 |
| Kidney Stones | 5.42 |
| Herpes | 5.08 |
| Shingles | 7.04 |
| Blood Clots | 1.87 |
| Drug Allergy | 21.04 |
| Mitral Valve Prolapse | 1.97 |
| Mononucleosis | 8.23 |

*Note*: Items included in final analysis marked with an asterisk (*).

confidence interval [CI] = 0.037 to 0.043) than both a hierarchical factor solution (*RMSEA* = 0.046, 95% CI = 0.043 to 0.049) and no general health factor rotation (*RMSEA* = 0.060, 95% CI = 0.058 to 0.063). Figure 3.2 shows the model fit to health conditions items.

Table 3.2. Factor loadings for final factor solution extracted from the 48 health conditions assessed in the WSTR.

| Health Condition | Factor 1: Obesity-Linked Health Conditions | Factor 2: Chronic Pain Conditions | Factor 3: Upper Airway Inflammatory Diseases | $u^2$ |
|---|---|---|---|---|
| Heart Disease | 0.35 | | | 0.88 |
| High Cholesterol | 0.62 | | | 0.61 |
| High Blood Pressure | 0.59 | | | 0.65 |
| Type 2 Diabetes | 0.38 | | | 0.85 |
| Hearing Loss | 0.34 | | | 0.88 |
| Herniated Disc | 0.41 | | | 0.79 |
| Arthritis | 0.49 | | | 0.72 |
| Low Back Pain | 0.42 | 0.33 | | 0.71 |
| Bladder Infection | | 0.45 | | 0.78 |
| Kidney Infection | | 0.37 | | 0.85 |
| Chronic Tension Headaches | | 0.45 | | 0.78 |
| Migraines | | 0.51 | | 0.73 |
| Asthma | | | 0.36 | 0.86 |
| Chronic Sinus Problems | | | 0.31 | 0.82 |
| Seasonal Allergies | | | 0.74 | 0.42 |

*Note:* Factor loadings below 0.30 are omitted from the table; $u^2$ is the item uniqueness.

**Body Mass Index (Add Health).** Self-reported height and weight was used to calculate body mass index (BMI; $kg/m^2$) at Wave I in the Add Health sample. Approximately 10.2% of the sample met criteria for being underweight (BMI < 18.5), 65.7% normal weight (BMI ranging from 18.5–24.9), 16.4% overweight (BMI ranging from 25–29.9), and 7.7% obese (BMI > 30).

**Body Mass Index (WSTR).** Self-reported height and weight was used to calculate BMI (in units of $kg/m^2$) in the WSTR. Approximately 2.5% of the sample met criteria for being underweight (BMI < 18.5), 48.1% normal weight (BMI ranging from 18.5–24.9), 30.6% overweight (BMI ranging from 25–29.9), and 18.8% obese (BMI > 30). Among 200 twin pairs from the WSTR, self-reported BMI was highly correlated with directly measured BMI ($r = 0.98$, $p < 0.01$), indicating a high degree of construct validity in this sample.

**Immune Functioning (Add Health).** At Wave IV, a subset of Add Health respondents provided capillary whole blood samples to provide measures of metabolic,

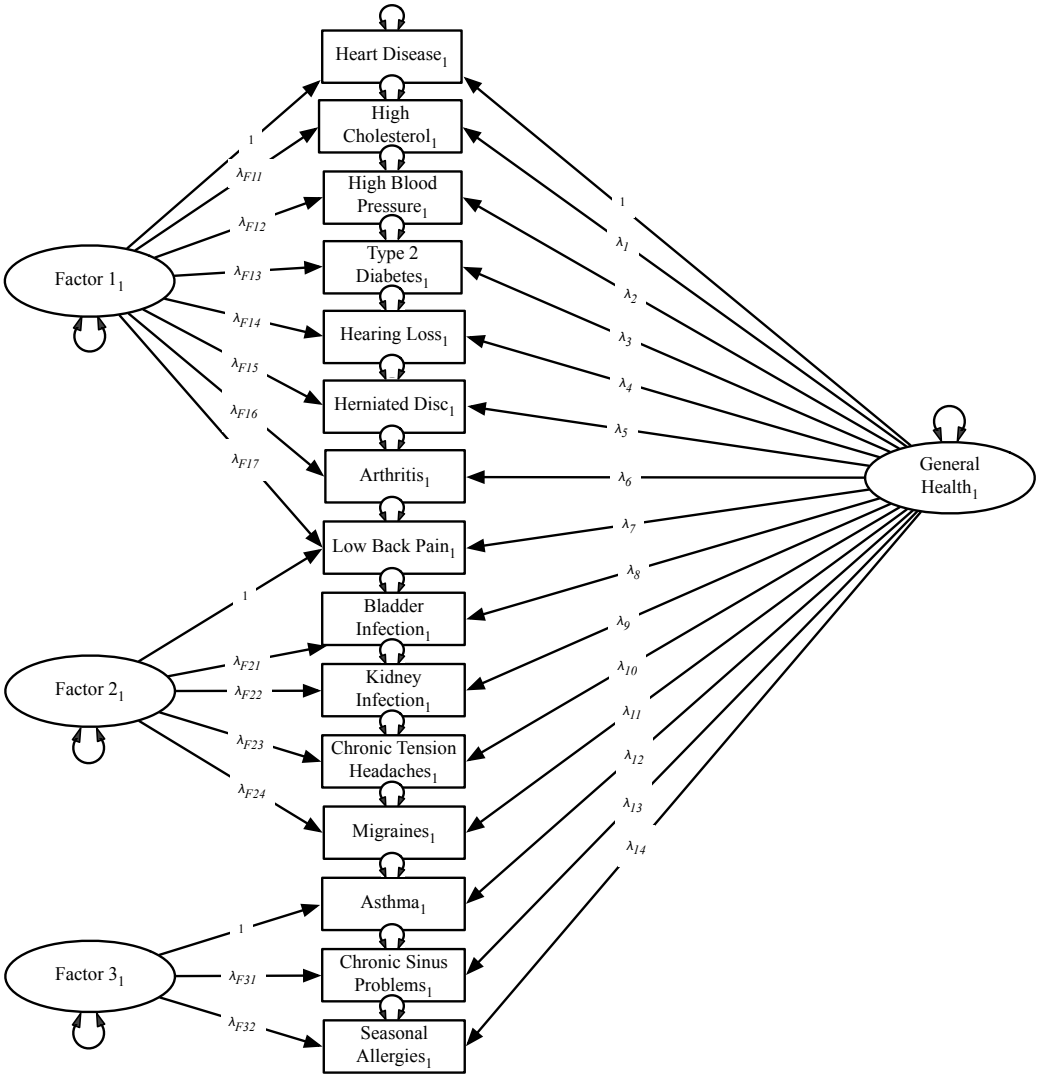


*Figure 3.2.* Path diagram of the CFA model fit to general health in the WSTR. All items load onto a general health factor and load onto a separate specific factor with other related health conditions.

inflammatory (high sensitivity C-reactive protein, or hsCRP, levels in units of mg/L), and immunological biomarkers (Epstein-Barr viral capsid immunoglobulin G, or EBV IgG, in units of AU/mL [antibody units per milliliter]). Details of biospecimen collection are described elsewhere (Whitsel et al., 2012). Each immunological biomarker was log-transformed prior to data analysis to approximate a normal distribution.

CRP is released by hepatic cells in the liver and is an acute-phase, non-specific inflammatory marker indicating compromised cell-mediated immunity (McDade et al., 2000; Stowe et al., 2007). Elevated CRP levels are related to bacterial infection, chronic inflammatory diseases, and physical trauma (Burtis, Ashwood & Bruns, 2006), and been implicated in increased risk for heart disease (see Ridker, 2003 for a review) and certain types of cancer (Allin & Nordestgaard, 2011). Elevated CRP levels have also been associated with depression (Howren, Lamkin, & Suls, 2009; Wium-Andersen, Ørsted, Nielsen, & Nordestgaard, 2013) and can be decreased by treatment with selective serotonin reuptake inhibitors (O'Brien, Scott, & Dinan, 2006).

Approximately 90% of adults worldwide are seropositive for the Epstein-Barr virus (Rickinson & Kieff, 2001), a herpesvirus that most commonly manifests itself as mononucleosis in 30-50% of immunocompetent individuals (Lennette, 1995; Steven, 1996). Stress compromises the immune system (Segerstrom & Miller, 2004), decreasing cell-mediated immunity and, subsequently, failure to inhibit reactivation of EBV. EBV reactivation triggers humoral-mediated immunity, including increased production of EBV IgG antibodies; that is, increased EBV IgG antibody titers represents poorer immune function (Glaser et al., 1991).

***Health Behavior.*** Several indicators of health behavior were used in the dissertation analyses. Each is described in detail below according to the sample to which it belongs.

**Substance Use (Add Health).** We used two measures to operationalize substance use in the Add Health sample: alcohol and cigarette use. A latent variable comprised of four drinking items was created to examine alcohol use. Respondents

indicated their frequency of drinking, heavy drinking (defined as 5 drinks or more in a row), and drunkenness during the 12 months prior to data collection: 1 = *never*; 2 = *once or twice*; 3 = *once a month or less*; 4 = *2 or 3 days a month*; 5 = *1 or 2 days a week*; 6 = *3 to 5 days a week*; 7 = *every day or almost every day*. Respondents also indicated how many drinks they typically consumed on each occasion. Cronbach's alpha and McDonald's omega for these items indicated adequate reliability ($\alpha = 0.92$, $\omega = 0.96$), so a latent alcohol use factor will be used in the dissertation analyses. Respondents also indicated how frequently they smoked during the past 30 days and how many cigarettes they typically smoked on each occasion. A single measure of smoking status (smoker, non-smoker) was created for the dissertation analyses. Approximately 25.1% of adolescents endorsed smoking at least once during the past 30 days.

**Substance Use (WSTR).** Substance use in the WSTR was operationalized as alcohol and cigarette use. Twins were asked how often they have a drink containing alcohol (0 = *never*; 1 = *monthly or less*; 2 = *2–4 times a month*; 3 = *2–3 times a week*; 4 = *4 or more times a week*), the number of alcoholic beverages typically consumed when drinking (0 = *don't drink alcohol*; 1 = *1 to 2*; 2 = *3 to 4*; 3 = *5 to 6*; 4 = *7 to 9*; 5 = *10 or more*) and the frequency with which they consume six or more drinks on one occasion (0 = *never*; 1 = *less than monthly*; 2 = *monthly*; 3 = *weekly*; 4 = *daily or almost daily*). These three alcohol use items (borrowed from the Alcohol Use Disorders Identification Test [AUDIT]; Babor, Higgins-Biddle, Saunders, & Monteiro, 2001) demonstrated adequate internal reliability ($\alpha = 0.79$) and strong general factor saturation ($\omega = 0.91$), so a latent alcohol use variable will be used in the dissertation analyses. Twins also indicated whether they currently smoked cigarettes. Approximately 11.0% of

respondents endorsed smoking.

**Exercise (Add Health).** Exercise in the Add Health sample was operationalized as times in the past week the individual participated in various physical activities: *roller-blading, roller-skating, skate-boarding, or bicycling; play an active sport, such as baseball, softball, basketball, soccer, swimming, or football;* and *exercise, such as jogging, walking, karate, jumping rope, gymnastics or dancing.* Response options included: 0 = *never*; 1 = *1–2 times*; 2 = *3–4 times*; 3 = *5 or more times*. These items demonstrated poor reliability ($\alpha = 0.41$) but adequate general factor saturation ($\omega = 0.94$), supporting the use of a latent exercise variable in the dissertation analyses.

Measures of sedentary behavior were also available for the Add Health sample. Adolescents indicated the number of hours per week they spend engaged in various behaviors associated with sedentary behavior: *watching television; watching videos; playing video or computer games.* These items (which were square root-transformed to correct for positive skew) demonstrated inadequate reliability ($\alpha = 0.57$) but adequate general factor saturation ($\omega = 0.86$), supporting the use of a latent sedentary behavior variable in the dissertation analyses.

**Exercise (WSTR).** We operationalized exercise in the WSTR as minutes per week of moderate-to-vigorous physical activity. Twins reported the number of days per week they engaged in vigorous physical activity for *at least 20 minutes* and moderate physical activity for *at least 30 minutes*. A single, continuous activity measure was constructed by summing moderate and vigorous physical activity days weighted by their respective durations. This measure provides an estimate that corresponds to activity levels recommended for health (*i.e.*, U.S. adults should engage in moderate-intensity

activity for ≥ 30 minutes per day on ≥ 5 days per week for a total of ≥ 150 minutes per week, vigorous-intensity activity for ≥ 20 minutes per day on ≥ 3 days per week for a total of ≥ 75 minutes per week, or a combination of moderate- and vigorous-intensity activity to achieve a total energy expenditure of ≥ 500-1,000 metabolic equivalent (MET) minutes per week) (Garber et al., 2011; U.S. Department of Health and Human Services, 1996). In a sample of 104 twins who wore accelerometers and GPS devices over a two-week period to collect objective measures of physical activity, this subjective measure of MVPA correlated moderately with objectively measured MVPA ($r = 0.46$, $p < 0.01$). There was a tendency for more active individuals to under-report MVPA to a greater extent than less active individuals ($r = –0.71$, $p < 0.01$; each minute increase in accelerometer-measured MVPA was associated with underreporting of MVPA by 0.64 minutes).

**Statistical Analyses**

Data were analyzed using maximum likelihood estimation in the structural equation modeling program Mplus (v. 7.0, Los Angeles, CA) (Muthen and Muthen, 2012) and the R (v. 3.1; R Core Team, 2014) structural equation modeling package OpenMx (Boker et al., 2011; Boker et al., 2014). Analyses were conducted using same-sex pairs and controlled for the linear effects of age and gender. Some covariates also predict residual variance in certain phenotypes (*e.g.*, age has been shown to modify biometric variance components of BMI; McCaffery et al., 2009); linear effects of covariates previously identified in the literature as moderators of variance in a phenotype were also controlled for in the prediction of residual variance in the phenotypes we examine in our analyses of the SES–health gradient.

***Genetic and Environmental Influences on Behavior.*** Although it is not the primary goal of our analyses, we will use the classical twin model (Neale & Maes, 2004) to decompose variation in health (and socioeconomic indicators) into three components: additive genetic (A), shared environmental (C), and nonshared environmental (E) variance. The A variance components, which represents the additive effect of an individual's genes, correlate between members of a sibling pair according to the proportion of segregating genes shared by that sibling dyad ($r$ = 1.0 between MZ twins, who share 100% of their genetic sequence; $r$ = 0.5 between DZ twins and full siblings, who share on average 50% of their segregating genes). The C variance components correlate at $r$ = 1.0 regardless of degree of genetic relatedness, because it represents environmental experiences that make members of the same family more similar than would be expected on the basis of genetics alone (*e.g.*, childhood rearing environment, attendance at the same schools, socioeconomic conditions of the family of origin). The E variance components, which represent environmental experiences unique to the individual, do not correlate between twins ($r$ = 0.0). It should be noted that E variance, in the absence of a measurement model, also includes measurement error.

The classical twin model is illustrated in the path diagram in Figure 3.3. Squares represent manifest, or measured, variables (*e.g.*, self-rated physical health of Twin 1 and Twin 2); the triangle represents a constant of one and is used to estimate the mean of the phenotype; circles represent latent, or unobserved, variables (in this case, the A, C, and E variance components of health or SES); double-headed arrows from one variable to itself represent variances (labeled $\sigma^2$ for each ACE component); double-headed arrows from one variable to another represent covariances; single-headed arrows represent causal

paths (*i.e.*, each ACE component's influence on health or SES). Heritability ($h^2$), the proportion of total variance in the phenotype accounted for by genetic factors, is calculated as:

$$h^2 = \frac{{\sigma_A}^2}{({\sigma_A}^2+{\sigma_C}^2+{\sigma_E}^2)} \quad (3.1)$$

There are several standard assumptions of the classical twin model (Neale & Maes, 2004). As noted above and evident in the path diagram in Figure 3.3, MZ twins are assumed to share 100% of their genes and DZ twins 50%. Related is the assumption that sibling dyads are products of random mating (*i.e.*, individuals are equally likely to choose partners to whom they are similar on a particular trait as they are partners from whom they differ; if assortative mating is occurring, this would create greater genetic similarity among non-MZ sibling dyads). Pair types are also assumed to experience equally similar environments (*i.e.*, MZ twins are not treated as more similar than DZ twins). Twins' or siblings' phenotypes are assumed to not have influence on one another (*i.e.*, each member of a sibling pair is equally likely to initiate, for example, smoking, regardless of the smoking behavior of the other twin). As shown in the path diagram, the classical twin model also assumes that genotypes and environmental influences are independent of one another; that is, genotype-environment correlation is not present (*i.e.*, the A component is not correlated with either the C or E components), nor is genotype × environment interaction (*i.e.*, the observed phenotypes are a linear function of the A, C, and E variance components). As we will see below, we can relax this last assumption to test for dependence of genotypic expression on environmental exposure (*i.e.*, G×E interaction).

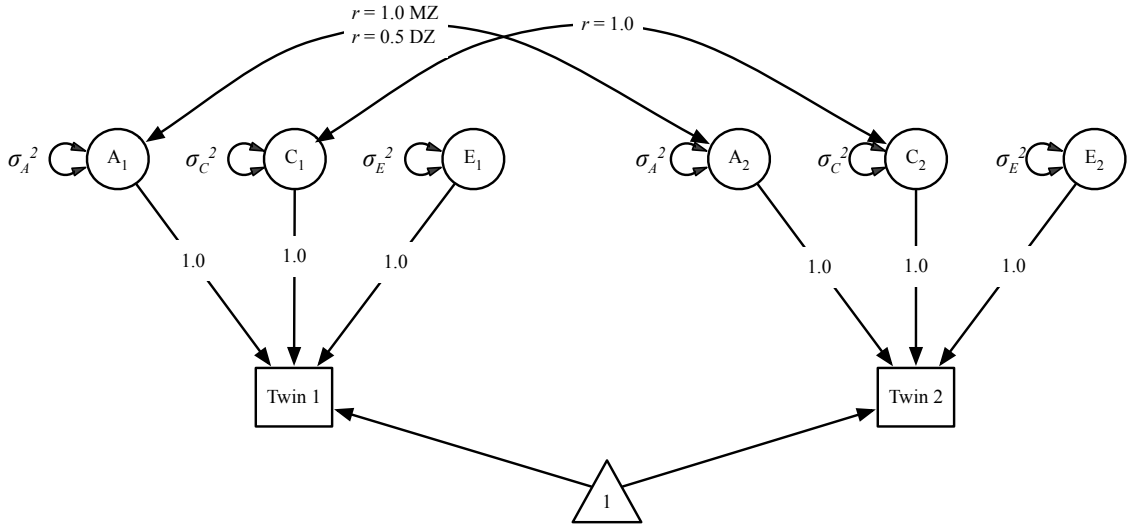


*Figure 3.3.* Path diagram of the classical twin model. The variances of the traits (*e.g.*, physical health) are decomposed into three components: additive genetic (A), shared environmental (C), and nonshared environmental (E) influences. The A components correlate at $r = 1.0$ for MZ twins and $r = 0.5$ for DZ twins. The C components correlate at $r = 1.0$ regardless of pair type. The E components do not correlate across twin pairs. Subscripts refer to twin 1 and twin 2.

***Causal Pathways vs. Gene-Environment Correlation (rGE): Effect of SES on Mean Levels of Health (Dissertation Study Aim 1).*** The association between socioeconomic status and physical or mental health and health behaviors within pairs of MZ and DZ twins raised in the same family provides the closest approximation of the causal effect of SES on health short of random assignment to socioeconomic advantage or disadvantage. Using the co-twin control design, we can assess this relationship within twin pairs, which controls for the effects of many measured and unmeasured confounds that vary between families, such as underlying genetic or environmental backgrounds that SES and health outcomes may share. That is, using twin pairs raised together allows us to decompose the main effect of SES on health into a genetic regression (or gene-

environment correlation, *r*GE) and a nonshared environmental regression[17], and this process allows us to make inferences about selection versus causal processes (Turkheimer & Harden, 2014).

A path diagram of the co-twin control design is illustrated in Figure 3.4. In this model, the outcome (health) is regressed on the A, C, and E terms of the predictor (SES). If socioeconomic status has a causal relationship with health, the effect should be observable both *between* twin pairs (pairs who are on average more socioeconomically advantaged are healthier) and *within* twin pairs (the pair member who is of higher socioeconomic status is healthier than his or her co-twin who is of lower SES). The within-pair association, which is the most valid measure of the causal effect of SES on health, is represented by the $b_E$ path in Figure 3.4. On the other hand, if the SES–health association is observed between families but not within them, a non-causal process that operates through shared genetic ($b_A$ in Figure 3.4) or common shared environmental ($b_C$ in Figure 3.4) pathways can be inferred.

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 co-twin control design, we choose to use the term *quasi-causal* to refer to causal pathways (c.f. Turkheimer & Harden, 2014).

**Note about Standardized vs. Unstandardized Regression Coefficients.** In behavior genetics research, Cholesky decomposition traditionally involves variables that

[17] Shared environmental factors (*e.g.*, poor healthcare during childhood, parental divorce) may also be inducing this correlation. We chose to use *r*GE in our example, however, because phenotypes in adulthood often do not contain variance attributable to shared environmental influences.

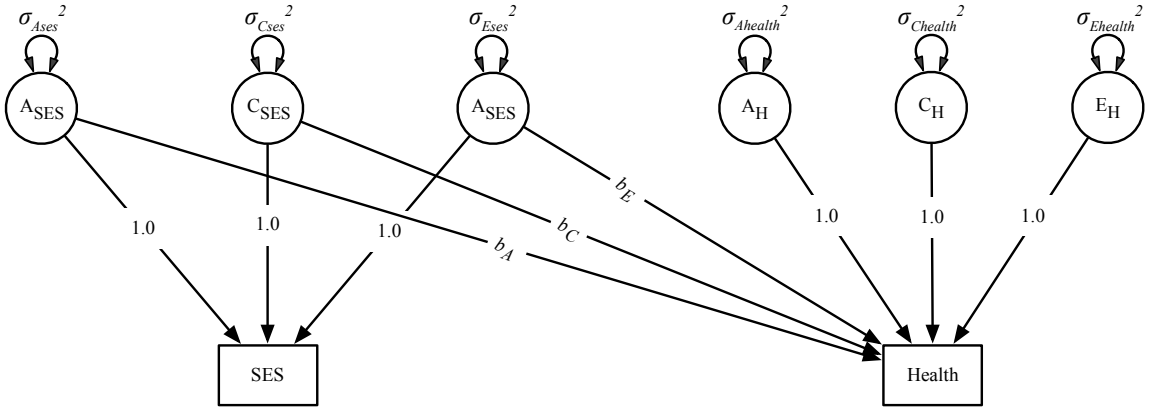


*Figure 3.4.* Path diagram of the co-twin control model (only one twin shown for clarity). The main effect of socioeconomic status on health is divided into a genetic regression ($b_A$), a shared environmental regression ($b_C$), and a nonshared environmental regression ($b_E$). The regression of health on the A and C components of SES (labeled $A_{SES}$, $C_{SES}$, and $E_{SES}$) represents the between-twin pair or population-level effect; the nonshared environmental regression represents the within-twin pair or causal effect of SES on health. Health has residual variance after accounting for the main effects of SES that can also be decomposed into ACE components (labeled $A_H$, $C_H$, and $E_H$). The A and C components of the SES and health correlate across twins ($r_A$ = 1.0 for monozygotic twins, 0.5 for dizygotic twins; $r_C$ = 1.0 for all pair types); the E components are not correlated across twins.

have been standardized. That is, the variances of the ACE components are constrained to equal 1.0, and the pathways from the latent ACE variables to the phenotypes are estimated. Although useful when estimating correlated factor models, the utility of standardizing ACE variance components is limited when evaluating causal pathways between an environmental exposure (*e.g.*, SES) and an associated outcome (*e.g.*, self-rated health). Because of standardization, the pathways from the ACE components of the SES to perceived health are not directly comparable to one another. Rather, they carry information about the increase in perceived health per standard deviation unit of the respective ACE variance component of SES. Because the raw variances of these ACE components are not necessarily equal to one another, the interpretability of these

estimates in is limited.

What we are generally interested in is the effect of *units of SES* on perceived health. A model that estimates *unstandardized regression coefficients*, equivalent to the co-twin control model described above, can answer this question. Unstandardized ACE regression coefficients have two distinct advantages. First, they are invariant to the magnitude of the variance of the predictor variable—that is, they can be compared directly to one another. Second, they carry information not only about the increase in perceived health per unit of SES, but also about the source of that increase (*e.g.*, genes, environment). For this reason, we presented and interpreted unstandardized parameter estimates throughout this dissertation.

***Genotype-By-Environment (G×E) Interaction: Effect of SES on Genetic and Environmental Etiology of Health (Dissertation Study Aim 2).*** Classical twin models use correlations of MZ and DZ twins to partition variance in outcome into additive components attributable to genetic and environmental variance. These models can be extended by dropping the assumption of additivity and testing for G×E interaction by permitting the magnitudes of genetic and environmental variances of health to vary as a function of a moderating variable such as socioeconomic status (Purcell, 2002):

$$\sigma^2_{\text{Health}} = (b_{0Au} + b_{1Au}Mod)^2 + (b_{0Cu} + b_{1Cu}Mod)^2 + (b_{0Eu} + b_{1Eu}Mod)^2 \qquad (3.2)$$

In this equation, the ACE variance components of health are expressed as linear functions of a moderating variable, $Mod$ (SES in this dissertation); the regression coefficients have the subscripts Au, Cu, or Eu, which indicate to which ACE variance component the regression coefficients correspond; the squares of the $b_0$ terms yield the values of the

ACE variances where $Mod = 0$; and the $b_1$ terms represent the rate of increase or decrease in a respective variance component as a function of $Mod$. Additional moderating variables can be included in Equation 3.2 as well (Horn, Turkheimer, Strachan, & Duncan, 2015; McCaffery et al., 2009).

For individual-level moderators that can differ between twins from the same family (such as twin-level socioeconomic indicators as opposed to family-of-origin SES during childhood), the correlation between genes and environment (*r*GE), or other forms of confounding (such as through the shared environment), 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).[18] The purpose of adjusting for *r*GE is not just statistical; important substantive interpretations can be made as well. As we alluded to above, it is possible, for example, that individuals genetically predisposed to better health select into environments (such as electing to live in cleaner, safer, or more affluent neighborhoods) or choose environmental experiences (such as accepting jobs that offer greater health insurance coverage or other health-promoting benefits) that further promote better health. On the other hand, individuals with a disposition toward being highly educated or holding a well-paying job may, for example, have a similar predisposition (operating through genetic pathways or through the family environment) toward engaging in healthier behaviors (such as exercising regularly or having yearly physicals or preventative screenings). Both of these scenarios can affect total genetic and environmental variance in health.

[18] To account for *r*GE when using family-level indicators of SES, the main effect of the SES indicator is simply partialled from each twin's health score (Purcell, 2002).

Indeed, the tendency of socioeconomically advantaged individuals to demonstrate a reduced genetic influence on mental or physical health may be due to (a) suppression of genetic propensity to experience poor health by SES (*i.e.*, G×E interaction), (b) lower genetic susceptibility to poor health in more wealthy or socioeconomically advantaged individuals (*i.e.*, *r*GE), or (c) a combination of the two. To account for *r*GE, the regressions of health on the ACE components of SES are also allowed to vary as a function of SES (*i.e.*, the effect that SES has on health can depend on level of SES)—this procedure accounts for changes in total ACE variances of health that are instead attributable to the main effects of SES on health being non-static across levels of SES. Equation 3.2 can be extended to account for *any* effects an environmental exposure may have on variance in health (Purcell, 2002; Johnson, 2007; Rathouz, van Hulle, Rodgers, Waldman, & Lahey, 2008; van der Sluis et al., 2012):

$$\sigma^2_{\text{Health}} = (b_{0A} + b_{1A}Mod)^2\sigma^2_{\text{AMod}} + (b_{0C} + b_{1C}Mod)^2\sigma^2_{\text{CMod}} + (b_{0E} + b_{1E}Mod)^2\sigma^2_{\text{EMod}}$$
$$+(b_{0Au} + b_{1Au}Mod)^2 + (b_{0Cu} + b_{1Cu}Mod)^2 + (b_{0Eu} + b_{1Eu}Mod)^2 \quad (3.3)$$

where the subscripts A, C, and E correspond to the regressions of health on the ACE components of $Mod$. It should be noted that the regression of health on the ACE components of the moderator (SES) correspond to the genetic and quasi-causal pathways described in the previous section. Figure 3.5 illustrates a path diagram of the G×E in the presence of *r*GE model we used in this dissertation (note that Rathouz et al. (2008) have proposed several other possible parameterizations of this model; we select this parameterization because it tests our particular set of research hypotheses).

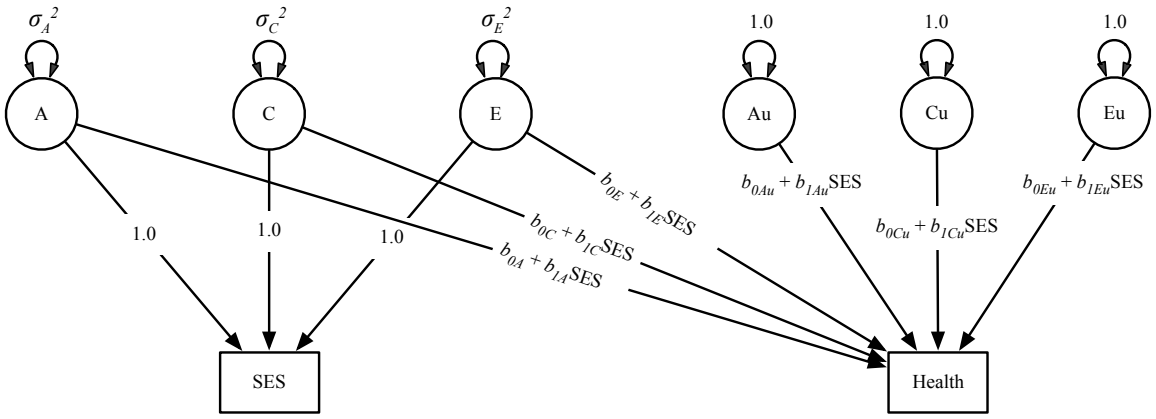


*Figure 3.5.* Path diagram of the G×E in the presence of *r*GE model (only one twin shown for clarity). The paths representing the ACE covariances between the environmental exposure (SES) and health vary as a function of the environmental exposure. Au, Cu, and Eu are the residual variance components of health (*i.e.*, the variance in health that remains after accounting for variance shared with SES), and are functions of SES. The A and C components of SES and health correlate across twins ($r_A$ = 1.0 for monozygotic twins, 0.5 for dizygotic twins; $r_C$ = 1.0 for all pair types); the E components are not correlated across twins.

**Note about Substantive Interpretations of G×E and rGE.** Johnson (2007) highlighted the importance of interpreting gene-by-environment interaction in the context of gene-environment correlation in order to make theory-driven inferences about social selection versus social causation processes. We have already alluded to these processes earlier in this section as well as in the section titled *Causal Pathways vs. Gene-Environment Correlation*, but we will make their definitions more explicit here. A *social selection* process is one in which individuals with preexisting genetic or shared environmental vulnerabilities (*e.g.*, toward poorer health) also select into (or create) more stressful environments. A *social causation* process is one in which poorer health is being triggered by adverse environmental conditions. Social causation can extend beyond influencing the mean level of health—social forces may enhance or restrict deleterious

expression of genotypes (or environments) that influence health as well[19]. Johnson (2007) stated that, in processes involving G×E in the presence of *r*GE, "the primary marker of a social causation process is that it involves moderation of genetic and/or environmental variance *unique* to the trait rather than genetic and/or environmental influences *common* to the trait of the social cause" (Johnson, 2007, p. 433).

The Johnson (2007) paper outlined a number of G×E scenarios, each within a different *r*GE context to explicate the particular process at hand. Johnson (2007) noted that genetically or environmentally influenced selection takes place where the respective A, C, or E correlation between phenotypes is high, and the magnitude of the A, C, or E variance in the outcome variable where this correlation is high characterizes the predominant selection process at hand. In particular, social selection predominates when the correlation between the environment (SES) and the outcome (health) and the magnitude of variance in health are highest at the same end of the SES range. On the other hand, social causation predominates when the correlation between SES and health is low where the magnitude of variance in health is greatest (or vice versa)—that is, something about SES is triggering the expression of genetic influences unique to health, not the genetic or environmental background that SES and health may share.

[19] Social forces may also enhance or restrict adaptive expression of genotypes or environments, but such processes are less likely to be occurring (Johnson, 2007).

# Chapter 4: Preliminary Results

## Biometric Decomposition of Phenotypes

***Add Health.*** Descriptive statistics, twin correlations, and genetic and environmental contributions for the socioeconomic predictors and health outcomes in the Add Health sample are presented in Table 4.1. In the Add Health sample, individual-level socioeconomic indicators showed moderate influence from the shared environment, particularly educational attainment. Each also showed substantial nonshared environmental influences, but variance in educational attainment contained little additive genetic variation. Most health outcomes showed minimal shared environmental influence. Mental health outcomes appeared to be substantially influenced by nonshared environmental factors but also showed moderate influence from genetic factors. Physical health outcomes showed a range of influence from genetic and nonshared environmental factors, again with nonshared environmental factors tending to account for a greater proportion of the phenotypic variation (with the exception of BMI). Health behaviors showed heavy influence from both genetic and nonshared environmental influences; negative health behaviors (*i.e.*, alcohol use and smoking status) showed greater shared environmental influences compared with positive health behaviors (*e.g.*, physical activity and sedentary behavior) and both physical and mental health outcomes. That each phenotype contains within- and between-family variance suggests the possibility of family-level confounds in the main effect of SES on health and health behaviors, and supports use of the co-twin control design to examine this association.

***WSTR.*** Descriptive statistics, twin correlations, and genetic and environmental contributions for the socioeconomic predictors and health outcomes in the WSTR are

Table 4.1. Descriptive statistics, twin correlations, and genetic and environmental proportions of variation in socioeconomic status, mental and physical health outcomes, and health behaviors in the Add Health sample.

| Phenotype | M | SD | 1st Q | 3rd Q | Range | *r*MZ | *r*DZ | $h^2$ | $c^2$ | $e^2$ |
|---|---|---|---|---|---|---|---|---|---|---|
| Socioeconomic Status | | | | | | | | | | |
| *Parental Education* | 3.88 | 1.30 | 3.00 | 5.00 | 0–6 | | | | | |
| *Family Income ($1K)* | 44.32 | 49.57 | 20.00 | 55.00 | 0–999 | | | | | |
| *Neighborhood SES* | 0.02 | 0.91 | –0.67 | 0.63 | –2.09–2.87 | | | | | |
| Mental Health | | | | | | | | | | |
| *Depression** | 11.68 | 7.76 | 6.00 | 16.00 | 0–57 | 0.38 | 0.33 | 0.11 (0.21) | 0.26 (0.18) | **0.63 (0.06)** |
| *Neuroticism** | 32.96 | 7.28 | 28.00 | 38.00 | 12–60 | 0.24 | 0.02 | **0.20 (0.08)** | 0.00 (0.00) | **0.80 (0.08)** |
| *ADHD** | 13.57 | 8.95 | 7.00 | 19.00 | 0–54 | 0.46 | 0.13 | **0.43 (0.08)** | 0.00 (0.00) | **0.57 (0.08)** |
| *Delinquency** | 4.43 | 5.58 | 1.00 | 6.00 | 0–45 | 0.44 | 0.61 | 0.00 (0.00) | **0.46 (0.05)** | **0.54 (0.05)** |
| Physical Health | | | | | | | | | | |
| *General Health* | 3.87 | 0.93 | 3.00 | 5.00 | 1–6 | 0.38 | 0.16 | **0.38 (0.05)** | 0.00 (0.00) | **0.62 (0.05)** |
| *Body Mass Index (kg/m$^2$)* | 22.99 | 4.56 | 19.95 | 24.94 | 13.72–65.03 | 0.81 | 0.40 | **0.81 (0.02)** | 0.00 (0.00) | **0.19 (0.02)** |
| *hsCRP (mg/L)†* | 0.88 | 1.69 | –0.22 | 1.73 | –2.50–6.91 | 0.09 | 0.03 | 0.08 (0.15) | 0.00 (0.04) | **0.92 (0.14)** |
| *EBV IgG (AU/mL)†* | 4.94 | 0.96 | 4.38 | 5.37 | 3.22–9.21 | | | | | |
| Health Behavior | | | | | | | | | | |
| *Alcohol Use Frequency** | 5.22 | 3.63 | 3.00 | 6.00 | 3–21 | 0.60 | 0.34 | **0.61 (0.05)** | 0.00 (0.01) | **0.38 (0.04)** |
| *Alcohol Use Amount* | 1.77 | 2.98 | 0.00 | 3.00 | 0–16 | | | | | |
| *Smoking§* | 25.1% | | | | | 0.81 | 0.48 | **0.66 (0.22, 0.90)** | 0.15 (0.00, 0.53) | **0.19 (0.10, 0.32)** |
| *Physical Activity** | 3.70 | 2.14 | 2.00 | 5.00 | 0–9 | 0.63 | 0.26 | **0.63 (0.10)** | 0.00 (0.00) | **0.37 (0.10)** |
| *Sedentary Behavior‡* | 2.11 | 1.08 | 1.33 | 2.67 | 0–9.69 | 0.60 | 0.05 | **0.54 (0.10)** | 0.00 (0.00) | **0.46 (0.10)** |

*Note:* Certain phenotypes are log-transformed (denoted with †) or square root-transformed (denoted with ‡). Phenotypes (denoted with §) are dichotomous and descriptive statistics are thus presented as the percentage of respondents endorsing the item A summed score was used for phenotypes marked *, although it should be noted that latent scores will be used in the dissertation analyses. Standard errors presented within parentheses; **bolded** estimates statistically significant at $p < 0.05$.

presented in Table 4.2. Although the socioeconomic indicators showed moderate influence from the shared environment, most health outcomes showed minimal shared environmental influence (this observation is consistent with genetically informed research suggesting that many clinical outcomes in adulthood are not influenced by family environmental factors; Plomin & Daniels, 1987). Mental health outcomes appeared to be predominantly influenced by nonshared environmental factors (all $e^2 \geq$ 55%) but also showed substantial influence from genetic factors. Physical health outcomes showed a range of influence from genetic and nonshared environmental factors, again with nonshared environmental factors tending to account for greater than 50% of the phenotypic variation. Negative health behaviors (*i.e.*, alcohol use and smoking status)

Table 4.2. Descriptive statistics, twin intraclass correlations, and genetic and environmental proportions of variation in socioeconomic status, mental and physical health outcomes, and health behaviors in the WSTR.

| Phenotype | M | SD | 1st Q | 3rd Q | Range | *r*MZ | *r*DZ | $h^2$ | $c^2$ | $e^2$ |
|---|---|---|---|---|---|---|---|---|---|---|
| Socioeconomic Status | | | | | | | | | | |
| *Education* | 6.47 | 1.9 | 5.00 | 7.00 | 2–8 | 0.72 | 0.53 | **0.39 (0.05)** | **0.33 (0.05)** | **0.28 (0.01)** |
| *Income* | 5.15 | 2.65 | 3.00 | 8.00 | 1–8 | 0.53 | 0.35 | **0.36 (0.07)** | **0.17 (0.06)** | **0.47 (0.02)** |
| *Area Deprivation Index* | 109.9 | 19.42 | 97.53 | 116.70 | 73.26–252.30 | 0.45 | 0.50 | 0.00 (0.02) | **0.46 (0.02)** | **0.54 (0.02)** |
| *Gini Index* | 0.43 | 0.03 | 0.41 | 0.45 | 0.33–0.59 | 0.37 | 0.32 | 0.10 (0.08) | **0.27 (0.07)** | **0.63 (0.02)** |
| Mental Health | | | | | | | | | | |
| *Internalizing** | 4.02 | 4.58 | 1.00 | 6.00 | 0–33 | 0.33 | 0.23 | **0.19 (0.09)** | 0.14 (0.08) | **0.67 (0.02)** |
| *Neuroticism** | 23.58 | 6.04 | 19.00 | 28.00 | 9–45 | 0.48 | 0.17 | **0.47 (0.02)** | 0.00 (0.00) | **0.53 (0.02)** |
| *ADHD*† | 4.41% | | | | | 0.77 | 0.50 | **0.56 (0.14, 0.85)** | **0.21 (0.00, 0.60)** | **0.22 (0.15, 0.32)** |
| Physical Health | | | | | | | | | | |
| *Self-Rated Health* | 4.86 | 0.88 | 1.00 | 3.00 | 1–6 | 0.40 | 0.23 | **0.34 (0.07)** | 0.06 (0.07) | **0.60 (0.02)** |
| *General Health (Factor)* | | | See Table 3.1 | | | 0.61 | 0.17 | **0.59 (0.03)** | 0.00 (0.00) | **0.41 (0.03)** |
| *Body Mass Index* | 25.98 | 5.63 | 22.04 | 28.48 | 13.73–71.91 | 0.77 | 0.43 | **0.69 (0.08)** | 0.08 (0.07) | **0.23 (0.01)** |
| Health Behavior | | | | | | | | | | |
| *Alcohol Use** | 2.89 | 2.68 | 0.00 | 5.00 | 0–13 | 0.61 | 0.40 | **0.42 (0.08)** | **0.19 (0.08)** | **0.39 (0.02)** |
| *Smoking*† | 11.04% | | | | | 0.76 | 0.46 | **0.62 (0.33, 0.81)** | **0.14 (0.00, 0.42)** | **0.24 (0.18, 0.30)** |
| *Physical Activity* | 112.58 | 89.66 | 30 | 170.00 | 0–350 | 0.32 | 0.16 | **0.32 (0.06)** | 0.00 (0.06) | **0.68 (0.02)** |

*Note:* Certain phenotypes (denoted with †) are dichotomous and descriptive statistics are thus presented as the percentage of respondents endorsing the item. Descriptive statistics for phenotypes denoted with * used a summed score, although it should be noted that latent scores will be used in the dissertation analyses. Standard errors presented within parentheses (95% confidence intervals for ADHD and Smoking phenotypes); **bolded** estimates statistically significant at $p < 0.05$.

were more strongly heritable compared with positive health behaviors (*e.g.*, physical activity), which showed greatest influence from the nonshared environment. That each phenotype contains within- and between-family variance suggests the possibility of family-level confounds in the main effect of SES on health and health behaviors, and supports use of the co-twin control design to examine this association.

**Phenotypic Correlations Among SES and Health Indicators**

***Add Health.*** Correlations between socioeconomic indicators and health outcomes and behaviors in the Add Health sample are presented in Table 4.3. We hypothesized that better socioeconomic conditions would be associated with lower symptomatology or presence of physical and mental health conditions, lower participation in negative health behaviors (*e.g.*, substance use), and increased participation in positive health behaviors

Table 4.3. Phenotypic associations between socioeconomic status and physical and mental health outcomes and health behavior in the Add Health sample.

| Outcome | Parental Education | Family Income | Neighborhood Socioeconomic Advantage |
|---|---|---|---|
| Mental Health | | | |
| *Depression** | **–.14** | **–.10** | –.09 |
| *Neuroticism** | **–.09** | –.06 | .00 |
| *ADHD** | –.06 | –.05 | .00 |
| *Delinquency** | .00 | .00 | –.02 |
| Physical Health | | | |
| *General Health* | **.12** | **.09** | .06 |
| *Body Mass Index (kg/m$^2$)* | **–.13** | **–.08** | **–.16** |
| *hsCRP (mg/L)†* | .01 | –.01 | –.01 |
| *EBV IgG (AU/mL)†* | .00 | –.03 | –.02 |
| Health Behavior | | | |
| *Alcohol Use Frequency** | .00 | .02 | .03 |
| *Alcohol Use Amount (drinks)* | .00 | .03 | .04 |
| *Smoking (cigarettes/day)* | .00 | –.01 | –.01 |
| *Physical Activity** | .05 | **.07** | **.13** |
| *Sedentary Behavior** | .00 | **–.11** | –.07 |

Note: **Bolded** coefficients are statistically significant with a Bonferroni correction ($p < .05/12$, or $p < .0042$). Certain phenotypes (denoted with †) are dichotomous; other phenotypes (denoted with *) are a based on a summed score, although it should be noted that latent scores are used in the primary dissertation analyses.

(*e.g.*, physical activity). We used a Bonferroni correction to account for multiple comparisons when testing for significance. In general, parental education and household income of the family of origin showed few statistically significant correlations with most outcomes; all that were significant were also in the expected direction. Neighborhood socioeconomic status was related primarily to BMI and exercise.

***WSTR.*** Correlations between SES indicators and health outcomes and behaviors are presented in Table 4.4. In general, education and income showed phenotypic correlations with most outcomes, all of which were in the expected direction with the exception of alcohol use. Area deprivation was associated primarily with mental health and health behaviors, with alcohol use again being in the opposite the direction hypothesized. Income inequality was associated with some outcomes and with health behaviors, and showed the same unexpected pattern with alcohol use that the other SES

Table 4.4. Phenotypic associations between socioeconomic status and physical and mental health outcomes and health behavior in the WSTR.

| Outcome | Education | Income | Area Deprivation Index | Gini Index |
|---|---|---|---|---|
| Mental Health | | | | |
| *Internalizing** | **–.11** | **–.19** | **–.08** | –.02 |
| *Neuroticism** | **–.12** | **–.14** | **–.06** | .00 |
| *ADHD*† | **–.08** | –.05 | .01 | –.03 |
| Physical Health | | | | |
| *General Health* | **.21** | **.22** | **.14** | **.07** |
| *Arthritis*† | **–.08** | –.02 | **–.06** | **–.08** |
| *Asthma*† | –.04 | **–.06** | .00 | .00 |
| *Heart Disease*† | **–.04** | **–.04** | –.04 | –.03 |
| *Hypercholesterolemia*† | –.01 | .04 | –.02 | **–.05** |
| *Hypertension*† | **–.06** | –.03 | –.05 | **–.09** |
| *Migraine Headaches*† | .01 | –.02 | –.04 | –.02 |
| *Type 2 Diabetes Mellitus*† | **–.09** | **–.07** | –.05 | –.04 |
| *Body Mass Index* | **–.12** | **–.07** | **–.17** | **–.11** |
| Health Behavior | | | | |
| *Alcohol Use** | **.09** | **.05** | **.09** | **.11** |
| *Smoking*† | **–.24** | **–.19** | **–.10** | **–.07** |
| *Moderate-to-Vigorous Physical Activity* | **.10** | **.07** | **.07** | **.05** |

Note: **Bolded** coefficients are statistically significant with a Bonferroni correction ($p < .05/15$, or $p < .0033$). Certain phenotypes (denoted with †) are dichotomous; other phenotypes (denoted with *) are a based on a summed score, although it should be noted that latent scores are used in the primary dissertation analyses.

indicators demonstrated. ADHD, asthma, high cholesterol, and migraines showed few to no significant associations with any of the SES indicators.

**Genetic and Environmental Influences on Health as a Function of SES**

***Add Health.*** To examine how genetic and environmental contributions to health outcomes vary as a function of SES, we identified twin pairs concordant for low SES (≤ bottom third quantile of SES indicator) and high SES (≥ top third quantile of SES indicator). Twin correlations and genetic and environmental contributions for each health outcome or behavior as a function of SES are presented in Table 4.5–Table 4.7. We hypothesized that lower socioeconomic conditions potentiate expression of variation (particularly additive genetic variation) in mental and physical health outcomes or negative health behaviors (*e.g.*, substance use), and restrict expression of positive health

behaviors (*e.g.*, physical activity). To provide context for heritability (or $h^2$), which is an expression of standardized variance (as is $c^2$ and $e^2$), we also present the phenotypic variance in each outcome as a function of high or low SES. For example, increasing $h^2$ in the context of decreasing overall variance as a function of SES may represent suppression, not amplification, of genetic variance by SES. As such, the preliminary results that follow should be used only to obtain an overall picture of the G×E process at hand. The dissertation analyses will use structural equation modeling and the twin models described in Chapter 3 to take a more nuanced approach to studying this phenomenon.

Overall, results for the Add Health sample are mixed and somewhat surprising based on prior research. The heritability of mental health appears to remain stable or increase with respect to parental education and income, respectively, while variance remains stable or decreases (we expected both heritability *and* variance to decrease with increasing SES). Physical health outcomes showed a similar pattern. No consistent patterns emerged for health behaviors. These inconsistent (and sometimes conflicting) results may be a result of the nature of the Add Health sample, which covers a wide developmental range (ages 11 to 20). Age differences in genetic and environmental influences may be masking any effect that SES has on variance in health. To address this issue, we controlled for the linear effects of age on variance in health in the primary dissertation analyses.

***WSTR.*** To examine how genetic and environmental contributions to health outcomes vary as a function of SES, we identified twin pairs concordant for low SES (≤ bottom third quantile of SES indicator) and high SES (≥ top third quantile of SES

Table 4.5. Twin correlations, genetic and environmental proportions of variation, and total variance of health outcomes as a function of low and high parental educational attainment in the Add Health sample.

| Outcome | Low (≤ High School Diploma) | | | | | | High (≥ Some College) | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *rMZ* | *rDZ* | $h^2$ | $c^2$ | $e^2$ | $\sigma^2$ | *rMZ* | *rDZ* | $h^2$ | $c^2$ | $e^2$ | $\sigma^2$ |
| Mental Health | | | | | | | | | | | | |
| *Depression** | 0.35 | 0.24 | 0.22 | 0.13 | 0.65 | 60.96 | 0.44 | 0.37 | 0.14 | 0.30 | 0.56 | 56.32 |
| *Neuroticism** | 0.25 | –0.02 | 0.25 | 0.00 | 0.75 | 50.98 | 0.32 | 0.00 | 0.32 | 0.00 | 0.68 | 54.44 |
| *ADHD** | 0.44 | 0.06 | 0.44 | 0.00 | 0.56 | 83.40 | 0.45 | 0.17 | 0.45 | 0.00 | 0.55 | 79.00 |
| *Delinquency** | 0.49‡ | 0.34‡ | 0.30 | 0.19 | 0.51 | 28.81 | 0.50‡ | 0.34‡ | 0.32 | 0.18 | 0.50 | 31.80 |
| Physical Health | | | | | | | | | | | | |
| *General Health* | 0.54 | 0.19 | 0.54 | 0.00 | 0.46 | 0.92 | 0.50 | 0.19 | 0.50 | 0.00 | 0.50 | 0.79 |
| *Body Mass Index (kg/m$^2$)* | 0.73 | 0.45 | 0.56 | 0.17 | 0.27 | 25.61 | 0.87 | 0.29 | 0.87 | 0.00 | 0.13 | 17.24 |
| *hsCRP (mg/L)†* | 0.31 | 0.10 | 0.31 | 0.00 | 0.69 | 2.73 | –0.03 | –0.01 | | | | 2.95 |
| *EBV IgG (AU/mL)†* | 0.34 | –0.01 | 0.34 | 0.00 | 0.66 | 0.97 | 0.45 | 0.08 | 0.45 | 0.00 | 0.55 | 0.90 |
| Health Behavior | | | | | | | | | | | | |
| *Alcohol Use Frequency** | 0.73 | 0.40 | 0.66 | 0.07 | 0.27 | 13.66 | 0.52 | 0.42 | 0.20 | 0.32 | 0.48 | 12.17 |
| *Alcohol Use Amount* | 0.58 | 0.44 | 0.28 | 0.30 | 0.42 | 9.26 | 0.47 | 0.35 | 0.24 | 0.23 | 0.53 | 8.79 |
| *Smoking†* | 0.78 | 0.64 | 0.28 | 0.50 | 0.22 | 1.00 | 0.60 | 0.45 | 0.30 | 0.30 | 0.40 | 1.00 |
| *Physical Activity** | 0.49 | 0.39 | 0.20 | 0.29 | 0.51 | 4.57 | 0.56 | 0.25 | 0.56 | 0.00 | 0.44 | 4.65 |
| *Sedentary Behavior** | 0.51 | 0.31 | 0.40 | 0.11 | 0.49 | 1.27 | 0.55 | 0.17 | 0.55 | 0.00 | 0.45 | 1.15 |

*Note:* Certain phenotypes (denoted with †) are dichotomous; other phenotypes (denoted with *) are a based on a summed score, although it should be noted that latent scores will be used in the dissertation analyses. The symbol ‡ indicates that correlations were derived using a rank transformation.

Table 4.6. Twin correlations, genetic and environmental proportions of variation, and total variance of health outcomes as a function of low and high family income in the Add Health sample.

| Outcome | Low (≤ $25K) | | | | | | High (≥ $50K) | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *rMZ* | *rDZ* | $h^2$ | $c^2$ | $e^2$ | $\sigma^2$ | *rMZ* | *rDZ* | $h^2$ | $c^2$ | $e^2$ | $\sigma^2$ |
| Mental Health | | | | | | | | | | | | |
| *Depression** | 0.44 | 0.36 | 0.16 | 0.28 | 0.56 | 64.59 | 0.42 | 0.11 | 0.42 | 0.00 | 0.58 | 53.62 |
| *Neuroticism** | 0.44 | 0.16 | 0.44 | 0.00 | 0.56 | 51.70 | 0.13 | 0.02 | 0.13 | 0.00 | 0.87 | 54.88 |
| *ADHD** | 0.29 | 0.26 | 0.06 | 0.23 | 0.71 | 82.65 | 0.46 | 0.16 | 0.46 | 0.00 | 0.54 | 76.57 |
| *Delinquency** | 0.54‡ | 0.49‡ | 0.10 | 0.44 | 0.46 | 38.83 | 0.51‡ | 0.33‡ | 0.36 | 0.15 | 0.49 | 26.66 |
| Physical Health | | | | | | | | | | | | |
| *General Health* | 0.58 | 0.18 | 0.58 | 0.00 | 0.42 | 0.95 | 0.43 | 0.01 | 0.43 | 0.00 | 0.57 | 0.75 |
| *Body Mass Index (kg/m$^2$)* | 0.84 | 0.37 | 0.84 | 0.00 | 0.16 | 22.72 | 0.83 | 0.36 | 0.83 | 0.00 | 0.17 | 17.75 |
| *hsCRP (mg/L)†* | 0.09 | –0.05 | 0.09 | 0.00 | 0.91 | 2.56 | –0.17 | 0.39 | | | | 2.97 |
| *EBV IgG (AU/mL)†* | 0.21 | 0.03 | 0.21 | 0.00 | 0.79 | 0.89 | 0.54 | 0.08 | 0.54 | 0.00 | 0.46 | 0.79 |
| Health Behavior | | | | | | | | | | | | |
| *Alcohol Use Frequency** | 0.68 | 0.33 | 0.68 | 0.00 | 0.32 | 13.92 | 0.66 | 0.37 | 0.58 | 0.08 | 0.34 | 12.48 |
| *Alcohol Use Amount* | 0.64 | 0.42 | 0.44 | 0.20 | 0.36 | 8.24 | 0.54 | 0.23 | 0.54 | 0.00 | 0.46 | 9.62 |
| *Smoking†* | 0.89 | 0.67 | 0.44 | 0.45 | 0.11 | 1.00 | 0.84 | 0.22 | 0.84 | 0.00 | 0.16 | 1.00 |
| *Physical Activity** | 0.58 | 0.44 | 0.28 | 0.30 | 0.42 | 9.26 | 0.47 | 0.35 | 0.24 | 0.23 | 0.53 | 8.79 |
| *Sedentary Behavior** | 0.57 | 0.56 | 0.02 | 0.55 | 0.43 | 1.44 | 0.61 | 0.19 | 0.61 | 0.00 | 0.39 | 1.01 |

*Note:* Certain phenotypes (denoted with †) are dichotomous; other phenotypes (denoted with *) are a based on a summed score, although it should be noted that latent scores will be used in the dissertation analyses. The symbol ‡ indicates that correlations were derived using a rank transformation.

Table 4.7. Twin correlations, genetic and environmental proportions of variation, and total variance of health outcomes as a function of low and high neighborhood socioeconomic advantage in the Add Health sample.

| Outcome | Low (≤ –0.24) | | | | | | High (≥ 0.47) | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *rMZ* | *rDZ* | $h^2$ | $c^2$ | $e^2$ | $\sigma^2$ | *rMZ* | *rDZ* | $h^2$ | $c^2$ | $e^2$ | $\sigma^2$ |
| Mental Health | | | | | | | | | | | | |
| *Depression** | 0.21 | 0.10 | 0.21 | 0.00 | 0.79 | 56.49 | 0.60 | 0.61 | 0.00 | 0.60 | 0.50 | 55.55 |
| *Neuroticism** | 0.19 | –0.10 | 0.39 | 0.00 | 0.61 | 57.52 | 0.31 | 0.13 | 0.31 | 0.00 | 0.69 | 49.30 |
| *ADHD** | 0.62 | -0.02 | 0.62 | 0.00 | 0.38 | 78.24 | 0.23 | 0.51 | 0.00 | 0.23 | 0.77 | 77.86 |
| *Delinquency** | 0.60‡ | 0.30‡ | 0.60 | 0.00 | 0.40 | 32.19 | 0.62‡ | 0.58‡ | 0.08 | 0.54 | 0.38 | 29.72 |
| Physical Health | | | | | | | | | | | | |
| *General Health* | 0.38 | 0.07 | 0.38 | 0.00 | 0.62 | 0.93 | 0.47 | 0.11 | 0.47 | 0.00 | 0.53 | 0.81 |
| *Body Mass Index (kg/m$^2$)* | 0.78 | 0.53 | 0.50 | 0.28 | 0.22 | 26.75 | 0.78 | 0.36 | 0.78 | 0.00 | 0.22 | 14.81 |
| *hsCRP (mg/L)*† | 0.09 | 0.17 | 0.00 | 0.09 | 0.91 | 2.81 | 0.11 | 0.16 | 0.00 | 0.11 | 0.89 | 3.26 |
| *EBV IgG (AU/mL)*† | 0.28 | 0.04 | 0.28 | 0.00 | 0.72 | 0.96 | 0.49 | 0.08 | 0.49 | 0.00 | 0.51 | 0.86 |
| Health Behavior | | | | | | | | | | | | |
| *Alcohol Use Frequency** | 0.74 | 0.42 | 0.64 | 0.10 | 0.26 | 12.73 | 0.78 | 0.41 | 0.74 | 0.04 | 0.22 | 11.93 |
| *Alcohol Use Amount* | 0.68 | 0.69 | 0.00 | 0.68 | 0.32 | 10.09 | 0.31 | 0.43 | 0.00 | 0.31 | 0.69 | 7.83 |
| *Smoking*† | 0.77 | 0.31 | 0.77 | 0.00 | 0.23 | 1.00 | 0.75 | 0.48 | 0.54 | 0.21 | 0.25 | 1.00 |
| *Physical Activity** | 0.51 | 0.33 | 0.36 | 0.15 | 0.49 | 4.30 | 0.58 | 0.42 | 0.32 | 0.26 | 0.42 | 4.65 |
| *Sedentary Behavior** | 0.62 | 0.43 | 0.38 | 0.24 | 0.38 | 1.56 | 0.44 | 0.10 | 0.44 | 0.00 | 0.56 | 1.04 |

*Note:* Certain phenotypes (denoted with †) are dichotomous; other phenotypes (denoted with *) are a based on a summed score, although it should be noted that latent scores will be used in the dissertation analyses. The symbol ‡ indicates that correlations were derived using a rank transformation.

indicator). Twin correlations and genetic and environmental contributions for each health outcome or behavior as a function of SES are presented in

Table **4.8**–Table 4.11. Overall, results are consistent with lower socioeconomic conditions potentiating expression of phenotypic variation in health outcomes and health behaviors (we expected restricted variation in positive health behaviors, but we did not observe this to be the case). Income inequality did not demonstrate this effect on total variance in health outcomes, but greater income inequality restricted variation in positive health outcomes. Genetic influences on mental health appeared to remain stable or increase slightly with increasing SES or income inequality (although note too that overall variance decreased with increasing SES, suggesting that genetic variance is also decreasing but at a slower rate than environmental variance), whereas heritability of health behaviors tended to decrease. Genetic influences on physical health conditions as

a function of SES showed a less consistent picture, increasing for some outcomes and/or SES indicators and decreasing for others.

Table 4.8. Twin correlations, genetic and environmental proportions of variation, and total variance of health outcomes as a function of low and high educational attainment in the WSTR.

| Outcome | Low (≤ Some College) | | | | | | High (≥ Bachelor's Degree) | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *rMZ* | *rDZ* | $h^2$ | $c^2$ | $e^2$ | $\sigma^2$ | *rMZ* | *rDZ* | $h^2$ | $c^2$ | $e^2$ | $\sigma^2$ |
| Mental Health | | | | | | | | | | | | |
| *Internalizing** | 0.32 | 0.24 | 0.16 | 0.16 | 0.68 | 25.17 | 0.32 | 0.18 | 0.28 | 0.04 | 0.68 | 15.52 |
| *Neuroticism** | 0.45 | 0.17 | 0.45 | 0.00 | 0.55 | 34.73 | 0.46 | 0.16 | 0.46 | 0.00 | 0.54 | 36.62 |
| *ADHD*† | 0.48 | 0.27 | 0.42 | 0.06 | 0.52 | 1.00 | 0.42 | 0.10 | 0.42 | 0.00 | 0.58 | 1.00 |
| Physical Health | | | | | | | | | | | | |
| *General Health* | 0.48 | 0.33 | 0.30 | 0.18 | 0.52 | 0.81 | 0.33 | 0.15 | 0.33 | 0.00 | 0.67 | 0.64 |
| *Arthritis*† | 0.47 | 0.39 | 0.16 | 0.31 | 0.53 | 1.00 | 0.36 | 0.23 | 0.26 | 0.10 | 0.64 | 1.00 |
| *Asthma*† | 0.37 | 0.14 | 0.37 | 0.00 | 0.63 | 1.00 | 0.34 | 0.20 | 0.28 | 0.06 | 0.66 | 1.00 |
| *Heart Disease*† | 0.39 | 0.06 | 0.39 | 0.00 | 0.61 | 1.00 | 0.35 | 0.23 | 0.24 | 0.11 | 0.65 | 1.00 |
| *Hypercholesterolemia*† | 0.58 | 0.34 | 0.48 | 0.10 | 0.42 | 1.00 | 0.47 | 0.30 | 0.34 | 0.13 | 0.53 | 1.00 |
| *Hypertension*† | 0.62 | 0.30 | 0.62 | 0.00 | 0.48 | 1.00 | 0.47 | 0.33 | 0.28 | 0.19 | 0.53 | 1.00 |
| *Migraine Headaches*† | 0.20 | 0.01 | 0.20 | 0.00 | 0.80 | 1.00 | 0.26 | –0.06 | 0.26 | 0.00 | 0.74 | 1.00 |
| *Type 2 Diabetes*† | 0.57 | 0.17 | 0.57 | 0.00 | 0.43 | 1.00 | 0.55 | 0.14 | 0.55 | 0.00 | 0.45 | 1.00 |
| *Body Mass Index* | 0.77 | 0.44 | 0.66 | 0.11 | 0.33 | 36.27 | 0.71 | 0.32 | 0.71 | 0.00 | 0.29 | 21.16 |
| Health Behavior | | | | | | | | | | | | |
| *Alcohol Use** | 0.62 | 0.33 | 0.38 | 0.04 | 0.38 | 8.40 | 0.59 | 0.35 | 0.48 | 0.11 | 0.41 | 5.23 |
| *Smoking*† | 0.55 | 0.20 | 0.55 | 0.00 | 0.45 | 1.00 | 0.30 | 0.08 | 0.30 | 0.00 | 0.70 | 1.00 |
| *Physical Activity* | 0.37 | 0.19 | 0.36 | 0.01 | 0.63 | 8511 | 0.34 | 0.14 | 0.34 | 0.00 | 0.66 | 7145 |

*Note:* Certain phenotypes (denoted with †) are dichotomous; other phenotypes (denoted with *) are a based on a summed score, although it should be noted that latent scores will be used in the dissertation analyses.

Table 4.9. Twin correlations, genetic and environmental proportions of variation, and total variance of health outcomes as a function of low and high income in the WSTR.

| Outcome | Low (≤ $40K–$50K) | | | | | | High (≥ $80K) | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *rMZ* | *rDZ* | $h^2$ | $c^2$ | $e^2$ | $\sigma^2$ | *rMZ* | *rDZ* | $h^2$ | $c^2$ | $e^2$ | $\sigma^2$ |
| Mental Health | | | | | | | | | | | | |
| *Internalizing** | 0.35 | 0.30 | 0.10 | 0.25 | 0.65 | 30.89 | 0.31 | 0.10 | 0.31 | 0.00 | 0.69 | 14.69 |
| *Neuroticism** | 0.40 | 0.19 | 0.40 | 0.00 | 0.60 | 37.58 | 0.51 | 0.12 | 0.51 | 0.00 | 0.49 | 37.17 |
| *ADHD*† | 0.37 | 0.21 | 0.32 | 0.05 | 0.63 | 1.00 | 0.34 | 0.26 | 0.16 | 0.18 | 0.66 | 1.00 |
| Physical Health | | | | | | | | | | | | |
| *General Health* | 0.42 | 0.27 | 0.30 | 0.12 | 0.58 | 0.80 | 0.33 | 0.25 | 0.16 | 0.17 | 0.67 | 0.61 |
| *Arthritis*† | 0.51 | 0.45 | 0.12 | 0.39 | 0.49 | 1.00 | 0.37 | 0.30 | 0.14 | 0.23 | 0.63 | 1.00 |
| *Asthma*† | 0.40 | 0.14 | 0.40 | 0.00 | 0.60 | 1.00 | 0.34 | 0.14 | 0.34 | 0.00 | 0.66 | 1.00 |
| *Heart Disease*† | 0.39 | 0.03 | 0.39 | 0.00 | 0.61 | 1.00 | 0.13 | 0.12 | 0.02 | 0.11 | 0.87 | 1.00 |
| *Hypercholesterolemia*† | 0.61 | 0.44 | 0.34 | 0.27 | 0.39 | 1.00 | 0.50 | 0.23 | 0.50 | 0.00 | 0.50 | 1.00 |
| *Hypertension*† | 0.52 | 0.32 | 0.40 | 0.12 | 0.48 | 1.00 | 0.54 | 0.27 | 0.54 | 0.00 | 0.46 | 1.00 |
| *Migraine Headaches*† | 0.28 | –0.03 | 0.28 | 0.00 | 0.72 | 1.00 | 0.35 | –0.10 | 0.35 | 0.00 | 0.65 | 1.00 |
| *Type 2 Diabetes* † | 0.45 | 0.28 | 0.34 | 0.11 | 0.55 | 1.00 | 0.52 | 0.21 | 0.52 | 0.00 | 0.48 | 1.00 |
| *Body Mass Index* | 0.75 | 0.48 | 0.54 | 0.21 | 0.25 | 38.31 | 0.72 | 0.37 | 0.70 | 0.02 | 0.28 | 20.97 |
| Health Behavior | | | | | | | | | | | | |
| *Alcohol Use** | 0.64 | 0.33 | 0.62 | 0.02 | 0.36 | 7.70 | 0.63 | 0.28 | 0.63 | 0.00 | 0.37 | 6.35 |
| *Smoking*† | 0.55 | 0.19 | 0.55 | 0.00 | 0.45 | 1.00 | 0.27 | 0.07 | 0.27 | 0.00 | 0.73 | 1.00 |
| *Physical Activity* | 0.31 | 0.13 | 0.31 | 0.00 | 0.69 | 8079 | 0.33 | 0.25 | 0.16 | 0.17 | 0.67 | 7273 |

*Note:* Certain phenotypes (denoted with †) are dichotomous; other phenotypes (denoted with *) are a based on a summed score, although it should be noted that latent scores will be used in the dissertation analyses.

Table 4.10. Twin correlations, genetic and environmental proportions of variation, and total variance of health outcomes as a function of low and high census tract-level socioeconomic advantage in the WSTR.

| Outcome | Low (≤ 100.22) | | | | | | High (≥ 111.97) | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *rMZ* | *rDZ* | $h^2$ | $c^2$ | $e^2$ | $\sigma^2$ | *rMZ* | *rDZ* | $h^2$ | $c^2$ | $e^2$ | $\sigma^2$ |
| Mental Health | | | | | | | | | | | | |
| *Internalizing** | 0.39 | 0.36 | 0.06 | 0.33 | 0.61 | 25.00 | 0.31 | 0.24 | 0.26 | 0.17 | 0.69 | 18.27 |
| *Neuroticism** | 0.41 | 0.18 | 0.41 | 0.00 | 0.59 | 38.26 | 0.47 | 0.13 | 0.47 | 0.00 | 0.53 | 33.65 |
| *ADHD*† | 0.58 | 0.11 | 0.58 | 0.00 | 0.42 | 1.00 | 0.51 | 0.23 | 0.51 | 0.00 | 0.49 | 1.00 |
| Physical Health | | | | | | | | | | | | |
| *General Health* | 0.49 | 0.29 | 0.40 | 0.09 | 0.51 | 0.82 | 0.46 | 0.17 | 0.46 | 0.00 | 0.54 | 0.67 |
| *Arthritis*† | 0.45 | 0.37 | 0.16 | 0.29 | 0.55 | 1.00 | 0.44 | 0.37 | 0.14 | 0.30 | 0.56 | 1.00 |
| *Asthma*† | 0.29 | 0.23 | 0.12 | 0.17 | 0.71 | 1.00 | 0.40 | 0.34 | 0.12 | 0.28 | 0.60 | 1.00 |
| *Heart Disease*† | 0.26 | 0.27 | 0.00 | 0.26 | 0.74 | 1.00 | –0.02 | –0.02 | 0.00 | 0.00 | 1.00 | 1.00 |
| *Hypercholesterolemia*† | 0.14 | 0.04 | 0.14 | 0.00 | 0.86 | 1.00 | 0.02 | –0.05 | 0.02 | 0.00 | 0.98 | 1.00 |
| *Hypertension*† | 0.47 | 0.25 | 0.44 | 0.03 | 0.53 | 1.00 | 0.59 | 0.29 | 0.59 | 0.00 | 0.41 | 1.00 |
| *Migraine Headaches*† | 0.25 | 0.05 | 0.25 | 0.00 | 0.75 | 1.00 | 0.11 | –0.03 | 0.11 | 0.00 | 0.89 | 1.00 |
| *Type 2 Diabetes* † | 0.59 | 0.12 | 0.59 | 0.00 | 0.41 | 1.00 | 0.35 | –0.02 | 0.35 | 0.00 | 0.65 | 1.00 |
| *Body Mass Index* | 0.70 | 0.44 | 0.52 | 0.18 | 0.30 | 48.36 | 0.77 | 0.40 | 0.74 | 0.03 | 0.23 | 17.98 |
| Health Behavior | | | | | | | | | | | | |
| *Alcohol Use** | 0.67 | 0.37 | 0.60 | 0.07 | 0.33 | 7.82 | 0.71 | 0.36 | 0.70 | 0.01 | 0.29 | 7.39 |
| *Smoking*† | 0.43 | 0.14 | 0.43 | 0.00 | 0.57 | 1.00 | 0.41 | 0.37 | 0.08 | 0.33 | 0.59 | 1.00 |
| *Physical Activity* | 0.47 | 0.13 | 0.47 | 0.00 | 0.53 | 8862 | 0.44 | 0.32 | 0.24 | 0.20 | 0.56 | 7369 |

*Note:* Certain phenotypes (denoted with †) are dichotomous; other phenotypes (denoted with *) are a based on a summed score, although it should be noted that latent scores will be used in the dissertation analyses.

Table 4.11. Twin correlations, genetic and environmental proportions of variation, and total variance of health outcomes as a function of low and high county-level income inequality in the WSTR.

| Outcome | Low (≤ 0.41) | | | | | | High (≥ 0.44) | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *rMZ* | *rDZ* | $h^2$ | $c^2$ | $e^2$ | $\sigma^2$ | *rMZ* | *rDZ* | $h^2$ | $c^2$ | $e^2$ | $\sigma^2$ |
| Mental Health | | | | | | | | | | | | |
| *Internalizing** | 0.26 | 0.35 | 0.00 | 0.26 | 0.74 | 21.82 | 0.40 | 0.25 | 0.30 | 0.10 | 0.60 | 20.92 |
| *Neuroticism** | 0.47 | 0.13 | 0.47 | 0.00 | 0.53 | 34.48 | 0.52 | 0.15 | 0.52 | 0.00 | 0.48 | 35.24 |
| *ADHD*† | 0.52 | 0.16 | 0.52 | 0.00 | 0.48 | 1.00 | 0.41 | 0.28 | 0.26 | 0.15 | 0.59 | 1.00 |
| Physical Health | | | | | | | | | | | | |
| *General Health* | 0.46 | 0.26 | 0.40 | 0.06 | 0.54 | 0.80 | 0.43 | 0.29 | 0.28 | 0.15 | 0.57 | 0.68 |
| *Arthritis*† | 0.50 | 0.35 | 0.30 | 0.20 | 0.50 | 1.00 | 0.48 | 0.30 | 0.36 | 0.12 | 0.52 | 1.00 |
| *Asthma*† | 0.34 | 0.17 | 0.34 | 0.00 | 0.66 | 1.00 | 0.37 | 0.36 | 0.02 | 0.35 | 0.63 | 1.00 |
| *Heart Disease*† | 0.21 | 0.13 | 0.16 | 0.05 | 0.79 | 1.00 | 0.14 | –0.01 | 0.14 | 0.00 | 0.86 | 1.00 |
| *Hypercholesterolemia*† | 0.54 | 0.31 | 0.46 | 0.08 | 0.46 | 1.00 | 0.47 | 0.33 | 0.28 | 0.19 | 0.53 | 1.00 |
| *Hypertension*† | 0.50 | 0.23 | 0.50 | 0.00 | 0.50 | 1.00 | 0.60 | 0.17 | 0.60 | 0.00 | 0.40 | 1.00 |
| *Migraine Headaches*† | 0.17 | 0.01 | 0.17 | 0.00 | 0.83 | 1.00 | 0.21 | –0.03 | 0.21 | 0.00 | 0.79 | 1.00 |
| *Type 2 Diabetes* † | 0.54 | –0.03 | 0.54 | 0.00 | 0.46 | 1.00 | --- | 0.66 | --- | --- | --- | 1.00 |
| *Body Mass Index* | 0.72 | 0.41 | 0.62 | 0.10 | 0.28 | 35.21 | 0.74 | 0.37 | 0.74 | 0.00 | 0.26 | 22.72 |
| Health Behavior | | | | | | | | | | | | |
| *Alcohol Use** | 0.65 | 0.22 | 0.65 | 0.00 | 0.35 | 6.83 | 0.68 | 0.41 | 0.54 | 0.14 | 0.32 | 7.25 |
| *Smoking*† | 0.57 | 0.15 | 0.57 | 0.00 | 0.43 | 1.00 | 0.49 | 0.11 | 0.49 | 0.00 | 0.51 | 1.00 |
| *Physical Activity* | 0.26 | 0.23 | 0.06 | 0.20 | 0.74 | 7961 | 0.40 | 0.15 | 0.40 | 0.00 | 0.60 | 8045 |

*Note:* Certain phenotypes (denoted with †) are dichotomous; other phenotypes (denoted with *) are a based on a summed score, although it should be noted that latent scores will be used in the dissertation analyses.

# Chapter 5: Results – Mental Health

The impact of socioeconomic status on mental health was evaluated using a variety of indicators tapping maladaptive psychological behaviors. These behaviors include internalizing (*e.g.*, depression, anxiety) and externalizing (*e.g.*, attention-deficit/hyperactivity disorder, delinquent activity) as well as personality traits (*i.e.*, neuroticism). We discuss the influence of socioeconomic status on each of these behaviors in turn.

## Internalizing Behavior

***Depression—Add Health.*** Internalizing in the Add Health sample was operationalized using 19 items from the CES-D (see Chapter 3). The model presented in Figure 5.1 was fit to the data separately for each socioeconomic status indicator, partialling for the linear effects of age and gender on level and variance in depression. We used likelihood ratio tests to determine the best fitting model. Biometric decomposition of both the SES indicators and the depression factor are presented in Table 4.1, and parameter estimates for each of these models are presented in Table 5.1. Family household income was log-transformed to correct for positive skew.

**Main Effects of SES on Depression (Dissertation Aim 1).** The main effects of SES on depression are presented in Table 5.1 under the heading *Main Effect of Moderator on Depression*. We discuss the main effects results for each moderator below.

**Parental Education.** Parental education was significantly associated with lower depression ($b = -0.050$, $p < 0.001$). Adolescents with a parent holding at least a college degree had depression scores that were 0.28 standard deviations lower than their counterparts whose parent holds a high school degree.

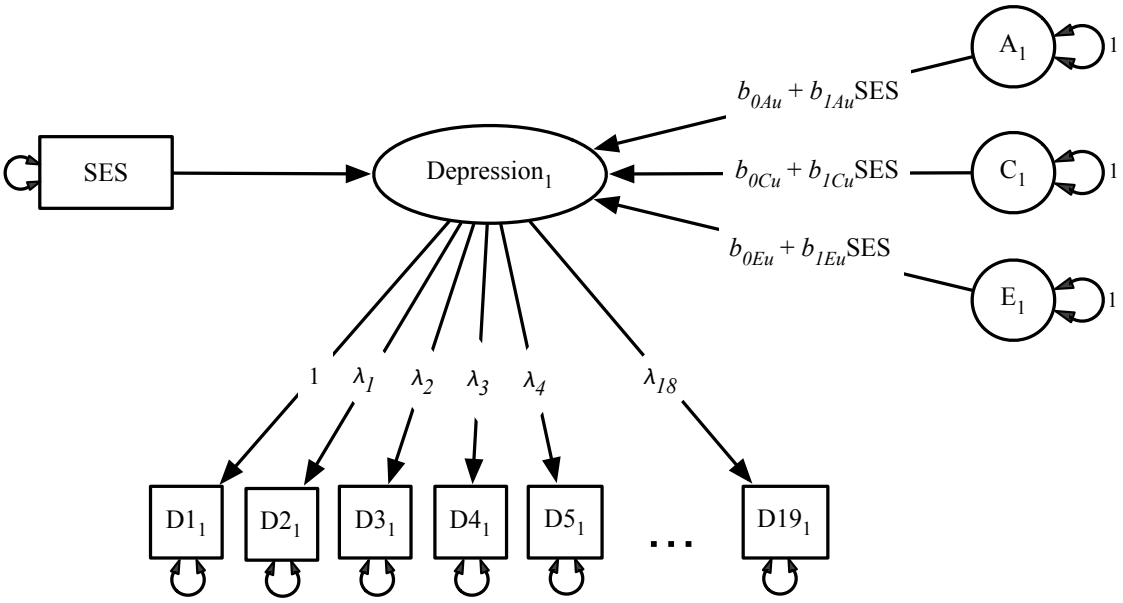


*Figure 5.1.* Path diagram of G×SES model fit to depression in the Add Health sample (only one twin shown for clarity). The residual variances for the depression items were permitted to correlate across twins, and were estimated freely according to zygosity.

**Family Income.** Depression was also negatively associated with family household income ($b$ = –0.022, $p$ = 0.048), although the effect size was minimal. Adolescents living with a family income at the first quartile ($20,000/year) had depression scores that were 0.03 standard deviations higher than adolescents with a family income at the third quartile ($56,000/year).

**Neighborhood Socioeconomic Advantage.** Neighborhood-level SES showed a negative association with depression ($b$ = –0.045, $p$ = 0.037); adolescents living in more deprived neighborhoods (first quartile) had depression scores that exceeded their peers (those living in more advantaged neighborhoods; third quartile) by 0.16 standard deviations.

**G×E Interaction: Effect of SES on Genetic and Environmental Etiology of Depression (Dissertation Aim 2).** The moderating effects of socioeconomic status on

Table 5.1. Parameter estimates and model fit statistics for G×SES models, depression in the Add Health sample.

| | Parameter | Phenotypic Model (No Moderation) | Moderation of Residual Variance | Best-Fitting Model |
|---|---|---|---|---|
| **Parental Education** | Main Effect of Parental Education on Depression | | | |
| | $b_1$ | ***–.050 (.013)*** | ***–.052 (.013)*** | ***–.050 (.013)*** |
| | Effect of Education on Residual ACE Components of Depression | | | |
| | $b_{0Au}$ | .326 (.389) | .137 (.339) | .326 (.389) |
| | $b_{1Au}$ | — | **–.066 (.022)** | — |
| | $b_{0Cu}$ | .322 (.349) | .188 (.352) | .322 (.349) |
| | $b_{1Cu}$ | — | .009 (.036) | — |
| | $b_{0Eu}$ | ***.231 (.160)*** | ***.690 (.165)*** | ***.231 (.160)*** |
| | $b_{1Eu}$ | — | .013 (.011) | — |
| | Model Fit | | | |
| | *-2LL* | 34899.146 | 34902.502 | 34899.146 |
| | *Δ-2LL (Δdf)* | — | 3.356 (+3) | — |
| | *p* | —† | .340 | — |
| **Family Household Income** | Main Effect of Family Income on Depression | | | |
| | $b_1$ | **–.022 (.011)** | –.047 (.026) | **–.022 (.011)** |
| | Effect of Family Income on Residual ACE Components of Depression | | | |
| | $b_{0Au}$ | .220 (.416) | .329 (.417) | .220 (.416) |
| | $b_{1Au}$ | — | .053 (.033) | — |
| | $b_{0Cu}$ | .314 (.278) | .574 (.380) | .314 (.278) |
| | $b_{1Cu}$ | — | .003 (.041) | — |
| | $b_{0Eu}$ | ***.622 (.159)*** | .371 (.177) | ***.622 (.159)*** |
| | $b_{1Eu}$ | — | –.009 (.007) | — |
| | Model Fit | | | |
| | *-2LL* | 31089.232 | 31085.394 | 31089.232 |
| | *Δ-2LL (Δdf)* | — | 3.838 (+2) | — |
| | *p* | —† | .279 | — |
| **Neighborhood Socioeconomic Advantage** | Main Effect of Neighborhood SES on Depression | | | |
| | $b_1$ | **–.045 (.022)** | **–.044 (.022)** | **–.045 (.022)** |
| | Effect of Neighborhood SES on Residual ACE Components of Depression | | | |
| | $b_{0Au}$ | .181 (.548) | .072 (.481) | .181 (.548) |
| | $b_{1Au}$ | — | –.048 (.033) | — |
| | $b_{0Cu}$ | .286 (.496) | .403 (.473) | .286 (.496) |
| | $b_{1Cu}$ | — | –.002 (.046) | — |
| | $b_{0Eu}$ | .132 (.179) | .122 (.166) | .132 (.179) |
| | $b_{1Eu}$ | — | –.009 (.016) | — |
| | Model Fit | | | |
| | *-2LL* | 29381.104 | 29378.842 | 29381.104 |
| | *Δ-2LL (Δdf)* | — | 2.262 (+3) | — |
| | *p* | —† | .520 | — |

*Note:* Standard errors presented within parentheses. Estimates p < .05 **bolded**. Estimates p < .01 *italicized*. Baseline model denoted by †.

variance in depression are presented in Table 5.1 under the heading *Effect of SES on Residual ACE Components of Depression.* The best-fitting model for each socioeconomic status indicator suggested that SES is not related to residual variance in depression.

**Brief Summary.** In general, socioeconomic status (both compositional and contextual) was related to mean levels of depression such that individuals living under more privileged socioeconomic circumstances reported fewer depressive symptoms on average. Socioeconomic status was unrelated to residual variance in depression in this sample of adolescents, however.

***Internalizing—WSTR.*** Internalizing in the WSTR sample was operationalized using 3 items from the PHQ-9 tapping depressive symptoms and 6 items from the BSI tapping symptoms of anxiety (see Chapter 3). We fit the model presented in Figure 5.2 to the data separately for each socioeconomic status indicator, partialling for the linear effects of age and gender on level of and variance in the internalizing factors. We used likelihood ratio tests to determine the best fitting model. We first established a baseline model by conducting omnibus tests for the moderation by SES of both the residual variance in the general internalizing factor and the main effects of SES on internalizing. We then systematically eliminated parameters from the baseline model to evaluate statistical significance of moderation parameters. Biometric decomposition of both the SES indicators and the internalizing factor are presented in Table 4.2, and parameter estimates and model fit statistics for the baseline and best-fitting models are presented in Table 5.2. The Area Deprivation Index was log-transformed to correct for positive skew, and the Gini Index was scaled by a factor of 10 to facilitate model convergence. These transformed values were used in all analyses in which the ADI or Gini Index was used as the indicator of contextual socioeconomic status. We did not include A for the ADI since there was no evidence of genetic influences on area deprivation in our univariate model.

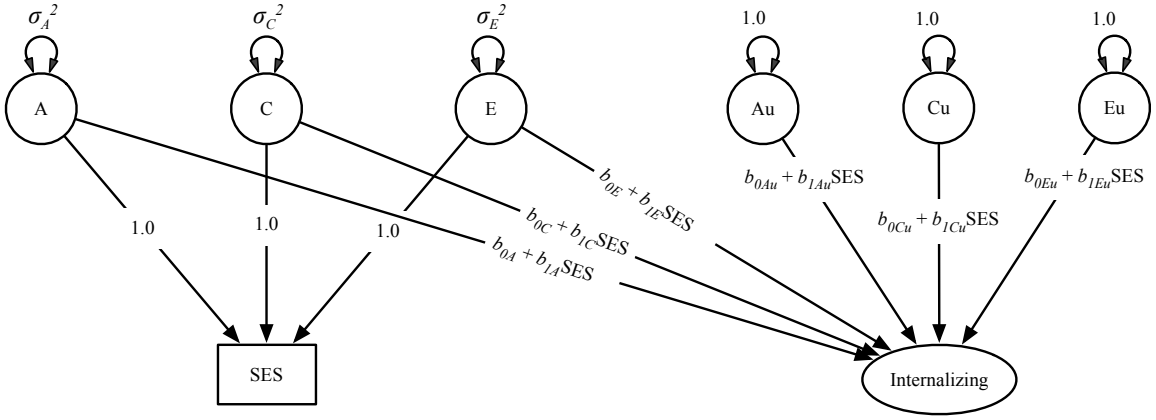


*Figure 5.2*. Path diagram of G×SES model fit to internalizing in the WSTR (only one twin and latent internalizing factors shown for clarity). In the fully saturated model, the main effects of SES on internalizing and the ACE variances of internalizing vary as a function of SES. The residual variances for the internalizing items, depression, and anxiety factors were permitted to correlate across twins, and were estimated freely according to zygosity.

**Causal Pathways vs. rGE: Main Effects of SES on Internalizing (Dissertation Aim 1).** The main effects of SES on the internalizing factors are presented in Table 5.2 under the heading *Main Effect of Moderator on Internalizing*. We discuss the main effects results for each moderator below.

**Education.** Education demonstrated a phenotypic association with internalizing ($b$ = –0.010, $p$ = 0.005), although the effect size was fairly small. Controlling for age and gender, individuals with a graduate or professional degree had internalizing scores that were 0.07 standard deviations lower than individuals holding a high school degree. The best-fitting model suggested that these associations are mediated by between-family factors; the quasi-causal association was nonsignificant and substantially reduced in magnitude compared with the phenotypic effect ($b_{0E}$ = 0.001, $p$ = 0.942). Although the additive genetic and shared environmental estimates were not statistically distinguishable from zero ($b_{0A}$ = –0.020, $p$ = 0.406; $b_{0C}$ = –0.012, $p$ = 0.652), they were not reduced in

Table 5.2. Parameter estimates and model fit statistics for G×SES models, internalizing behavior in the WSTR.

| | Parameter | Phenotypic Model | Quasi-Causal Model | Moderation of Residual Variance | Moderation of Main Effects | Best-Fitting Model |
|---|---|---|---|---|---|---|
| **Education** | Main Effect of Education on Internalizing | | | | | |
| | $b_{0A}$ | ***–.010 (.004)*** | –.020 (.024) | –.020 (.024) | –.007 (.107) | –.020 (.024) |
| | $b_{1A}$ | — | — | — | –.003 (.017) | — |
| | $b_{0C}$ | ***–.010 (.004)*** | –.008 (.027) | –.012 (.027) | .039 (.117) | –.012 (.027) |
| | $b_{1C}$ | — | — | — | –.008 (.018) | — |
| | $b_{0E}$ | ***–.010 (.004)*** | –.002 (.009) | .001 (.009) | –.073 (.184) | .001 (.009) |
| | $b_{1E}$ | — | — | — | .012 (.007) | — |
| | Effect of Education on Residual ACE Components of Internalizing | | | | | |
| | $b_{0Au}$ | **.221 (.095)** | **.221 (.095)** | .236 (.129) | .240 (.129) | ***.252 (.070)*** |
| | $b_{1Au}$ | — | — | .002 (.015) | .000 (.015) | — |
| | $b_{0Cu}$ | **.250 (.111)** | **.251 (.110)** | **.323 (.127)** | **.320 (.130)** | ***.309 (.093)*** |
| | $b_{1Cu}$ | — | — | –.021 (.015) | –.020 (.016) | –.020 (.010) |
| | $b_{0Eu}$ | ***.417 (.020)*** | ***417 (.020)*** | ***.538 (.030)*** | ***.538 (.030)*** | ***.537 (.029)*** |
| | $b_{1Eu}$ | — | — | ***–.022 (.004)*** | ***–.022 (.004)*** | ***–.022 (.004)*** |
| | Model Fit | | | | | |
| | *-2LL* | — | 101559.332 | 101472.528 | 101468.852 | 101472.548 |
| | *Δ-2LL (Δdf)* | — | — | 86.804 (+3) | 3.676 (+3) | .020 (–1) |
| | *p* | — | — | <.001† | .299 | .888 |
| **Household Income** | Main Effect of Income on Internalizing | | | | | |
| | $b_{0A}$ | ***–.014 (.002)*** | –.017 (.016) | –.016 (.015) | –.013 (.048) | –.015 (.014) |
| | $b_{1A}$ | — | — | — | .000 (.008) | — |
| | $b_{0C}$ | ***–.014 (.002)*** | –.024 (.031) | –.020 (.029) | –.100 (.099) | **–.108 (.053)** |
| | $b_{1C}$ | — | — | — | .015 (.017) | **.016 (.008)** |
| | $b_{0E}$ | ***–.014 (.002)*** | **–.010 (.004)** | **–.009 (.004)** | –.020 (.011) | –.007 (.004) |
| | $b_{1E}$ | — | — | — | .003 (.002) | — |
| | Effect of Income on Residual ACE Components of Internalizing | | | | | |
| | $b_{0Au}$ | **.222 (.102)** | **.218 (.104)** | ***.393 (.038)*** | ***.222 (.152)*** | ***.286 (.038)*** |
| | $b_{1Au}$ | — | — | ***–.013 (.002)*** | .007 (.015) | — |
| | $b_{0Cu}$ | **.255 (.114)** | **.257 (.113)** | ***.275 (.057)*** | ***.400 (.087)*** | ***.360 (.050)*** |
| | $b_{1Cu}$ | — | — | ***–.039 (.005)*** | ***–.038 (.009)*** | ***–.038 (.005)*** |
| | $b_{0Eu}$ | ***.407 (.020)*** | ***.407 (.020)*** | ***.436 (.021)*** | ***.445 (.022)*** | ***.442 (.021)*** |
| | $b_{1Eu}$ | — | — | **–.010 (.002)** | ***–.013 (.002)*** | ***–.013 (.002)*** |
| | Model Fit | | | | | |
| | *-2LL* | — | 104170.088 | 103961.860 | 103950.908 | 103953.106 |
| | *Δ-2LL (Δdf)* | — | — | 208.228 (+3) | 10.952 (+3) | 2.198 (–3) |
| | *p* | — | — | <.001 | .012† | .532 |
| **Area Deprivation** | Main Effect of Area Deprivation on Internalizing | | | | | |
| | $b_{0C}$ | ***–.144 (.049)*** | –.193 (.106) | **–.216 (.103)** | .935 (2.643) | **–.213 (.103)** |
| | $b_{1C}$ | — | — | — | –.245 (.551) | — |
| | $b_{0E}$ | ***–.144 (.049)*** | –.113 (.076) | –.108 (.078) | –2.188 (1.844) | –.110 (.077) |
| | $b_{1E}$ | — | — | — | .433 (.385) | — |
| | Effect of Area Deprivation on Residual ACE Components of Internalizing | | | | | |
| | $b_{0Au}$ | .000 (.719) | .000 (.781) | **1.720 (.873)** | **1.736 (.873)** | ***2.056 (.516)*** |
| | $b_{1Au}$ | — | — | **–.386 (.155)** | **–.391 (.157)** | ***–.428 (.108)*** |
| | $b_{0Cu}$ | ***.279 (.040)*** | ***.279 (.040)*** | 1.270 (1.091) | 1.322 (1.052) | ***.285 (.052)*** |
| | $b_{1Cu}$ | — | — | –.213 (.244) | –.226 (.236) | — |
| | $b_{0Eu}$ | ***.385 (.026)*** | ***.385 (.026)*** | ***1.159 (.236)*** | ***1.169 (.239)*** | ***1.161 (.236)*** |
| | $b_{1Eu}$ | — | — | ***–.169 (.050)*** | ***–.171 (.051)*** | ***–.170 (.050)*** |
| | Model Fit | | | | | |
| | *-2LL* | — | 41116.624 | 41080.246 | 41078.750 | 41080.836 |
| | *Δ-2LL (Δdf)* | — | — | 36.378 (+3) | 1.496 (+2) | 2.086 (–1) |
| | *p* | — | — | <.001† | .473 | .149 |
| **Income Inequality** | Main Effect of Income Inequality on Internalizing | | | | | |
| | $b_{0A}$ | .029 (.020) | .402 (.564) | 1.396 (1.379) | | .402 (.564) |
| | $b_{1A}$ | — | — | — | | — |
| | $b_{0C}$ | .029 (.020) | –.065 (.194) | 1.396 (1.379) | | –.065 (.194) |
| | $b_{1C}$ | — | — | — | | — |
| | $b_{0E}$ | .029 (.020) | .013 (.031) | .074 (.122) | | .013 (.031) |
| | $b_{1E}$ | — | — | — | | — |
| | Effect of Income Inequality on Residual ACE Components of Internalizing | | | | | |
| | $b_{0Au}$ | .187 (.193) | .182 (.214) | .280 (.561) | | .182 (.214) |
| | $b_{1Au}$ | — | — | .142 (.124) | | — |
| | $b_{0Cu}$ | .255 (.148) | .255 (.157) | .280 (.561) | | .255 (.157) |
| | $b_{1Cu}$ | — | — | .142 (.124) | | — |
| | $b_{0Eu}$ | ***.426 (.023)*** | ***.427 (.024)*** | ***1.397 (.359)*** | | ***.427 (.024)*** |
| | $b_{1Eu}$ | — | — | –.063 (.081) | | — |
| | Model Fit | | | | | |
| | *-2LL* | — | 69923.618 | 69921.650 | | 69923.618 |
| | *Δ-2LL (Δdf)* | — | — | 1.968 (+2) | | — |
| | *p* | — | —† | .579 | | — |

*Note:* Standard errors presented within parentheses. Estimates $p < .05$ **bolded**. Estimates $p < .01$ *italicized*. Baseline model denoted by †.

magnitude compared with the phenotypic effect. This observation likely represents a lack of power to differentiate between these sources of covariation. When estimating the total between-family effect (achieved by constraining $b_{0A}$ and $b_{0C}$ to be equal), however, there was significant positive between-family confounding between household income and internalizing ($b_{0A} = b_{0C} = –0.014$, $p = 0.007$; $b_{0E} = –0.002$, $p = 0.788$; results from this model not presented in Table 5.2). These results suggest that the relation between educational attainment and internalizing is best explained by underlying genetic or shared environmental factors (particularly the former) that are common to both phenotypes rather than due to systematic differences in exposure to socioeconomic factors.

As illustrated in the left panel of Figure 5.3, which shows the magnitude of the genetic and nonshared environmental regressions of internalizing on educational attainment, the between-family regressions are larger than the within-family regression. These regression lines are re-represented as genetic and nonshared environmental correlations in the middle panel of Figure 5.3. We also present the proportions of the total phenotypic correlation accounted for by the genetic and nonshared environmental correlations in the right panel of Figure 5.3. Evident in this figure is that the nonshared environmental correlation accounts for essentially none of the overall phenotypic correlation. Instead, approximately 60% of the phenotypic correlation is accounted for additive genetic factors common to both educational attainment and internalizing, and approximately 40% of the phenotypic effect is accounted for by a common shared environmental pathway.

These phenotypic and within-family effects are demonstrated in Figure 5.4, an

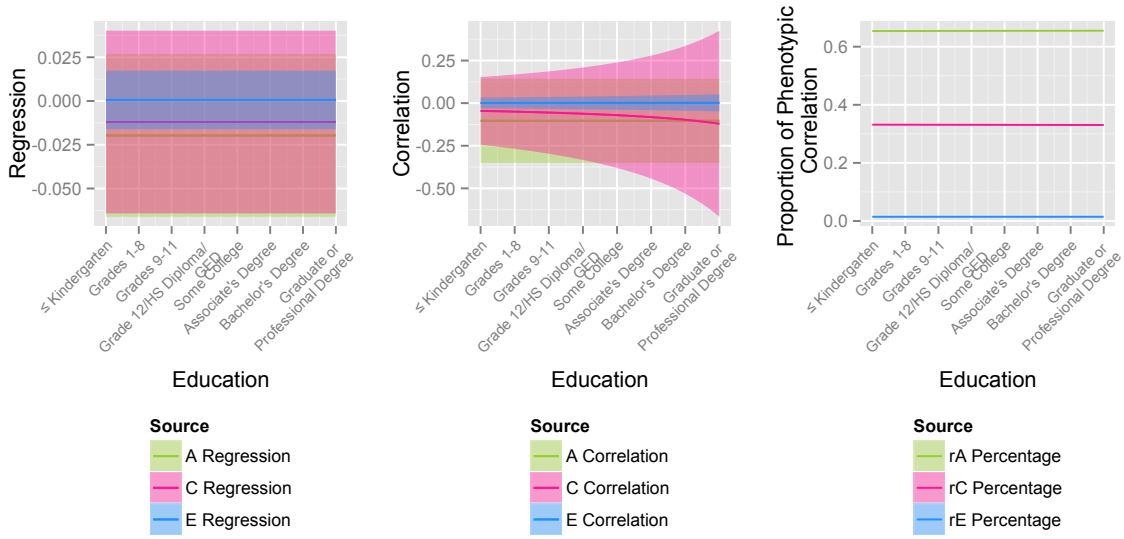


*Figure 5.3.* Main effects educational attainment on general internalizing in the WSTR. The left panel shows the regression of internalizing on educational attainment. The same relation is presented in the middle panel as genetic (green), shared environmental (pink), and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic, shared environmental, and nonshared environmental correlations. Asterisks (*) indicate that the magnitude of the regression of internalizing on educational attainment depends on level of education (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). Shaded regions represent 95% confidence intervals around regression lines.

illustrative analysis of the main effect of educational attainment on internalizing. We identified twin pairs concordant for lower education (up to Associate's degree; orange), pairs concordant for higher education (Bachelor's degree or higher; navy), and pairs discordant for educational status, and compared mean internalizing scores (using a summed score of the depression and anxiety items) of each of these groups. Overall, there is a main effect of educational attainment on internalizing such that higher education is associated with fewer internalizing symptoms on average (this is evident comparing the outer bars in this figure). Examining the relation at the within-pair level, however, shows that it does not exist within pairs of MZ twins.

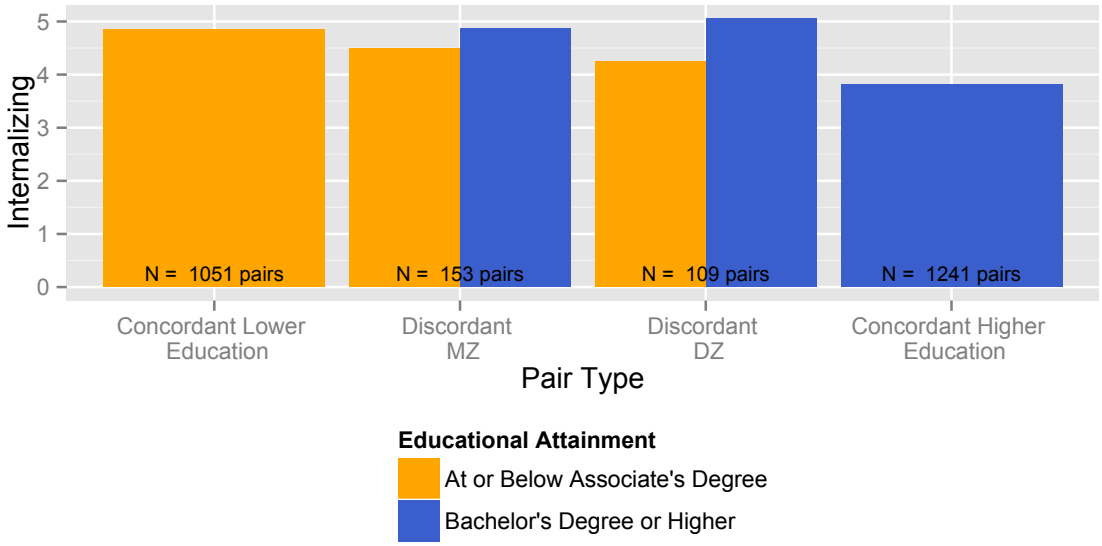


*Figure 5.4.* Illustrative analysis of the main effects of educational attainment on internalizing in the WSTR. The less educated twin (orange) is compared with the more educated twin (navy) in MZ and DZ twin pairs.

**Income.** The phenotypic regression of the general internalizing factor on income showed a significant negative relationship ($b$ = –0.014, $p$ < 0.001). That is, controlling for age and gender, individuals at the third quartile of earned income scored 0.15 standard deviations lower on the internalizing factor than their counterparts at the first quartile of earned income. The best-fitting model showed a quasi-causal effect that was somewhat reduced in magnitude and was not statistically significant ($b_{0E}$ = –0.007, $p$ = 0.089). The common genetic background to income and internalizing was also nonsignificant though not reduced in magnitude ($b_{0A}$ = –0.015, $p$ = 0.261), suggesting that genes contributing to earned income may also be the same genes influencing internalizing symptomatology. A statistically significant common shared environmental pathway also confounded this association ($b_{0C}$ = –0.108, $p$ = 0.042) and grew weaker as income level increased ($b_{1C}$ = 0.016, $p$ = 0.047). These results suggest that the relation between earned income and

internalizing is best explained by underlying genetic or shared environmental factors (primarily the latter) that are common to both phenotypes rather than due to systematic differences in exposure to socioeconomic factors. Results from the best-fitting model are illustrated in Figure 5.5. Like educational attainment, household income tended to be correlated with internalizing primarily via additive genetic and shared environmental pathways common to both phenotypes and accounted for the majority of the phenotypic correlation. These phenotypic and within-family effects are illustrated in Figure 5.6. Again, the overall effect of household income on internalizing is diminished within pairs of MZ and DZ twins, consisted with between-family mediation of this association.

**Area Deprivation.** At the phenotypic level, the ADI was statistically significantly associated with the general internalizing factor ($b$ = –0.144, $p$ = 0.003), although the effect size was minimal. Individuals at the third quartile of the ADI had scores on the general internalizing factor that were 0.06 standard deviations lower than individuals at the first quartile of the ADI. General internalizing was not quasi-causally associated with the ADI ($b_{0E}$ = –0.110, $p$ = 0.157) and instead was correlated via a common shared environmental pathway ($b_{0C}$ = –0.213, $p$ = 0.039). These results are largely consistent between-family confounding of the SES–internalizing association observed using individual-level SES measures. Model results are illustrated in Figure 5.7, where it is evident that the magnitude of the between-family correlation exceeds that of the within-family correlation. Likewise, the between-family pathway from neighborhood-level SES to the general internalizing factor accounted for nearly twice as much of the total phenotypic correlation than did the within-family pathway. Illustrative analyses (see Figure 5.8) also demonstrate the lack of effect of neighborhood-level SES

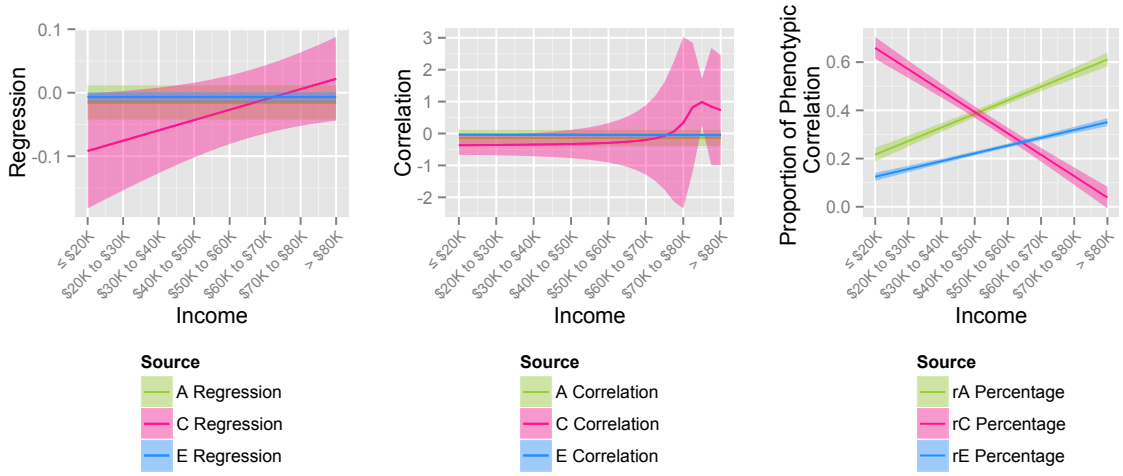


*Figure 5.5.* Main effects of household income on general internalizing in the WSTR. The left panel shows the regression of internalizing on income. The same relation is presented in the middle panel as genetic (green), shared environmental (pink), and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic, shared environmental, and nonshared environmental correlations. Asterisks (*) indicate that the magnitude of the regression of internalizing on household income depends on level of income (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). Shaded regions represent 95% confidence intervals around regression lines.

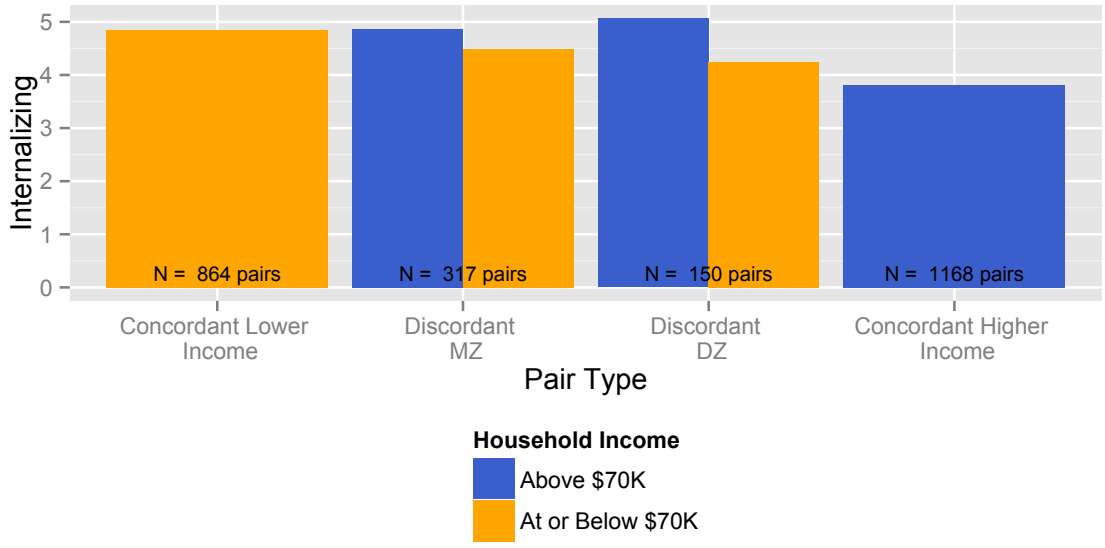


*Figure 5.6.* Illustrative analysis of the main effects of household income on internalizing in the WSTR. The twin earning less income (orange) is compared with the twin earning more income (navy) in MZ and DZ twin pairs.

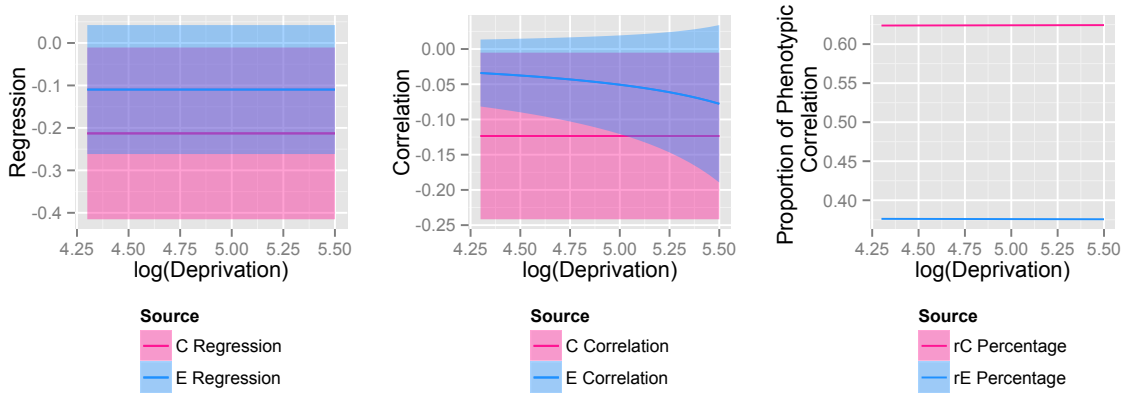


*Figure 5.7.* Main effects of area deprivation on general internalizing in the WSTR. The left panel shows the regression of internalizing on neighborhood-level SES. The same relation is presented in the middle panel as shared environmental (pink) and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic, shared environmental, and nonshared environmental correlations. Asterisks (*) indicate that the magnitude of the regression of internalizing on neighborhood-level SES depends on level of income (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). Shaded regions represent 95% confidence intervals around regression lines.

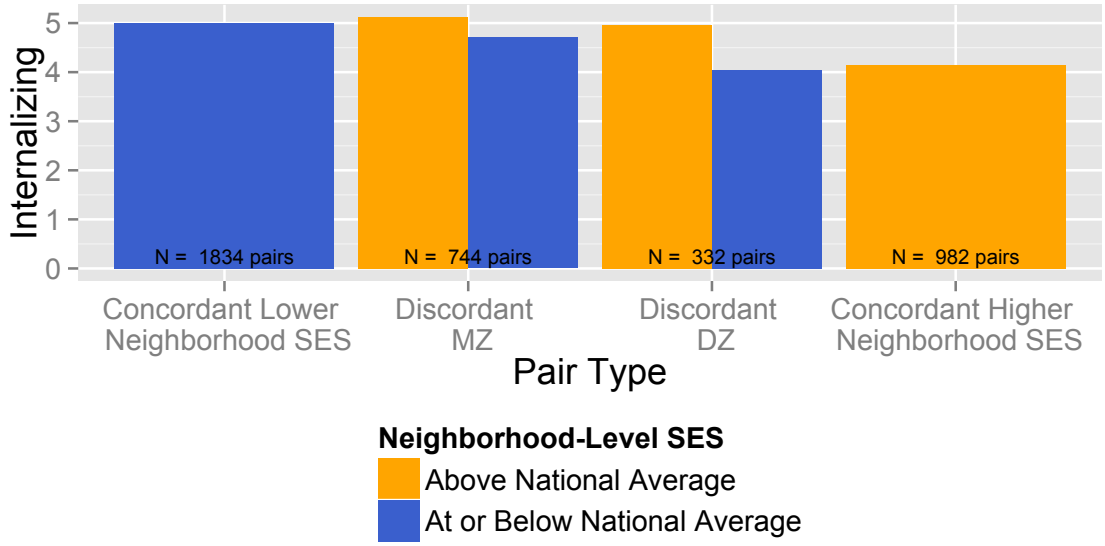


*Figure 5.8.* Illustrative analysis of the main effects of neighborhood-level socioeconomic status on internalizing in the WSTR. The twin residing in the less socioeconomically advantaged neighborhood (orange) is compared with the twin living in the more affluent neighborhood (navy) in MZ and DZ twin pairs.

within twin pairs.

**Income Inequality.** Income inequality showed no evidence of main effects on the general internalizing factor ($b$ = 0.029, $p$ = 0.131).

**G×E Interaction: Effect of SES on Genetic and Environmental Etiology of Internalizing (Dissertation Aim 2).** The moderating effects of SES on variance in internalizing are presented in Table 5.2 under the heading *Effect of SES on Residual ACE Components of Internalizing.* We discuss the interactive effect of each SES indicator on variance in the internalizing factors below.

**Education.** The best fitting model suggested that residual phenotypic variance in the general internalizing factor decreased with increasing educational attainment. This effect was driven by decreasing shared environmental ($b_{1Cu}$ = –0.020, $p$ = 0.054[20]) nonshared environmental variance ($b_{1Eu}$ = –0.022, $p$ < 0.001); there was no evidence that additive genetic variance depended on level of education. These model results are illustrated in Figure 5.9, which shows residual ACE variance components of the internalizing factor as a function of educational attainment, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure clearly demonstrates how residual phenotypic and environmental variance in internalizing decreases with increasing educational attainment. Considered within the context of the dynamics of the ACE correlations, it does not appear that the presence of factors contributing to internalizing depends on level of education, suggesting that education is acting to restrict nonshared environmental variation in internalizing. Stated differently,

[20] Although this parameter estimate was not statistically significant, it could not be dropped from the model without significant decrement in model fit.

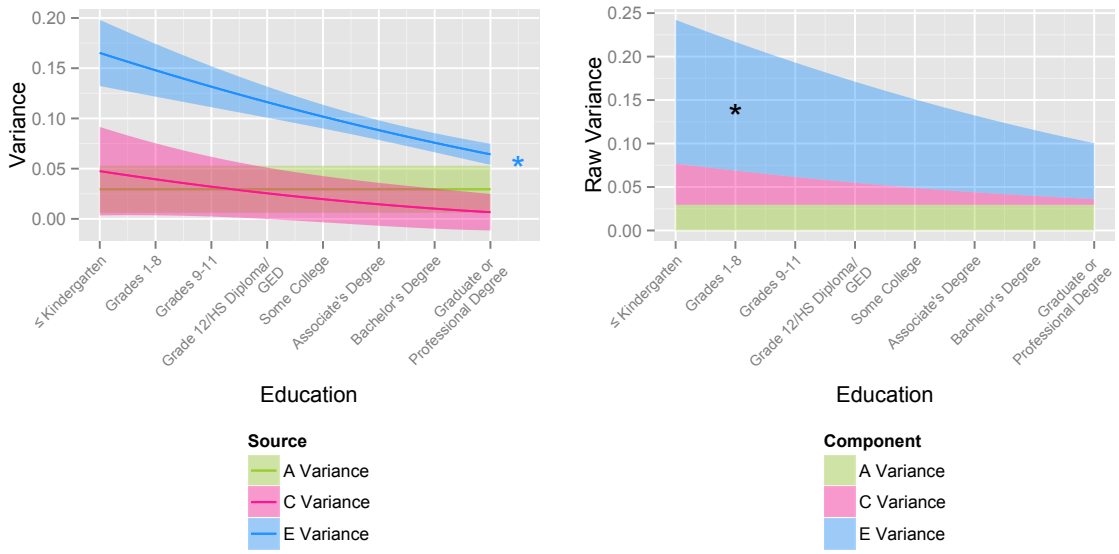


*Figure 5.9.* Gene-by-environment interaction between general internalizing and educational attainment in the WSTR. The left panel shows ACE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in the internalizing factors decreases as a function of increasing educational attainment. The asterisks (*) indicate that variance in internalizing depends on level of education.

high-SES environments appear to protect against nonshared environmental risk for endorsing internalizing symptoms.

To illustrate these results, we plotted absolute pair differences in internalizing for MZ and DZ twins against pair average educational attainment (see the left panel of Figure 5.10). In this plot, the gap between the MZ and DZ regression lines (*i.e.*, the tendency for MZ pairs to be more similar than DZ pairs) represents additive genetic variance in internalizing. Increases in this gap with respect to education would suggest that additive genetic variance in internalizing increases with increasing educational attainment; decreases would suggest the opposite. The location of the MZ line in reflects nonshared environmental variance in internalizing. A decreasing slope represents decreasing nonshared environmental variance in internalizing. Evident in this plot is that

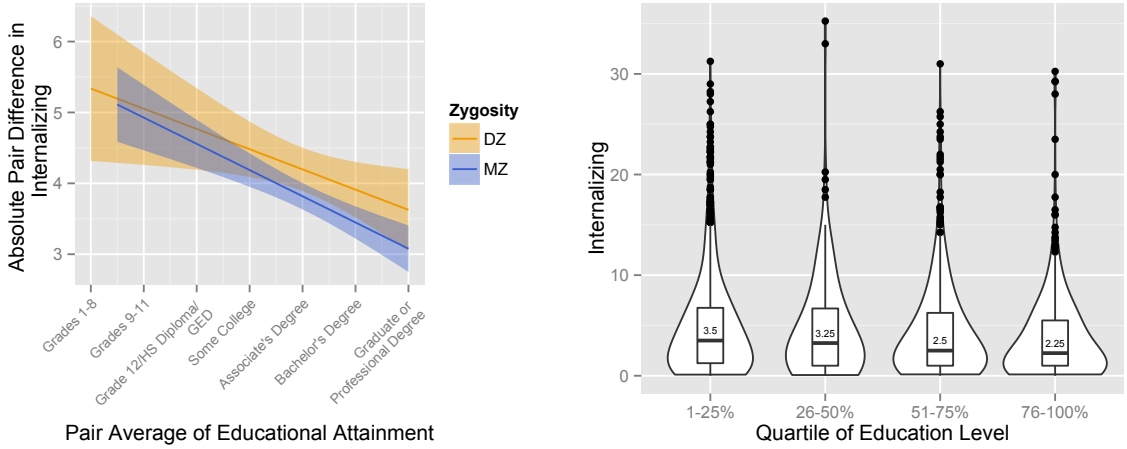


*Figure 5.10.* Illustrative analysis of the effects of educational attainment on variance in internalizing in the WSTR. The left panel shows absolute pair differences in internalizing as a function of the pair average educational attainment. The distance between the MZ and DZ lines represents the additive genetic variance in internalizing. The location of the MZ line represents nonshared environmental variance in internalizing. The right panel shows box plots overlaid with violin plots, internalizing as a function of quartile of educational attainment.

the distance between the MZ and DZ regression lines remains the same across all levels of education; that is, additive genetic variance in internalizing is stable with respect to educational attainment. The negative slope of the MZ regression line reflects decreasing nonshared environmental variance in internalizing.

This heteroscedasticity is further illustrated in the right panel of Figure 5.10, which shows box plots overlaid with violin plots (which show the probability density of the data) of internalizing by quartile of educational attainment. Several characteristics of this plot are worth mentioning. First, the median internalizing score tends to decrease with increasing educational attainment, illustrating the main effect of SES on internalizing at the phenotypic level. Second, the distribution of internalizing becomes less platykurtic at higher levels of educational attainment, demonstrating that overall variance in internalizing 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 internalizing distribution at higher SES levels.

**Income.** The best-fitting model suggested that variance in internalizing decreases as a function of increasing household income, an effect driven by decreasing shared environmental ($b_{1Cu}$ = –0.038, $p < 0.001$) nonshared environmental ($b_{1Eu}$ = –0.013, $p$ <0.001) variances. As with education, there was no evidence that additive genetic variance depended on income level. These model results are illustrated in Figure 5.11. Considered within the context of the dynamics of the ACE correlations (*i.e.*, static nonshared environmental relationship and a decreasing shared environmental correlation), it appears that the presence of depressogenic or anxiogenic factors characteristic to families may depend on household income, whereas those characteristic to individuals do not. That is, something about earning more money restricts nonshared environmental variation in internalizing. The association between household income and shared environmental variation, on the other hand, is due to selection factors.

These results are further illustrated in Figure 5.12. As we observed with education, nonshared environmental variance (but not additive genetic variance) in internalizing decreases as a function of increasing household income. The violin plots demonstrate that total phenotypic variance is decreasing, an effect that appears to be the result of fewer observations at the higher end of internalizing when income level is high.

**Area Deprivation.** The best fitting model suggested that residual phenotypic variance in the internalizing factors decreases with decreasing area deprivation. These effects were driven primarily by decreases in additive genetic ($b_{1Au}$ = –0.428, $p < 0.001$)

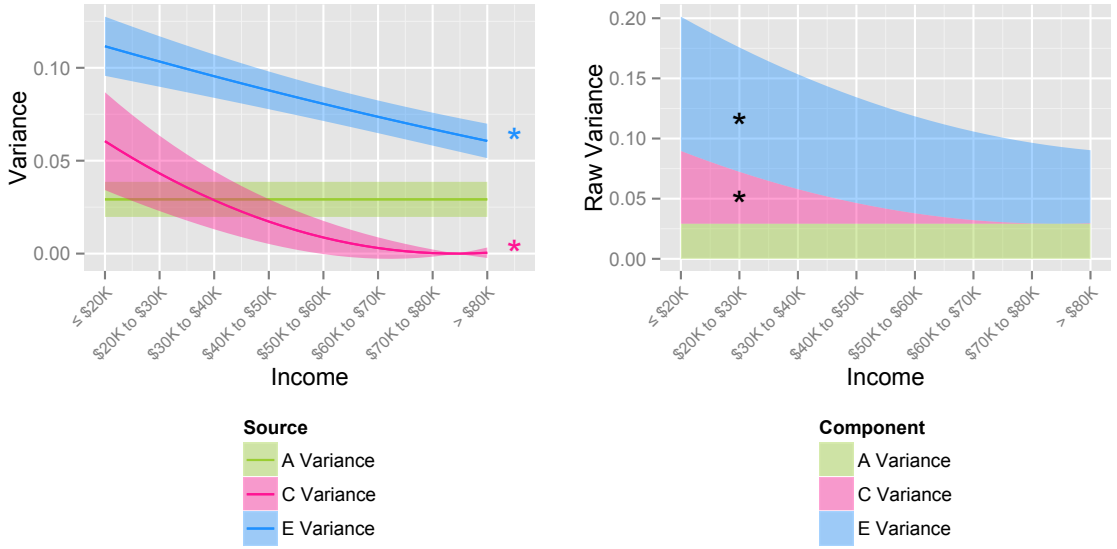


*Figure 5.11.* Gene-by-environment interaction between general internalizing and household income in the WSTR. The left panel shows ACE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in the internalizing factors decreases as a function of increasing income. The asterisks (*) indicate that variance in internalizing depends on level of income.

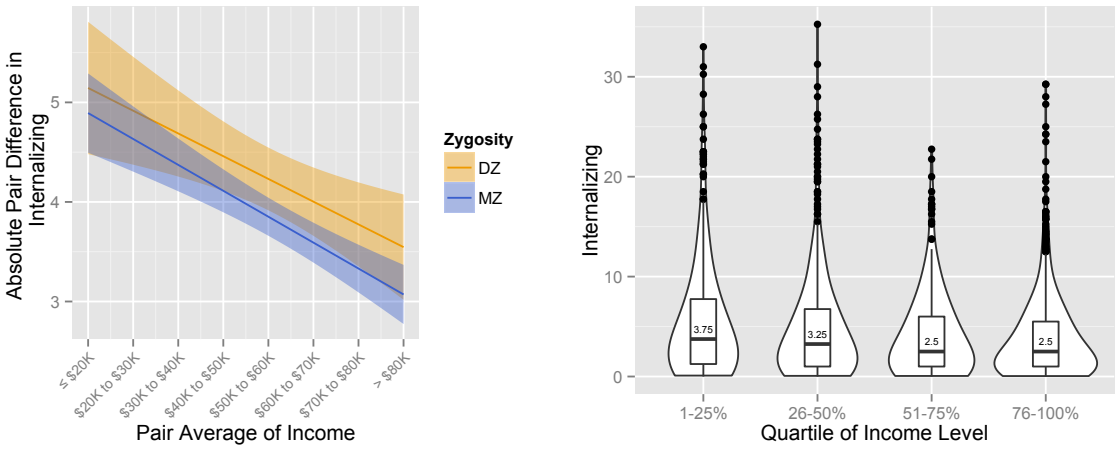


*Figure 5.12.* Illustrative analysis of the effects of income on variance in internalizing in the WSTR. The left panel shows absolute pair differences in internalizing as a function of the pair average household income. The distance between the MZ and DZ lines represents the additive genetic variance in internalizing. The location of the MZ line represents nonshared environmental variance in internalizing. The right panel shows box plots overlaid with violin plots, internalizing as a function of quartile of income level.

and nonshared environmental ($b_{1Eu}$ = –0.170, $p$ = 0.001) variances. These model results are illustrated in Figure 5.13. As with compositional measure of SES, total phenotypic variance in each internalizing phenotype decreased with increasing neighborhood-level SES, and this association tended to be driven by decreases in the additive genetic and nonshared environmental variance components. Considered within the context of stable ACE correlations with respect to the ADI, it does not appear that the presence of depressogenic or anxiogenic factors depends on level of area deprivation, suggesting that something about higher neighborhood-level SES is restricting variation (both genetic and environmental) in internalizing. That is, low-SES environments appear to exacerbate environmental and/or genetic risk for developing internalizing symptoms, and high-SES environments appear to protect against genetic and environmental risk for developing internalizing symptoms.

These results are further illustrated in Figure 5.14. As we observed with household education, nonshared environmental variance (but not additive genetic variance) in internalizing decreases as a function of increasing household income. The violin plots show that total phenotypic variance in internalizing is decreasing with increasing neighborhood SES, an effect that appears to be the result of fewer observations at the higher end of internalizing when neighborhood SES is high.

**Income Inequality.** The best fitting model suggested that residual phenotypic variance in the general internalizing factor remained stable with respect to county-level income inequality.

**Brief Summary.** Both compositional and contextual measures of socioeconomic status were found to be protective against symptoms of internalizing. Between-family

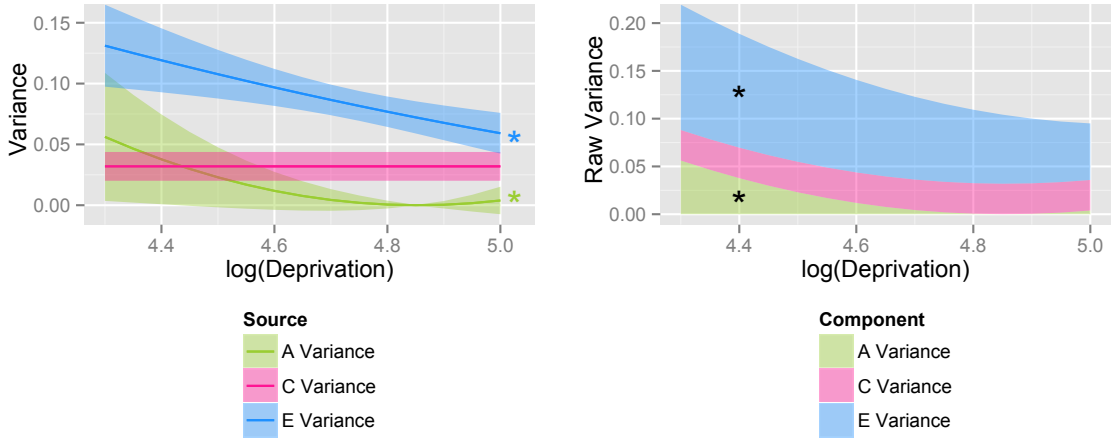


*Figure 5.13.* Gene-by-environment interaction between general internalizing and area deprivation in the WSTR. The left panel shows ACE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in the internalizing factor decreases as a function of decreasing area deprivation. The asterisks (*) indicate that variance in internalizing depends on level of the ADI.

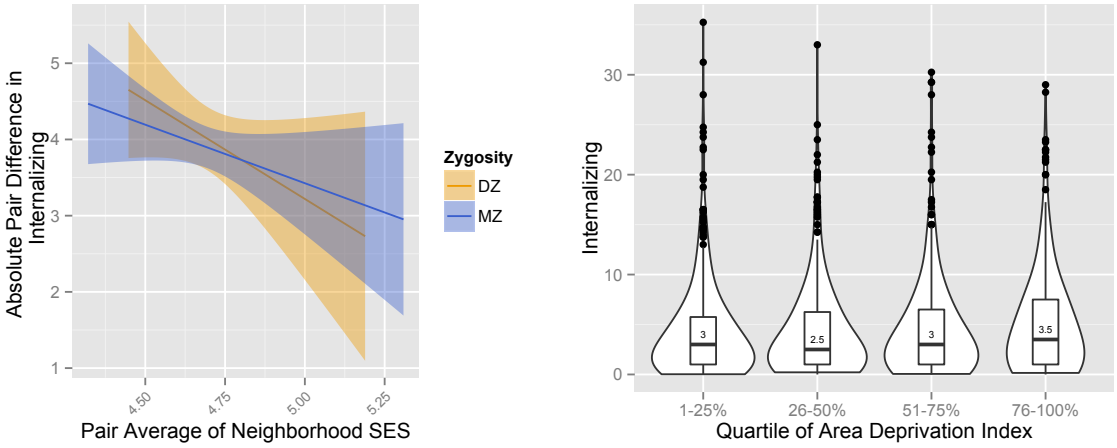


*Figure 5.14.* Illustrative analysis of the effects of neighborhood-level socioeconomic advantage on variance in internalizing in the WSTR. The left panel shows absolute pair differences in internalizing as a function of the pair average ADI. The distance between the MZ and DZ lines represents the additive genetic variance in internalizing. The location of the MZ line represents nonshared environmental variance in internalizing. The right panel shows box plots overlaid with violin plots, internalizing as a function of quartile of the ADI.

(*i.e.*, non-causal) factors tended to explain the majority of this association, however. Residual variance in internalizing was also influenced by compositional and contextual measures of SES. Residual phenotypic variance tended to decrease with increasing socioeconomic status, an effect driven primarily by decreases primarily in nonshared environmental variance. Finally, these decreases in these residual variances appeared to be potentiated by socioeconomic status, and not by social selection factors.

**Externalizing Behavior**

***ADHD—Add Health.*** Attention-deficit/hyperactivity disorder in the Add Health sample was operationalized using an 18-item ADHD symptom checklist (see Chapter 3). We fit the model presented in Figure 5.15 to the data separately for each socioeconomic status indicator, partialling for the linear effects of age and gender on level and variance in the ADHD factor. We used likelihood ratio tests to determine the best fitting model. Biometric decomposition of both the SES indicators and the ADHD factor are presented in Table 4.1, and parameter estimates for each of these models are presented in Table 5.3. Family household income was log-transformed to correct for positive skew. The ADHD factor did not show evidence of influence from shared environmental factors; therefore, residual C was not included in any of the fitted models.

**Causal Pathways vs. rGE: Main Effects of SES on ADHD (Dissertation Aim 1).** The main effects of SES on ADHD are presented in Table 5.3 under the heading *Main Effect of Moderator on ADHD*. We discuss the main effects results for each moderator below.

**Parental Education.** Parental education was significantly associated with ADHD symptomatology ($b = –0.027$, $p = 0.042$). That is, adolescents with a parent who

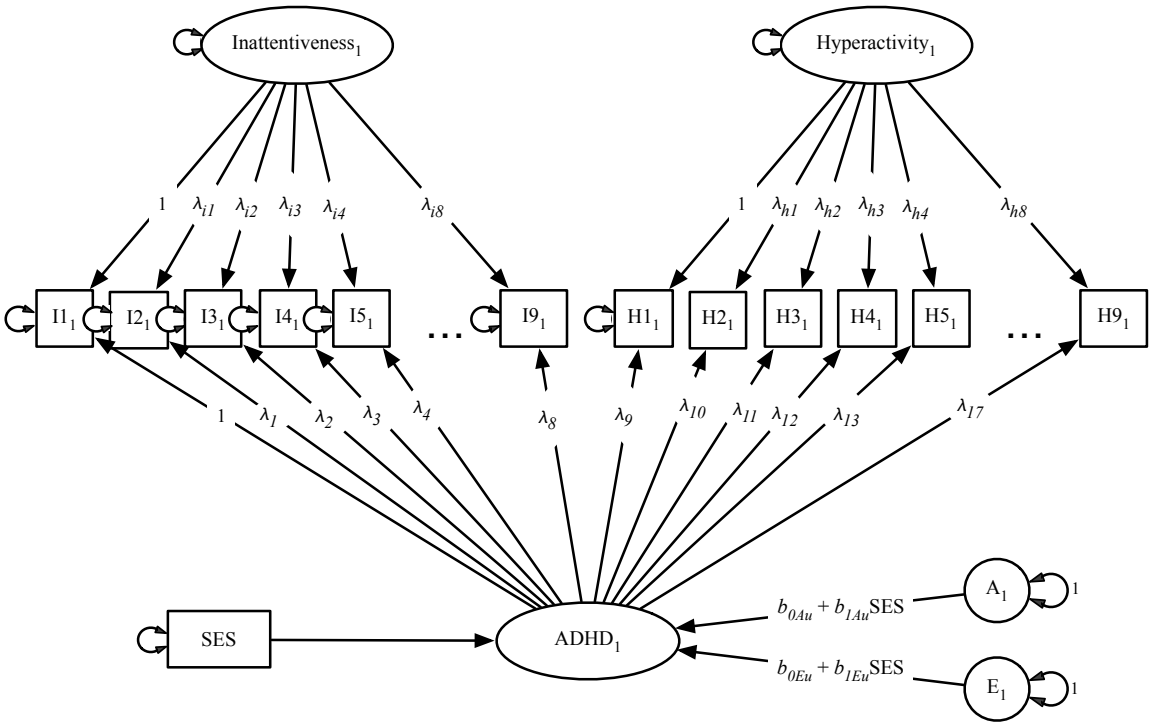


*Figure 5.15.* Path diagram G×SES model fit to ADHD in the Add Health sample (only one twin shown for clarity). The residual variances for the ADHD items were permitted to correlate across twins, and were estimated freely according to zygosity.

had earned at least a college degree had ADHD scores that were 0.13 standard deviations lower than their counterparts whose parent holds a high school degree.

**Family Income.** ADHD was not statistically significantly associated with family household income ($b = –0.006$, $p = 0.510$).

**Neighborhood Socioeconomic Advantage.** Area deprivation did not show evidence of an association with ADHD ($b = –0.008$, $p = 0.719$).

***G×E Interaction: Effect of SES on Genetic and Environmental Etiology of ADHD.*** The moderating effects of SES on variance in ADHD are presented in Table 5.3 under the heading *Effect of SES on Residual ACE Components of ADHD.*

**Education.** The best-fitting model suggested that residual phenotypic variance in

Table 5.3. Parameter estimates and model fit statistics for G×SES models, ADHD in the Add Health sample.

| | Parameter | Phenotypic Model (No Moderation) | Moderation of Residual Variance | Best-Fitting Model |
|---|---|---|---|---|
| **Parental Education** | Main Effect of Parental Education on ADHD | | | |
| | $b_1$ | **–.027 (.013)** | **–.026 (.013)** | **–.026 (.013)** |
| | Effect of Education on Residual ACE Components of ADHD | | | |
| | $b_{0Au}$ | .271 (.298) | .341 (.328) | .307 (.275) |
| | $b_{1Au}$ | — | –.004 (.019) | — |
| | $b_{0Eu}$ | **.463 (.210)** | .386 (.224) | .405 (.202) |
| | $b_{1Eu}$ | — | **.027 (.013)** | **.025 (.010)** |
| | Model Fit | | | |
| | *-2LL* | 30026.246 | 30019.980 | 30020.018 |
| | *Δ-2LL (Δdf)* | — | 6.266 (+2) | .038 (–1) |
| | *p* | — | .044† | .845 |
| **Family Household Income** | Main Effect of Family Income on ADHD | | | |
| | $b_1$ | –.006 (.010) | –.010 (.011) | –.006 (.010) |
| | Effect of Family Income on Residual ACE Components of ADHD | | | |
| | $b_{0Au}$ | .061 (.292) | .329 (.417) | .061 (.292) |
| | $b_{1Au}$ | — | .053 (.033) | — |
| | $b_{0Eu}$ | ***.570 (.205)*** | .371 (.177) | ***.570 (.205)*** |
| | $b_{1Eu}$ | — | –.009 (.007) | — |
| | Model Fit | | | |
| | *-2LL* | 26498.536 | 26493.214 | 26498.536 |
| | *Δ-2LL (Δdf)* | — | 5.322 (+2) | — |
| | *p* | —† | .070 | — |
| **Neighborhood Socioeconomic Advantage** | Main Effect of Neighborhood SES on ADHD | | | |
| | $b_1$ | –.008 (.022) | –.011 (.008) | –.011 (.008) |
| | Effect of Neighborhood SES on Residual ACE Components of ADHD | | | |
| | $b_{0Au}$ | .320 (.287) | .053 (.348) | .053 (.348) |
| | $b_{1Au}$ | — | **.099 (.039)** | **.099 (.039)** |
| | $b_{0Eu}$ | .294 (.186) | **.533 (.215)** | **.533 (.215)** |
| | $b_{1Eu}$ | — | **–.044 (.020)** | **–.044 (.020)** |
| | Model Fit | | | |
| | *-2LL* | 25761.612 | 25753.282 | 25753.282 |
| | *Δ-2LL (Δdf)* | — | 8.330 (+2) | — |
| | *p* | — | .016† | — |

*Note:* Standard errors presented within parentheses. Estimates p < .05 **bolded**. Estimates p < .01 *italicized*. Baseline model denoted by †.

ADHD increased with increasing parental educational attainment, an effect driven by increasing nonshared environmental variance ($b_{1Eu}$ = 0.025, $p$ = 0.012); there was no moderation of genetic variance as a function of educational attainment. These model results are illustrated in Figure 5.16, which shows residual AE variance components of ADHD as a function of parental educational attainment, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure clearly

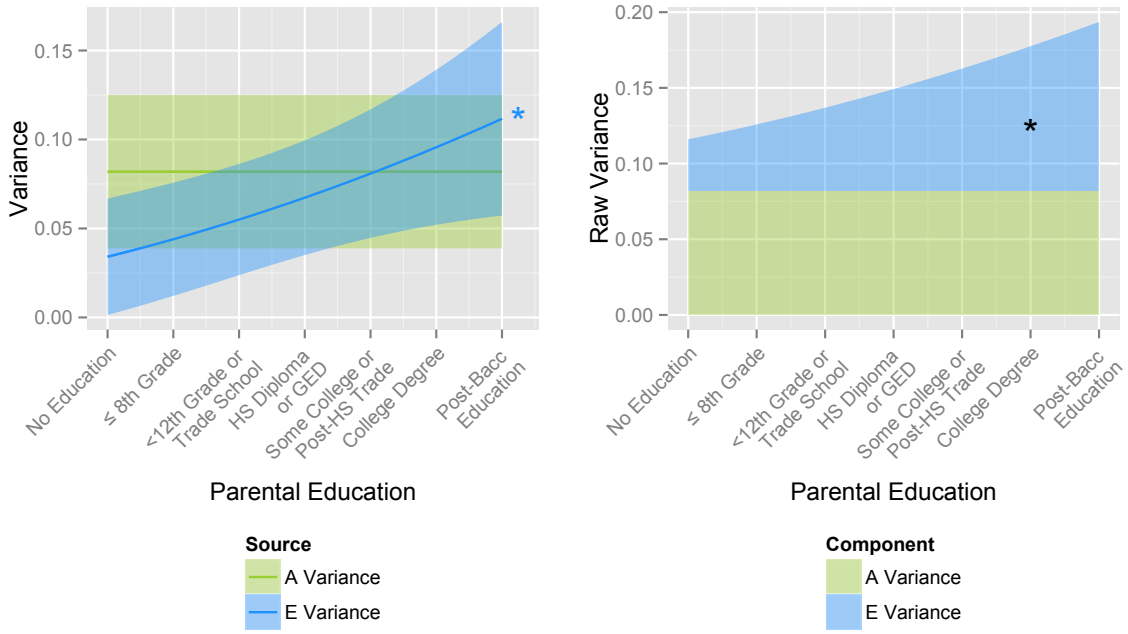


*Figure 5.16.* Gene-by-environment interaction between ADHD symptoms and parental educational attainment in the Add Health sample. The left panel shows AE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in ADHD symptoms increases as a function of increasing parental educational attainment. The asterisks (*) indicate that variance in ADHD symptomatology depends on level of parental education.

demonstrates how residual phenotypic and nonshared environmental variance in ADHD symptoms increases with increasing parental educational attainment. It seems that high-SES environments potentiate environmental risk for ADHD.

To illustrate these results, we plotted absolute pair differences in ADHD symptom counts for MZ and DZ twins against parental educational attainment (see the left panel of Figure 5.17). Evident in this plot is that the distance between the MZ and DZ regression lines increases across parental education, an effect apparently driven by DZ twins growing less similar to one another. The model results, however, indicated that this change in variance was attributable to changes in the nonshared environmental influences

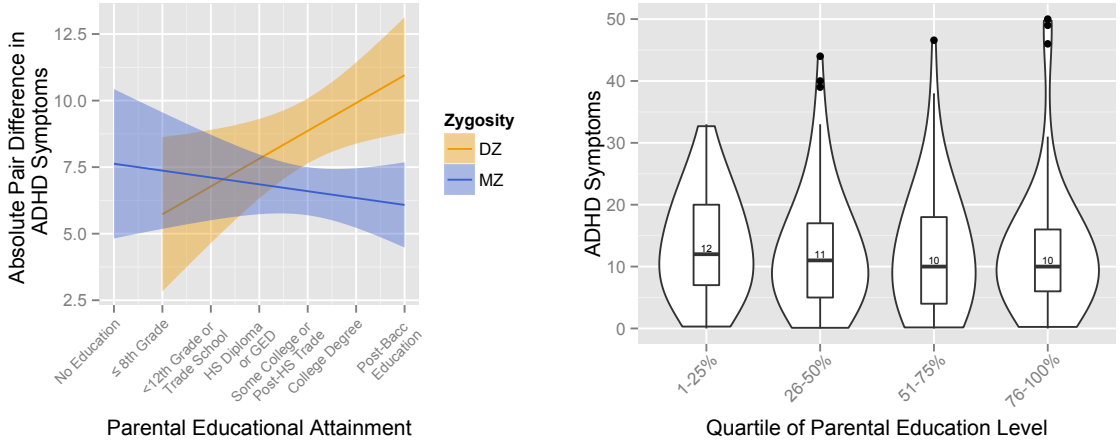


*Figure 5.17.* Illustrative analysis of the effects of parental education on variance in ADHD in the Add Health sample. The left panel shows absolute pair differences in ADHD as a function of parental educational attainment. The distance between the MZ and DZ lines represents the additive genetic variance in ADHD. The location of the MZ line represents nonshared environmental variance in ADHD. The right panel shows box plots overlaid with violin plots, ADHD as a function of quartile of parental education.

on ADHD. This could be the result of analyzing variance in ADHD after partialling for variance attributable to inattention and variance attributable to hyperactivity. The violin plot in the right panel of Figure 5.17 illustrates this increased variance in ADHD symptoms, as well as an overall protective main effect.

**Family Income.** The best-fitting model suggested that residual variance in ADHD symptomatology is not moderated by family income.

**Neighborhood Socioeconomic Advantage.** The best-fitting model suggested that residual phenotypic variance in ADHD decreased with increasing neighborhood-level socioeconomic status, an effect driven primarily by increasing additive genetic variance ($b_{1Au}$ = 0.099, $p$ = 0.010). Nonshared environmental variance decreased as a function of neighborhood-level SES ($b_{1Eu}$ = –0.044, $p$ = 0.024). These model results are illustrated in Figure 5.18, which shows residual AE variance components of ADHD as a

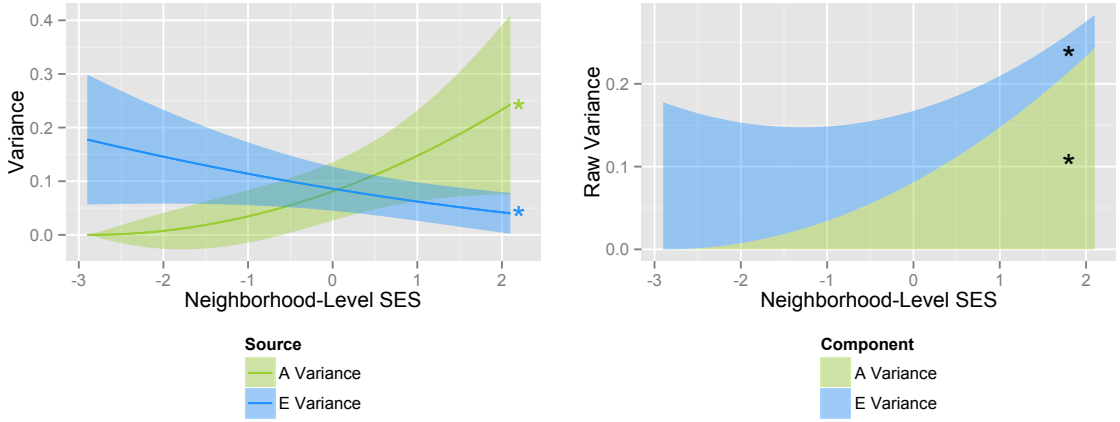


*Figure 5.18.* Gene-by-environment interaction between ADHD symptoms and neighborhood socioeconomic advantage in the Add Health sample. The left panel shows AE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in ADHD symptoms increases as a function of increasing neighborhood-level SES. The asterisks (*) indicate that variance in ADHD symptomatology depends on level of neighborhood socioeconomic advantage.

function of neighborhood-level SES, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure clearly demonstrates how residual phenotypic and additive genetic variance in ADHD increases with increasing neighborhood-level SES. It seems that high-SES environments reduce environmental risk for ADHD, but contribute to genetic risk for ADHD.

These results are further illustrated in Figure 5.19. As we observed with parental education, total variance in ADHD increases as a function of increasing neighborhood socioeconomic advantage. Consistent with increasing additive genetic variance, the gap between the MZ and DZ regression lines (left panel of Figure 5.19) increases as a function of neighborhood SES, an effect apparently driven by DZ twins growing more dissimilar. Consistent with decreasing nonshared environmental variance, the MZ regression line has a negative slope. The violin plots (right panel of Figure 5.19) show

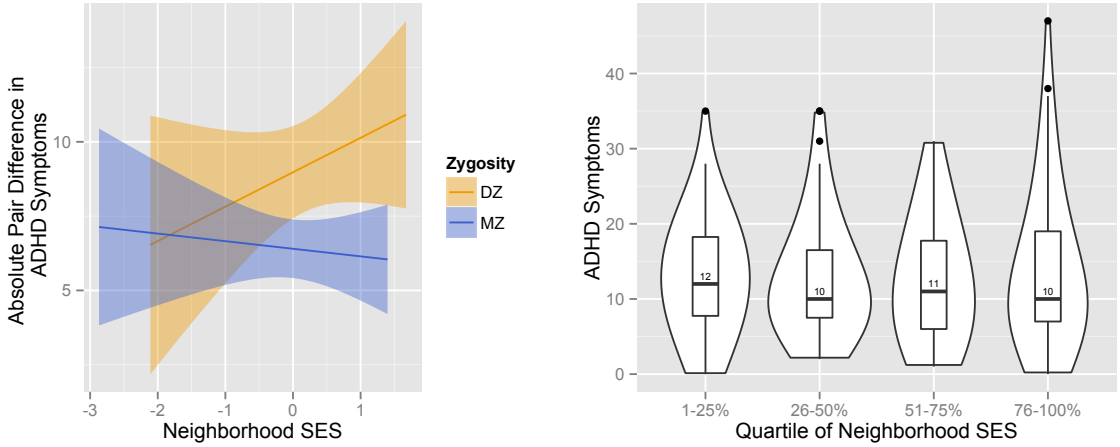


*Figure 5.19.* Illustrative analysis of the effects of neighborhood socioeconomic advantage on variance in ADHD in the Add Health sample. The left panel shows absolute pair differences in ADHD as a function of neighborhood SES. The distance between the MZ and DZ lines represents the additive genetic variance in ADHD. The location of the MZ line represents nonshared environmental variance in ADHD. The right panel shows box plots overlaid with violin plots, ADHD as a function of quartile of neighborhood SES.

that this expanding variance effect appears to be the result of more observations at the higher end of ADHD when neighborhood SES is high.

**Brief Summary.** Parental education was related to mean levels of depression such that higher SES was related to fewer symptoms of ADHD. Parental income and neighborhood-level SES showed similar trends, although their effects on ADHD did not reach statistical significance. In summary, it appears that both compositional and symptoms of ADHD. The findings regarding the impact of SES on residual variance in contextual measures of SES may confer some protection against experiencing ADHD was mixed. Parental education predicted *increased* nonshared environmental variance in ADHD, while neighborhood-level SES predicted *decreased* nonshared environmental variance in ADHD and increased additive genetic variance.

***ADHD—WSTR.*** Attention-deficit/hyperactivity disorder in the WSTR was

measured using a single binary item assessing whether the individual had ever received a diagnosis of ADHD by a physician (see Chapter 3). We fit the model presented in Figure 5.20 to the data separately for each socioeconomic indicator, partialling for the effects of age and gender on level and variance in ADHD diagnosis. We used likelihood ratio tests to determine the best fitting model. We first established a baseline model by conducting omnibus tests for the moderation by SES of both the residual variance in ADHD diagnosis and the main effects of SES on ADHD diagnosis. We then systematically eliminated parameters from the baseline model to evaluate statistical significance of moderation parameters. Biometric decomposition of both the SES indicators and ADHD diagnosis are presented in Table 4.2, and parameter estimates for each of these models are presented in Table 5.4. The Area Deprivation Index was log-transformed to correct for positive skew, and the Gini Index was scaled by a factor of 100 to facilitate model convergence. These transformed values were used in all analyses in which the ADI or Gini Index was used as the indicator of contextual socioeconomic

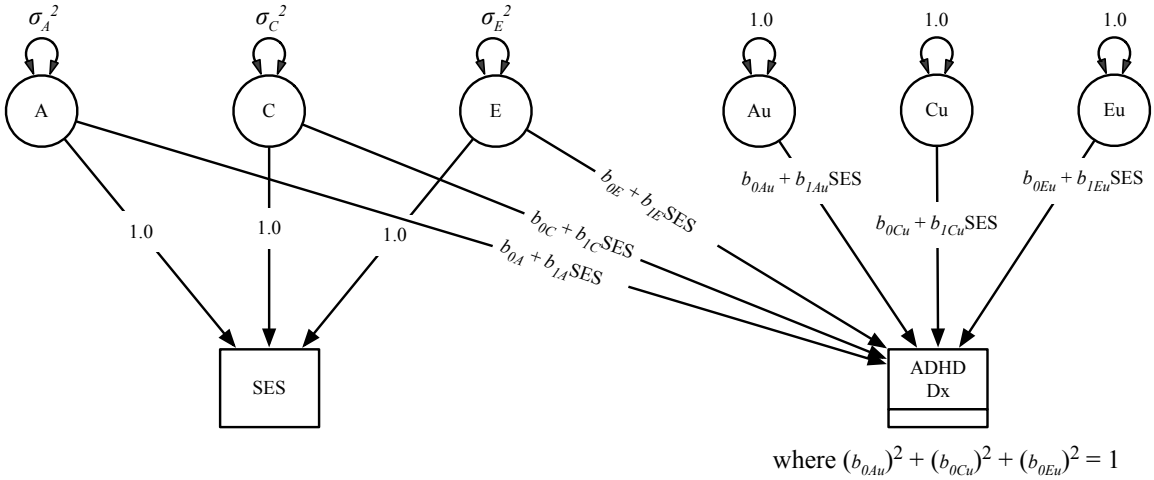


*Figure 5.20.* Path diagram G×SES model fit to ADHD diagnosis in the WSTR (only one twin shown for clarity).

Table 5.4. Parameter estimates and model fit statistics for G×SES models, ADHD diagnosis in the WSTR.

| | Parameter | Phenotypic Model | Quasi-Causal Model | Moderation of Residual Variance | Moderation of Main Effects | Best-Fitting Model |
|---|---|---|---|---|---|---|
| **Education** | Main Effect of Education on ADHD | | | | | |
| | $b_{0A}$ | **–.058 (–.114, –.015)** | .149 (–.189, .663) | .042 (–.469, 10.00) | –.282 (–4.294, .10.00) | .119 (–.305, 10.00) |
| | $b_{1A}$ | — | — | — | .062 (–10.00, 10.00) | — |
| | $b_{0C}$ | **–.058 (–.114, –.015)** | –.220 (–.812, .163) | **–.566 (–10.00, –.068)** | –.210 (–10.00, 2.459) | –.462 (–10.00, .026) |
| | $b_{1C}$ | — | — | — | –.058 (–.510, 10.00) | — |
| | $b_{0E}$ | **–.058 (–.114, –.015)** | **–.145 (–.318, –.035)** | **–.352 (–10.00, –.134)** | **–.610 (–10.00, –.002)** | **–.273 (–1.184, –.076)** |
| | $b_{1E}$ | — | — | — | .047 (–10.00, 10.00) | — |
| | Effect of Education on Residual ACE Components of ADHD | | | | | |
| | $b_{0Au}$ | **.981 (.223, 1.000)** | **.984 (.191, 1.000)** | .174 (–.931, .936) | .245 (–1.000, 1.000) | .924 (–1.000, 1.000) |
| | $b_{1Au}$ | — | — | .132 (–1.426, .024) | .133 (–.064, 10.00) | — |
| | $b_{0Cu}$ | .172 (–.965, .965) | .123 (–.928, .969) | .882 (–.990, 1.000) | .961 (–1.000, 1.000) | .238 (–1.000, 1.000) |
| | $b_{1Cu}$ | — | — | –.149 (–.856, .072) | –.130 (–10.00, 10.00) | — |
| | $b_{0Eu}$ | .092 (–.173, .497) | .129 (–.153, .570) | .437 (–1.000, 1.000) | .127 (–1.000, 1.000) | .299 (–.209, .736) |
| | $b_{1Eu}$ | — | — | –.144 (–1.124, .032) | –.075 (–10.00, .007) | **–.127 (–.462, –.031)** |
| | Model Fit | | | | | |
| | *-2LL* | — | 19771.750 | 19756.770 | 19751.360 | 19760.910 |
| | *Δ-2LL (Δdf)* | — | — | 14.979 (+3) | 5.414 (+3) | 4.133 (–2) |
| | *p* | — | — | .002† | .144 | .127 |
| **Household Income** | Main Effect of Income on ADHD | | | | | |
| | $b_{0A}$ | –.022 (–.064, .010) | .070 (–.639, 1.058) | .001 (–.616, .615) | | .070 (–.639, 1.058) |
| | $b_{1A}$ | — | — | — | | — |
| | $b_{0C}$ | –.022 (–.064, .010) | –.494 (–3.497, 1.113) | –.157 (–2.203, 1.006) | | –.494 (–3.497, 1.113) |
| | $b_{1C}$ | — | — | — | | — |
| | $b_{0E}$ | –.022 (–.064, .010) | .006 (–.117, .019) | .011 (–.089, .093) | | .006 (–.117, .019) |
| | $b_{1E}$ | — | — | — | | — |
| | Effect of Income on Residual ACE Components of ADHD | | | | | |
| | $b_{0Au}$ | .727 (–.073, 1.000) | .911 (–.998, 1.000) | .765 (–.071, 1.000) | | .911 (–.998, 1.000) |
| | $b_{1Au}$ | — | — | –.012 (–.093, .052) | | — |
| | $b_{0Cu}$ | .587 (–1.000, .980) | .492 (–.951, .991) | .544 (–1.000, .979) | | .492 (–.951, .991) |
| | $b_{1Cu}$ | — | — | –.015 (–.129, .085) | | — |
| | $b_{0Eu}$ | .356 (–.126, .897) | .435 (–.178, 1.000) | .345 (–.122, .977) | | .435 (–.178, 1.000) |
| | $b_{1Eu}$ | — | — | .013 (–.052, .114) | | — |
| | Model Fit | | | | | |
| | *-2LL* | — | 26155.890 | 26154.130 | | 26155.890 |
| | *Δ-2LL (Δdf)* | — | — | 1.760 (+3) | | — |
| | *p* | — | —† | .624 | | — |
| **Area Deprivation** | Main Effect of Area Deprivation on ADHD | | | | | |
| | $b_{0C}$ | .029 (–.493, .557) | –.104 (–.965, .709) | –.178 (–10.00, 10.00) | | –.104 (–.965, .709) |
| | $b_{1C}$ | — | — | — | | — |
| | $b_{0E}$ | .029 (–.493, .557) | .105 (–.441, .696) | .060 (–10.00, 10.00) | | .105 (–.441, .696) |
| | $b_{1E}$ | — | — | — | | — |
| | Effect of Area Deprivation on Residual ACE Components of ADHD | | | | | |
| | $b_{0Au}$ | .260 (–1.000, .907) | .731 (–.992, .992) | .134 (–1.000, 1.000) | | .731 (–.992, .992) |
| | $b_{1Au}$ | — | — | .010 (–10.00, .153) | | — |
| | $b_{0Cu}$ | .920 (–.993, .991) | .569 (–.298, .969) | .721 (-1.000, 1.000) | | .569 (–.298, .969) |
| | $b_{1Cu}$ | — | — | –.175 (–10.00, .153) | | — |
| | $b_{0Eu}$ | **.292 (.121, .898)** | **.276 (.081, .746)** | .680 (-1.000, 1.000) | | **.276 (.081, .746)** |
| | $b_{1Eu}$ | — | — | .159 (–10.00, .104) | | — |
| | Model Fit | | | | | |
| | *-2LL* | — | –2103.360 | –2108.197 | | –2103.360 |
| | *Δ-2LL (Δdf)* | — | — | 4.837 (+3) | | — |
| | *p* | — | —† | .184 | | — |
| **Income Inequality** | Main Effect of Income Inequality on ADHD | | | | | |
| | $b_{0A}$ | –.006 (–.020, .046) | 2.582 (–10.00, 10.00) | –.230 (–10.00, 10.00) | | 2.582 (–10.00, 10.00) |
| | $b_{1A}$ | — | — | — | | — |
| | $b_{0C}$ | –.006 (–.020, .046) | .045 (–.259, .141) | .089 (–10.00, 10.00) | | .045 (–.259, .141) |
| | $b_{1C}$ | — | — | — | | — |
| | $b_{0E}$ | –.006 (–.020, .046) | .013 (–.018, .058) | .012 (–.409, .713) | | .013 (–.018, .058) |
| | $b_{1E}$ | — | — | — | | — |
| | Effect of Income Inequality on Residual ACE Components of ADHD | | | | | |
| | $b_{0Au}$ | .846 (–1.000, 1.000) | .985 (–1.000, 1.000) | .535 (–1.000, 1.000) | | .985 (–1.000, 1.000) |
| | $b_{1Au}$ | — | — | .004 (–.003, 1.079) | | — |
| | $b_{0Cu}$ | .450 (–1.000, 1.000) | .119 (–1.000, 1.000) | .865 (–1.000, 1.000) | | .119 (–1.000, 1.000) |
| | $b_{1Cu}$ | — | — | –.015 (–.485, .485) | | — |
| | $b_{0Eu}$ | .288 (–.267, .828) | .131 (–.141, 1.000) | .079 (–1.000, 1.000) | | .131 (–.141, 1.000) |
| | $b_{1Eu}$ | — | — | –.005 (–.310, .004) | | — |
| | Model Fit | | | | | |
| | *-2LL* | — | 23542.840 | 23540.440 | | 23542.840 |
| | *Δ-2LL (Δdf)* | — | — | 2.402 (+3) | | — |
| | *p* | — | —† | .493 | | — |

*Note:* 95% confidence intervals presented within parentheses. Estimates $p < .05$ **bolded**. Baseline model denoted by †.

status. We did not include A for the ADI since there was no evidence of genetic influences on area deprivation in our univariate model.

**Causal Pathways vs. rGE: Main Effects of SES on ADHD Diagnosis (Dissertation Aim 1).** The main effects of SES on ADHD diagnosis are presented in Table 5.4 under the heading *Main Effect of Moderator on ADHD*. We discuss the main effects results for each moderator below.

**Education.** Education demonstrated a phenotypic association with ADHD diagnosis ($b$ = –0.058, 95% confidence interval = –0.114 to –0.015). Controlling for age and gender, individuals with a graduate or professional degree were 20% less likely to be diagnosed with ADHD than individuals with just a high school degree. The best-fitting model suggested that these associations are partially (but not entirely) mediated by shared environmental factors ($b_{0C}$ = –0.462, 95% confidence interval = –10.000 to –0.026). The quasi-causal association was also statistically significant ($b_{0E}$ = –0.273, 95% confidence interval = –1.184 to –0.076). The additive genetic regression was not statistically distinguishable from zero ($b_{0A}$ = 0.119, 95% confidence interval = –0.305 to 10.000). These results suggest that the protective effect of educational attainment on ADHD can be attributed to systematic differences in exposure to socioeconomic factors as well as shared environmental factors that are common to both phenotypes.

Because the inverse relationship between educational attainment and ADHD diagnosis can alternatively be explained by deficits in academic achievement that tend to be associated with ADHD (and therefore educational attainment; Daley & Birchwood, 2010), we elected to test the direction of causation (c.f. Duffy & Martin, 1994; Heath et

al., 1993) in the ADHD diagnosis–educational attainment association. The regression of educational attainment on ADHD diagnosis could be dropped from a bi-directional model without significant decrement in model fit ($p$ = 0.781); the regression of ADHD diagnosis on educational attainment could not be dropped ($p$ = 0.003). This analysis supported the direction of the association we have used in the present analysis.

As illustrated in the left panel of Figure 5.21, which shows the magnitude of the ACE regressions of ADHD diagnosis on educational attainment, it is evident that the shared environmental and nonshared environmental regressions both contribute to the phenotypic association. These regression lines are re-represented as genetic, shared environmental, and nonshared environmental correlations in the middle panel of Figure 5.21. We also present the proportions of the total phenotypic correlation accounted for by the ACE correlations in the right panel of Figure 5.21. Evident in this figure is that at low levels of education, the shared environmental correlation accounts for more than 60% of the total phenotypic correlation. At high levels of education, the nonshared environment appears to become more important to this association, accounting for approximately 40% of the correlation (while the shared environmental correlation accounts for approximately 50%) between ADHD diagnosis and educational attainment.

These phenotypic and within-family effects are demonstrated in Figure 5.22, an illustrative analysis of the main effect of educational attainment on ADHD diagnosis. We identified twin pairs concordant for lower education (up to Associate's degree; orange), pairs concordant for higher education (Bachelor's degree or higher; navy), and pairs discordant for educational status, and compared the proportion of individuals with a diagnosis of ADHD within each of these groups. Overall, there is a main effect of

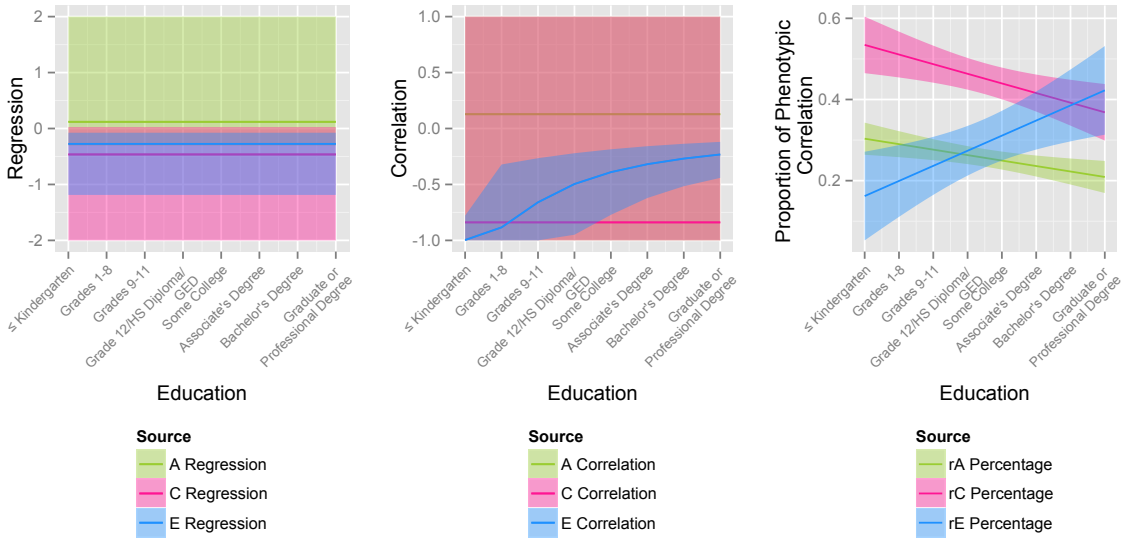


*Figure 5.21.* Main effects of educational attainment on ADHD diagnosis in the WSTR. The left panel shows the regression of ADHD diagnosis on educational attainment. The same relation is presented in the middle panel as genetic (green), shared environmental (pink), and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic, shared environmental, and nonshared environmental correlations. Asterisks (*) indicate that the magnitude of the regression of ADHD diagnosis on educational attainment depends on level of education (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). Shaded regions represent 95% confidence intervals around regression lines.

educational attainment on ADHD diagnosis such that higher education is associated with reduced likelihood of carrying an ADHD diagnosis (this is evident comparing the outer bars in this figure). Consistent with a causal effect of educational attainment on ADHD diagnosis, examination of this relation at the within-pair level (comparing the inner bars for MZ and DZ twins) shows that it exists within pairs of MZ and DZ twins.

**Income.** The phenotypic regression of ADHD diagnosis on income was not observed to be statistically significant ($b$ = –0.022, 95% confidence interval = –0.064 to 0.010).

**Area Deprivation.** The ADI was not statistically significantly associated with

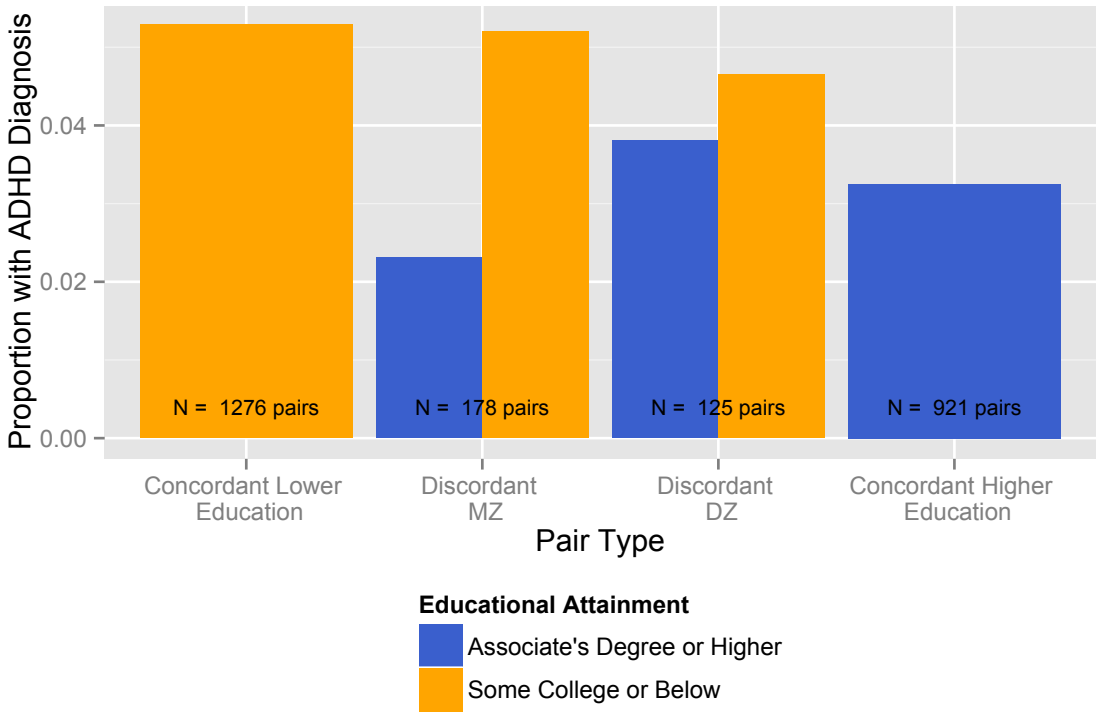


*Figure 5.22.* Illustrative analysis of the main effects of educational attainment on ADHD diagnosis in the WSTR. The less educated twin (orange) is compared with the more educated twin (navy) in MZ and DZ twin pairs.

ADHD diagnosis ($b$ = 0.029, 95% confidence interval = –0.493 to 0.557).

**Income Inequality.** Income inequality showed no evidence of a main effect on ADHD diagnosis ($b$ = –0.006, 95% confidence interval = –0.020 to 0.046).

**G×E Interaction: Effect of SES on Genetic and Environmental Etiology of ADHD Diagnosis (Dissertation Aim 2).** The moderating effects of SES on variance in ADHD diagnosis are presented in Table 5.4 under the heading *Effect of SES on Residual ACE Components of ADHD.* We discuss the interactive effect of each SES indicator on variance in ADHD diagnosis below.

**Education.** The best fitting model suggested that residual phenotypic variance in ADHD diagnosis increased with increasing educational attainment. This effect was

driven by increasing nonshared environmental variance ($b_{1Eu}$ = –0.127, 95% confidence interval = –0.462 to –0.031); there was no evidence that the additive genetic or shared environmental variances depended on level of education. These model results are illustrated in Figure 5.23, which shows residual ACE variance components of ADHD diagnosis as a function of educational attainment, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure clearly demonstrates how residual phenotypic and nonshared environmental variance in ADHD diagnosis increases with increasing educational attainment. Considered within the context of the dynamics of the ACE correlations, it appears that the presence of factors contributing to ADHD depends on level of education, suggesting that education is acting to potentiate nonshared environmental variation in ADHD diagnosis. As we noted at the beginning of this section, the variance in ADHD diagnosis is scaled according to the intercepts (*i.e.*, the intercepts of the residual ACE variances in ADHD diagnosis sum to unity); an alternative explanation for these model results is that high-SES environments protect against additive genetic risk for ADHD (because heritability is decreasing as a function of SES).

These model results are illustrated in Figure 5.24, which shows MZ and DZ twin correlations in ADHD diagnosis as a function of pair-average educational attainment. Both the MZ and DZ correlations decrease with respect to increasing educational attainment, indicating that MZ and DZ twins are less similar on this phenotype at higher levels of socioeconomic status. In addition, the decreasing correlations are consistent with our observation that nonshared environmental influences on ADHD diagnosis increase with respect to increasing educational attainment.

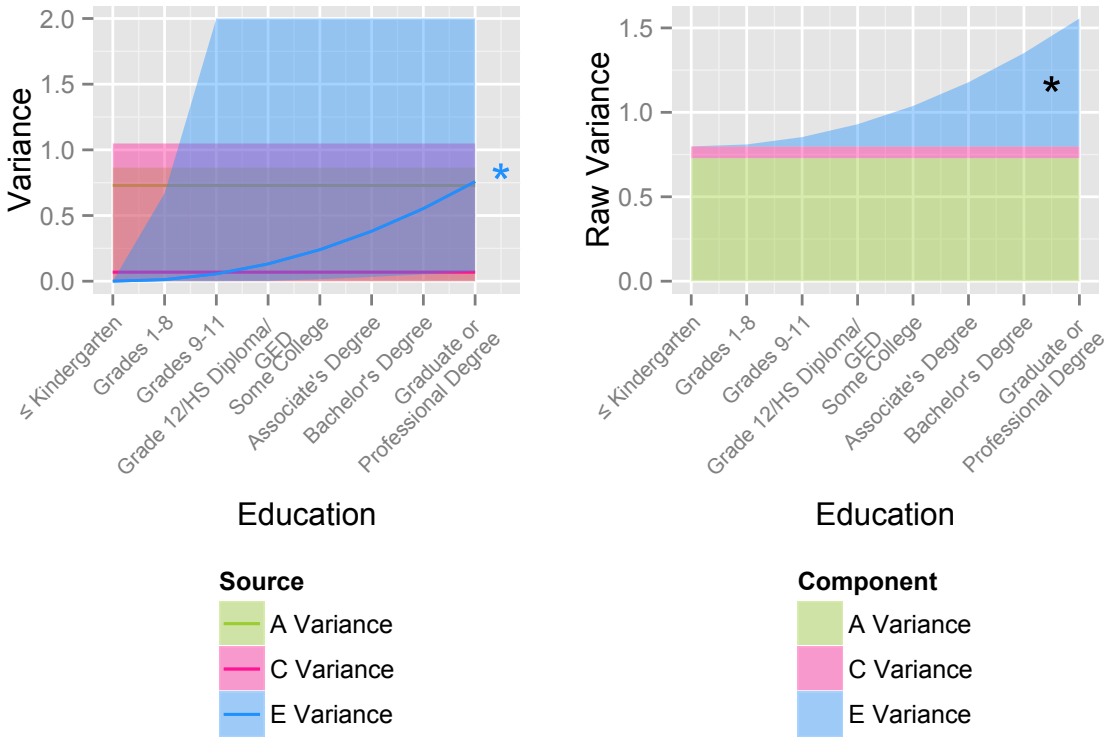


*Figure 5.23.* Gene-by-environment interaction between ADHD diagnosis and educational attainment in the WSTR. The left panel shows ACE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in ADHD diagnosis increases as a function of increasing educational attainment. The asterisks (*) indicate that variance in ADHD diagnosis depends on level of education.

**Income.** The best-fitting model suggested that variance in ADHD diagnosis does not vary as a function of household income.

**Area Deprivation.** The best fitting model suggested that residual phenotypic variance in ADHD diagnosis does not depend on area deprivation.

**Income Inequality.** The best fitting model suggested that residual phenotypic variance in ADHD diagnosis is stable with respect to county-level income inequality.

**Brief Summary.** Educational attainment protected against having received a diagnosis of ADHD by a physician; both shared and nonshared environmental factors

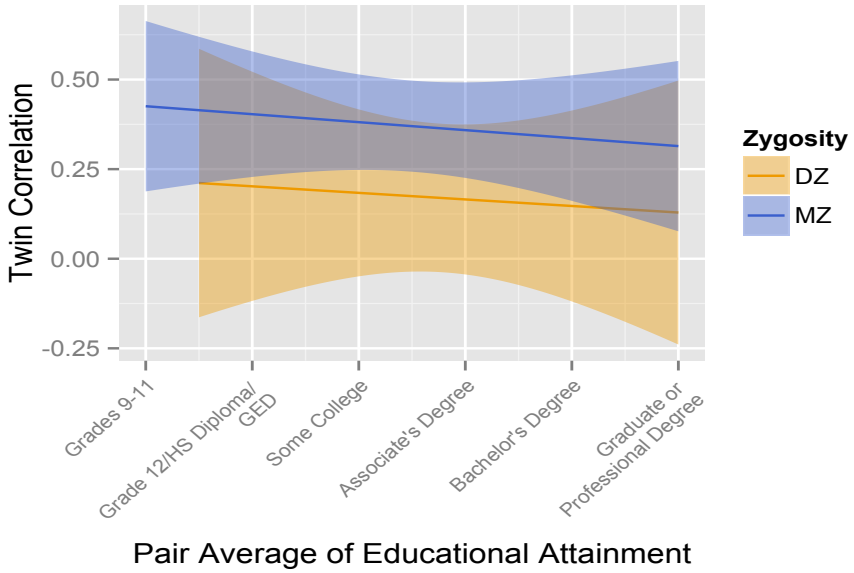


*Figure 5.24.* Illustrative analysis of the effects of educational attainment on MZ and DZ twin correlations in ADHD diagnosis in the WSTR. MZ and DZ twin correlations are plotted as a function of the average education level achieved by twin pairs.

contributed to this association. Contextual measures of socioeconomic status were not found to be related to likelihood of having an ADHD diagnosis. Residual variance in internalizing was also influenced by educational attainment. Residual phenotypic variance increased with increasing educational attainment, an effect driven primarily by increases in nonshared environmental variance. This increase appeared to be potentiated by socioeconomic status, and not by social selection factors.

***Delinquency—Add Health.*** Delinquency in the Add Health sample was measured using 15 items tapping engagement in various illegal behaviors (see Chapter 3). We fit the model presented in Figure 5.25 to the data separately for each socioeconomic status indicator, partialling for the linear effects of age and gender on level and variance in the delinquency factor. We used likelihood ratio tests to determine the best fitting model. Biometric decomposition of both the SES indicators and the delinquency factor

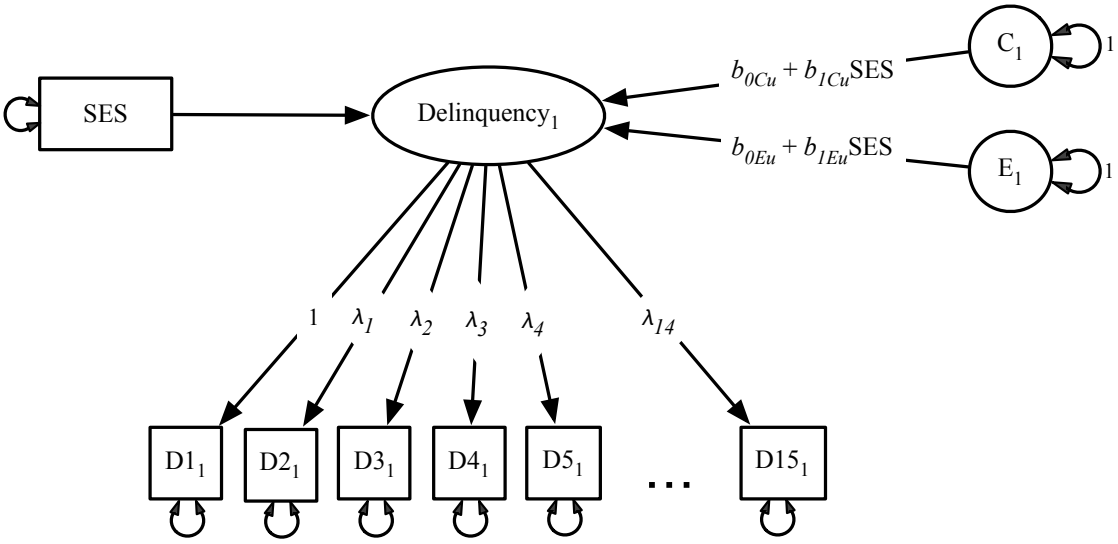


*Figure 5.25.* Path diagram G×SES model fit to delinquency in the Add Health sample (only one twin shown for clarity). The residual variances for the delinquency items were permitted to correlate across twins, and were estimated freely according to zygosity.

are presented in Table 4.1, and parameter estimates for each of these models are presented in Table 5.5. Family household income was log-transformed to correct for positive skew. The latent delinquency factor showed no evidence of additive genetic influence; therefore, we have not included A in the models for this phenotype.

**Main Effects of SES on Delinquency (Dissertation Aim 1).** The main effects of SES on delinquency are presented in Table 5.5 under the heading *Main Effect of Moderator on Delinquency*. We discuss the main effects results for each moderator below.

**Parental Education.** Parental education was not statistically significantly associated with mean levels of delinquency ($b = 0.000$, $p = 0.941$).

**Family Income.** Delinquency was not associated with family household income($b = –0.003$, $p = 0.742$) in the phenotypic model, although showed a significant negative association when residual variance in delinquency was freed to vary with family

Table 5.5. Parameter estimates and model fit statistics for G×SES models, delinquency in the Add Health sample.

| | Parameter | Phenotypic Model | Moderation of Residual Variance | Best-Fitting Model |
|---|---|---|---|---|
| **Parental Education** | Main Effect of Parental Education on Delinquency | | | |
| | $b_1$ | .000 (.005) | .001 (.005) | .000 (.005) |
| | Effect of Education on Residual ACE Components of Delinquency | | | |
| | $b_{0Cu}$ | **.338 (.155)** | ***.347 (.122)*** | **.338 (.155)** |
| | $b_{1Cu}$ | — | .001 (.006) | — |
| | $b_{0Eu}$ | ***.910 (.112)*** | ***.501 (.111)*** | ***.910 (.112)*** |
| | $b_{1Eu}$ | — | ***.065 (.005)*** | — |
| | Model Fit | | | |
| | *-2LL* | 22286.094 | 22284.782 | 22286.094 |
| | *Δ-2LL (Δdf)* | — | 1.312 (+2) | — |
| | *p* | —† | .519 | — |
| **Family Household Income** | Main Effect of Family Income on Delinquency | | | |
| | $b_1$ | –.003 (.008) | **–.039 (.016)** | **–.039 (.016)** |
| | Effect of Family Income on Residual ACE Components of Delinquency | | | |
| | $b_{0Cu}$ | .239 (.155) | **.698 (.176)** | **.698 (.176)** |
| | $b_{1Cu}$ | — | ***–.089 (.021)*** | ***–.089 (.021)*** |
| | $b_{0Eu}$ | ***.922 (.115)*** | ***.950 (.122)*** | ***.950 (.122)*** |
| | $b_{1Eu}$ | — | ***–.037 (.013)*** | ***–.037 (.013)*** |
| | Model Fit | | | |
| | *-2LL* | 19354.610 | 19320.786 | 19320.786 |
| | *Δ-2LL (Δdf)* | — | 33.824 (+2) | — |
| | *p* | — | <.001† | — |
| **Neighborhood Socioeconomic Advantage** | Main Effect of Neighborhood SES on Delinquency | | | |
| | $b_1$ | .002 (.011) | .000 (.012) | .002 (.011) |
| | Effect of Area Deprivation on Residual ACE Components of Delinquency | | | |
| | $b_{0Cu}$ | .045 (.142) | .052 (.153) | .045 (.142) |
| | $b_{1Cu}$ | — | .023 (.020) | — |
| | $b_{0Eu}$ | ***.814 (.116)*** | ***.817 (.114)*** | ***.814 (.116)*** |
| | $b_{1Eu}$ | — | .015 (.009) | — |
| | Model Fit | | | |
| | *-2LL* | 19076.228 | 19071.300 | 19076.228 |
| | *Δ-2LL (Δdf)* | — | 4.928 (+2) | — |
| | *p* | —† | .085 | — |

*Note:* Standard errors presented within parentheses. Estimates p < .05 **bolded**. Estimates p < .01 *italicized*. Baseline model denoted by †.

income ($b$ = –0.039, $p$ = 0.013). Adolescents living with a household income at the first quartile ($20,000/year) had delinquency scores that were approximately 0.11 standard deviations higher than adolescents living with a household income at the third quartile ($56,000/year).

**Neighborhood Socioeconomic Advantage.** Neighborhood-level SES was not associated with mean levels of delinquency ($b$ = 0.002, $p$ = 0.836).

**G×E Interaction: Effect of SES on Genetic and Environmental Etiology of Delinquency.** The moderating effects of SES on variance in delinquency are presented in Table 5.5 under the heading *Effect of SES on Residual ACE Components of Delinquency.*

**Parental Education.** The best-fitting model suggested that residual phenotypic variance in delinquency was not associated with parental educational attainment.

**Family Income.** The best-fitting model suggested that residual variance in delinquency decreased as a function of increasing family income, an effect driven by decreases in both shared environmental ($b_{1Cu}$ = –0.089, $p$ < 0.001) and nonshared environmental ($b_{1Eu}$ = –0.037, $p$ = 0.005) variances. These model results are illustrated in Figure 5.26, which shows residual CE variance components of delinquency as a function of family income, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure clearly demonstrates how residual phenotypic and shared/nonshared environmental variance in delinquency decreases with increasing family income. It seems that high-SES environments protect against environmental risk for engaging in delinquent behavior.

To illustrate these results, we plotted absolute pair differences in delinquency for MZ and DZ twins against family household income (see the left panel of Figure 5.27). Evident in this plot is that the slope of the MZ regression line is negative, consistent with both nonshared environmental variance decreasing as a function of family income. The violin plot in the right panel of Figure 5.27 illustrates both an overall protective effect of family income on delinquency, as well as decreased variance in delinquency with increasing family income. Notably, this decrease in variance appears to be due to fewer

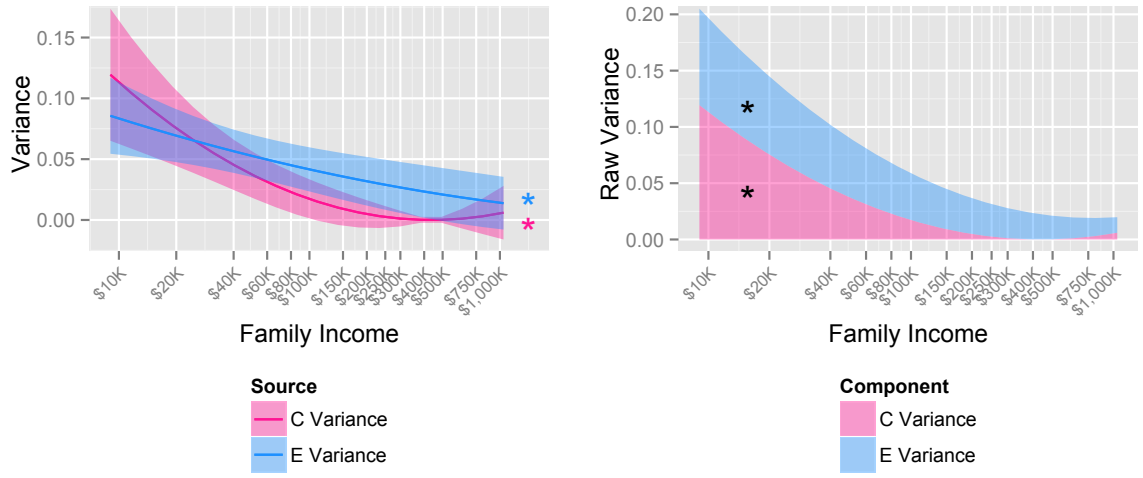


*Figure 5.26.* Gene-by-environment interaction between delinquency and family income in the Add Health sample. The left panel shows CE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in delinquency decreases as a function of increasing family income. The asterisks (*) indicate that variance in delinquency depends on family income.

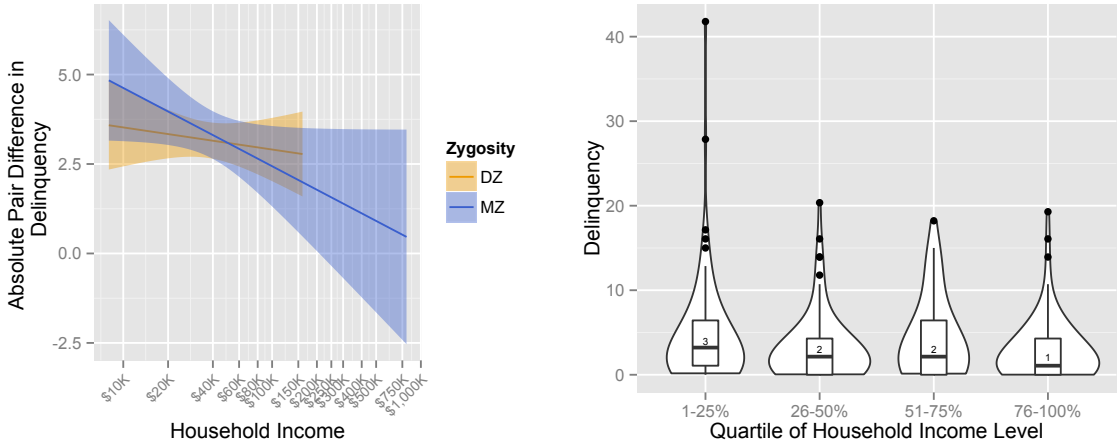


*Figure 5.27.* Illustrative analysis of the effects of family income on variance in delinquency in the Add Health sample. The left panel shows absolute pair differences in delinquency as a function of family income. The distance between the MZ and DZ lines represents the additive genetic variance in delinquency. The location of the MZ line represents nonshared environmental variance in delinquency. The right panel shows box plots overlaid with violin plots, delinquency as a function of quartile of family income.

observations in the upper tail of the delinquency distribution at high levels of family income.

**Neighborhood Socioeconomic Advantage.** Like parental education, neighborhood-level SES was not a predictor of residual variance in delinquency in this sample.

**Brief Summary.** In general, socioeconomic status (both compositional and contextual) was unrelated to mean levels or variance in delinquency. The exception was family income, which appeared to protect against engaging in delinquent behaviors and reduced shared and nonshared environmental risk for delinquency.

**Neuroticism Traits**

***Neuroticism—Add Health.*** Neuroticism in the Add Health sample was measured using nine items from the NEO-PI administered to respondents at Wave IV (see Chapter 3). We fit the model presented in Figure 5.28 to the data separately for each

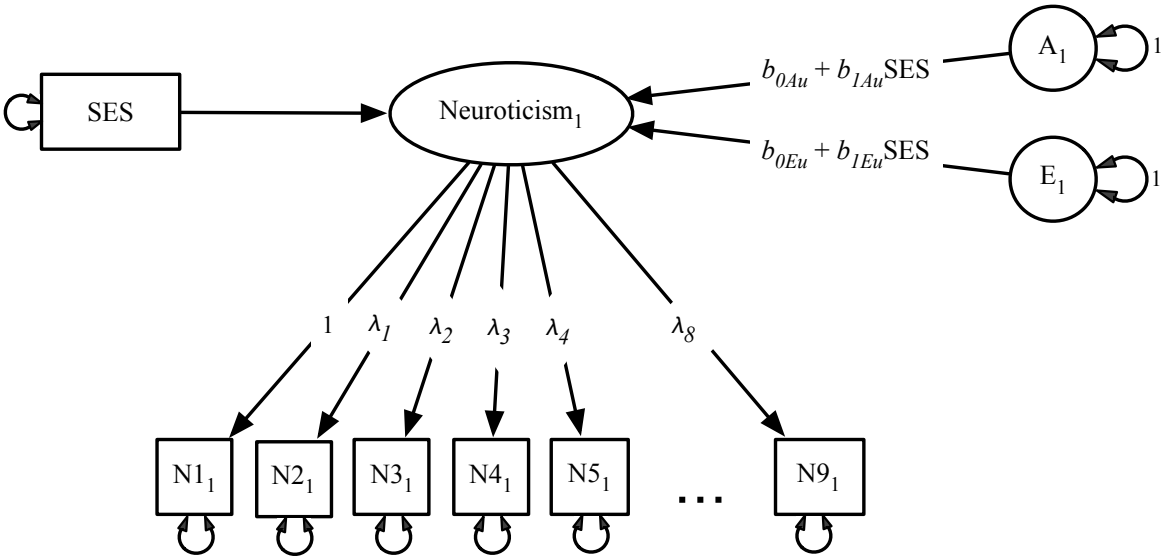


*Figure 5.28.* Path diagram G×SES model fit to neuroticism in the Add Health sample (only one twin shown for clarity). The residual variances for the neuroticism items were permitted to correlate across twins, and were estimated freely according to zygosity.

socioeconomic status indicator, partialling for the linear effects of age and gender on level and variance in the neuroticism factor. We used likelihood ratio tests to determine the best fitting model. Biometric decomposition of both the SES indicators and the neuroticism factor are presented in Table 4.1, and parameter estimates for each of these models are presented in Table 5.6. Family household income was log-transformed to correct for positive skew. We did not include C for the neuroticism factor because there was no evidence of shared environmental influences on neuroticism in our univariate model.

**Main Effects of SES on Neuroticism (Dissertation Aim 1).** The main effects of SES on neuroticism are presented in Table 5.6 under the heading *Main Effect of Moderator on Neuroticism*. None of the socioeconomic indicators were related to mean levels of this phenotype (parental education: $b = –0.003$, $p = 0.894$; family household income: $b = –0.013$, $p = 0.499$; neighborhood socioeconomic advantage: $b = 0.007$, $p = 0.839$).

**G×E Interaction: Effect of SES on Genetic and Environmental Etiology of Neuroticism (Dissertation Aim 2).** The moderating effects of SES on variance in neuroticism are presented in Table 5.6 under the heading *Effect of SES on Residual ACE Components of Neuroticism.* The best fitting model for each SES indicator suggested that variance in neuroticism is static with respect to socioeconomic status.

**Brief Summary.** In general, socioeconomic status was not observed to be related to mean levels or neuroticism, nor with variance in neuroticism.

***Neuroticism—WSTR.*** Neuroticism in the WSTR sample was measured using nine items from the IPIP (see Chapter 3). We fit the model presented in Figure 5.29 to

Table 5.6. Parameter estimates and model fit statistics for G×SES models, neuroticism in the Add Health sample.

| | Parameter | Phenotypic Model (No Moderation) | Moderation of Residual Variance | Best-Fitting Model |
|---|---|---|---|---|
| **Parental Education** | Main Effect of Parental Education on Neuroticism | | | |
| | $b_1$ | –.003 (.022) | –.006 (.022) | –.003 (.022) |
| | Effect of Education on Residual ACE Components of Neuroticism | | | |
| | $b_{0Au}$ | **1.834 (.789)** | 1.932 (2.166) | **1.834 (.789)** |
| | $b_{1Au}$ | — | –.037 (.058) | — |
| | $b_{0Eu}$ | .488 (.440) | **1.152 (.567)** | .488 (.440) |
| | $b_{1Eu}$ | — | –.001 (.021) | — |
| | Model Fit | | | |
| | *-2LL* | 22288.852 | 22289.068 | 22288.852 |
| | *Δ-2LL (Δdf)* | — | .216 (+2) | — |
| | *p* | —† | .898 | — |
| **Family Household Income** | Main Effect of Family Income on Neuroticism | | | |
| | $b_1$ | –.013 (.019) | –.008 (.018) | –.013 (.019) |
| | Effect of Family Income on Residual ACE Components of Neuroticism | | | |
| | $b_{0Au}$ | **2.501 (1.324)** | **2.555 (1.269)** | **2.501 (1.324)** |
| | $b_{1Au}$ | — | .007 (.018) | — |
| | $b_{0Eu}$ | **1.225 (.573)** | **1.198 (.584)** | **1.225 (.573)** |
| | $b_{1Eu}$ | — | ***.036 (.014)*** | — |
| | Model Fit | | | |
| | *-2LL* | 19847.746 | 19844.008 | 19847.746 |
| | *Δ-2LL (Δdf)* | — | 3.738 (+2) | — |
| | *p* | —† | .154 | — |
| **Neighborhood Socioeconomic Advantage** | Main Effect of Neighborhood SES on Neuroticism | | | |
| | $b_1$ | .007 (.035) | .008 (.036) | .007 (.035) |
| | Effect of Neighborhood SES on Residual ACE Components of Neuroticism | | | |
| | $b_{0Au}$ | .299 (1.678) | .714 (1.632) | .299 (1.678) |
| | $b_{1Au}$ | — | .044 (.076) | — |
| | $b_{0Eu}$ | 1.032 (.790) | .869 (.785) | 1.032 (.790) |
| | $b_{1Eu}$ | — | –.030 (.040) | — |
| | Model Fit | | | |
| | *-2LL* | 19078.714 | 19078.186 | 19078.714 |
| | *Δ-2LL (Δdf)* | — | .528 (+2) | — |
| | *p* | —† | .768 | — |

*Note:* Standard errors presented within parentheses. Estimates p < .05 **bolded**. Estimates p < .01 *italicized*.

the data separately for each socioeconomic status indicator, partialling for the linear effects of age and gender on level and variance in the neuroticism factor. We used likelihood ratio tests to determine the best fitting model. Biometric decomposition of both the SES indicators and the neuroticism factor are presented in Table 4.2, and parameter estimates for each of these models are presented in Table 5.7. The Area Deprivation Index was log-transformed to correct for positive skew, and the Gini Index

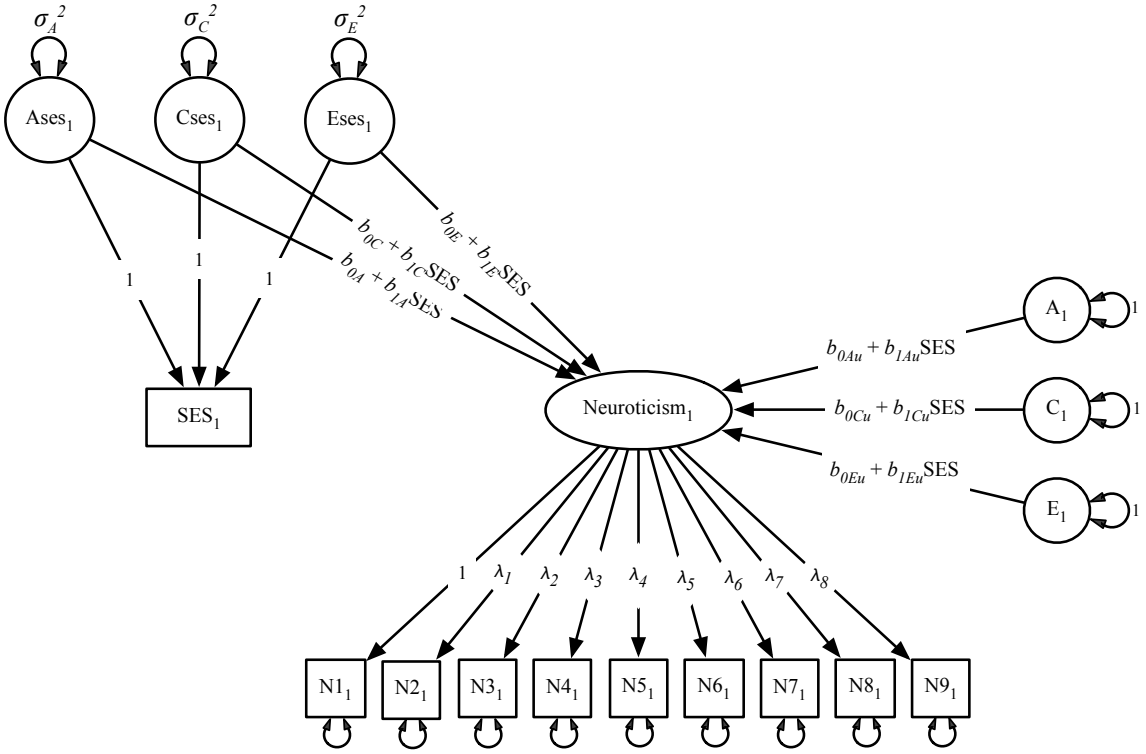


*Figure 5.29.* Path diagram G×SES model fit to neuroticism in the WSTR (only one twin shown for clarity). In the fully saturated model, the main effects of SES on neuroticism and the ACE variances of neuroticism vary as a function of SES. The residual variances for the neuroticism items were permitted to correlate across twins, and were estimated freely according to zygosity.

was scaled by a factor of 10 to facilitate model convergence. These transformed values were used in all analyses in which the ADI or Gini Index was used as the indicator of contextual socioeconomic status. We did not include C for the neuroticism factor because there was no evidence of shared environmental influences on neuroticism in our univariate model. Likewise, we did not include A for the ADI since there was no evidence of genetic influences on area deprivation in our univariate model.

**Causal Pathways vs. rGE: Main Effects of SES on Neuroticism (Dissertation Aim 1).** The main effects of SES on neuroticism are presented in Table 5.7 under the heading *Main Effect of Moderator on Neuroticism*. We discuss the main effects results

Table 5.7. Parameter estimates and model fit statistics for G×SES models, neuroticism in the WSTR.

| | Parameter | Phenotypic Model | Quasi-Causal Model (No Moderation) | Moderation of Residual Variance |
|---|---|---|---|---|
| **Education** | Main Effect of Education on Neuroticism | | | |
| | $b_{0A}$ | ***–.076 (.011)*** | ***–.149 (.029)*** | ***–.149 (.029)*** |
| | $b_{1A}$ | — | — | — |
| | $b_{0E}$ | ***–.076 (.011)*** | –.027 (.018) | –.027 (.018) |
| | $b_{1E}$ | — | — | — |
| | Effect of Education on Residual ACE Components of Neuroticism | | | |
| | $b_{0Au}$ | ***.535 (.056)*** | ***.526 (.056)*** | ***.471 (.074)*** |
| | $b_{1Au}$ | — | — | .020 (.012) |
| | $b_{0Eu}$ | ***.607 (.042)*** | ***.609 (.042)*** | ***.646 (.065)*** |
| | $b_{1Eu}$ | — | — | .008 (.008) |
| | Model Fit | | | |
| | *-2LL* | — | 163438.890 | 163435.688 |
| | *Δ-2LL (Δdf)* | — | — | 3.202 (+2) |
| | *p* | — | —† | .202 |
| **Household Income** | Main Effect of Income on Neuroticism | | | |
| | $b_{0A}$ | ***–.028 (.005)*** | ***–.065 (.018)*** | **–.067 (.019)** |
| | $b_{1A}$ | — | — | — |
| | $b_{0E}$ | ***–.028 (.005)*** | –.012 (.023) | –.011 (.008) |
| | $b_{1E}$ | — | — | — |
| | Effect of Income on Residual ACE Components of Neuroticism | | | |
| | $b_{0Au}$ | ***.554 (.056)*** | ***.554 (.057)*** | ***.493 (.063)*** |
| | $b_{1Au}$ | — | — | **–.018 (.009)** |
| | $b_{0Eu}$ | ***.611 (.062)*** | ***.606 (.043)*** | ***.660 (.050)*** |
| | $b_{1Eu}$ | — | — | –.005 (.007) |
| | Model Fit | | | |
| | *-2LL* | — | 163308.930 | 163303.052 |
| | *Δ-2LL (Δdf)* | — | — | 5.878 (+2) |
| | *p* | — | —† | .053 |
| **Area Deprivation** | Main Effect of Area Deprivation on Neuroticism | | | |
| | $b_{0E}$ | | **–.305 (.138)** | **–.307 (.135)** |
| | $b_{1E}$ | | — | — |
| | Effect of Area Deprivation on Residual ACE Components of Neuroticism | | | |
| | $b_{0Au}$ | | ***.574 (.075)*** | .468 (.631) |
| | $b_{1Au}$ | | — | .022 (.134) |
| | $b_{0Eu}$ | | ***.563 (.056)*** | ***1.223 (.101)*** |
| | $b_{1Eu}$ | | — | –.142 (.111) |
| | Model Fit | | | |
| | *-2LL* | | 72930.364 | 72928.224 |
| | *Δ-2LL (Δdf)* | | — | 2.140 (+2) |
| | *p* | | —† | .343 |
| **Income Inequality** | Main Effect of Income Inequality on Neuroticism | | | |
| | $b_{0A}$ | .006 (.049) | –.227 (.507) | –.204 (.501) |
| | $b_{1A}$ | — | — | — |
| | $b_{0E}$ | .006 (.049) | .024 (.061) | .024 (.061) |
| | $b_{1E}$ | — | — | — |
| | Effect of Income Inequality on Residual ACE Components of Neuroticism | | | |
| | $b_{0Au}$ | ***.552 (.061)*** | ***.552 (.061)*** | **.403 (.179)** |
| | $b_{1Au}$ | — | — | .014 (.040) |
| | $b_{0Eu}$ | ***.586 (.047)*** | ***.586 (.047)*** | ***.700 (.181)*** |
| | $b_{1Eu}$ | — | — | **–.081 (.040)** |
| | Model Fit | | | |
| | *-2LL* | — | 118679.564 | 118676.426 |
| | *Δ-2LL (Δdf)* | — | — | 3.138 (+2) |
| | *p* | — | —† | .208 |

*Note:* Standard errors presented within parentheses. Estimates p < .05 **bolded**. Estimates p < .01 *italicized*. Best-fitting model denoted by †.

for each moderator below.

**Education.** The phenotypic regression of neuroticism on education level showed a significant negative relationship ($b$ = –0.076, $p$ < 0.001). That is, controlling for age and gender, individuals with a graduate or professional degree had a score on the neuroticism factor that was 0.42 standard deviations lower than individuals with just a high school degree. The best-fitting model suggested genetic mediation of this association ($b_{0A}$ = –0.149, $p$ < 0.001); the quasi-causal pathway for the regression of neuroticism on educational attainment was substantially reduced in magnitude ($b_{0E}$ = –0.027, $p$ = 0.133) relative to the phenotypic effect and was no longer statistically significant. These results indicate that the genes contributing to educational attainment are also the same genes influencing neuroticism traits, meaning that the relation between education and neuroticism is best explained by underlying genetic factors that are common to both phenotypes rather than due to systematic differences in exposure to socioeconomic factors. These results are presented in Figure 5.30. The genetic correlation between educational attainment and neuroticism (green line) is much larger in magnitude than the nonshared environmental correlation (blue line), and accounts for nearly eight times as much of the total phenotypic correlation.

To demonstrate what these effects look like within and between twins, we conducted an illustrative analysis of the main effect of educational attainment on neuroticism, which we present in Figure 5.31. We identified twin pairs concordant for lower education (up to Associate's degree; orange), pairs concordant for higher education (Bachelor's degree or higher; navy), and pairs discordant for educational status, and compared mean neuroticism scores (using a summed score of the IPIP neuroticism items)

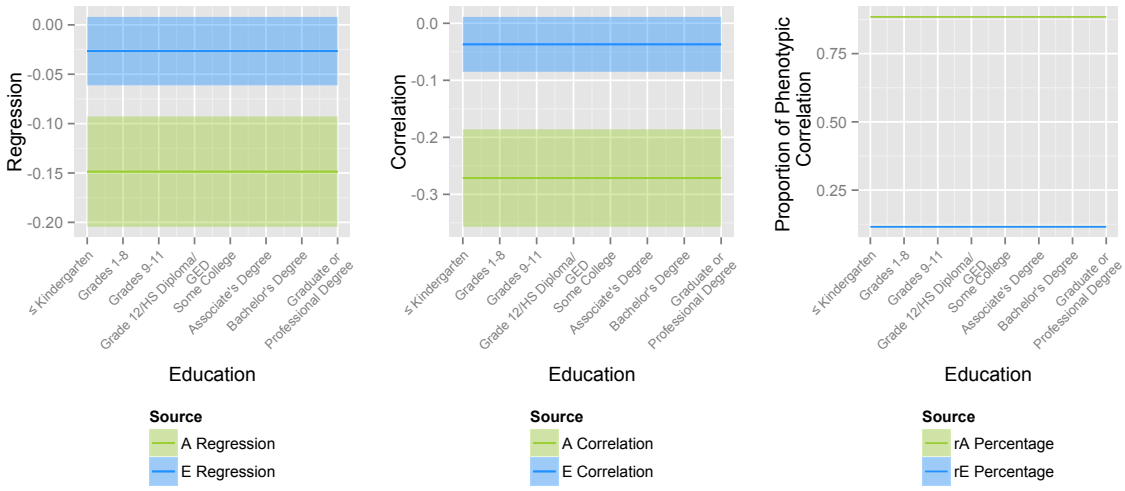


*Figure 5.30.* Main effects of educational attainment on neuroticism in the WSTR. The left panel shows the regression of neuroticism on educational attainment. The same relation is presented in the middle panel as genetic (green) and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic and nonshared environmental correlations. The shaded regions represent 95% confidence intervals around the regression lines.

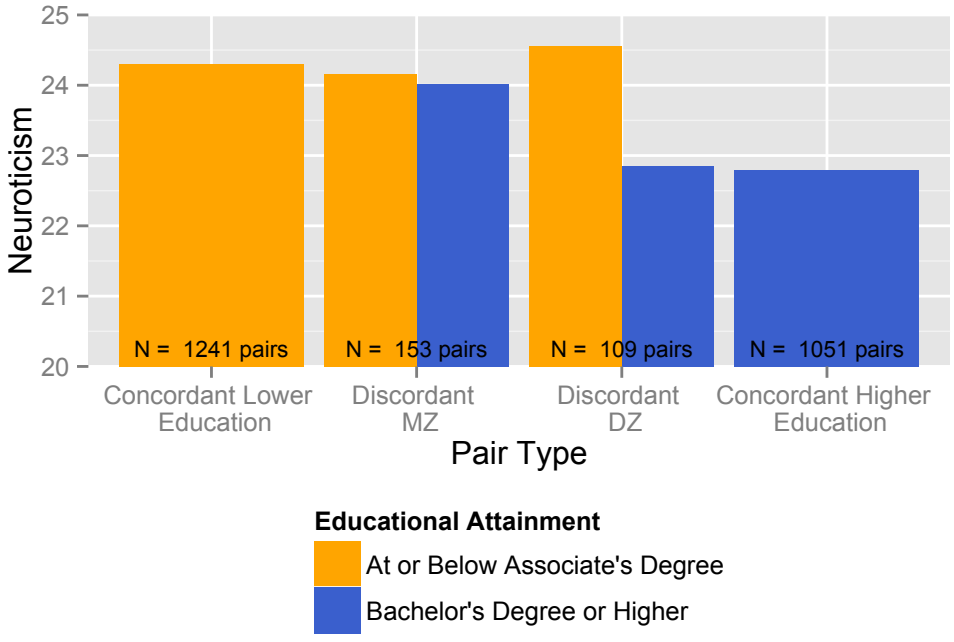


*Figure 5.31.* Illustrative analysis of the main effects of educational attainment on neuroticism in the WSTR. The less educated twin (orange) is compared with the more educated twin (navy) in MZ and DZ twin pairs.

of each of these groups. Overall, there is a main effect of educational attainment on neuroticism such that higher education is associated with fewer neuroticism traits on average (this is evident comparing the outer bars in this figure). Examining the relation at the within-pair level, however, shows that it does not exist within pairs of MZ twins. Consistent with genetic selection, the difference in neuroticism scores within DZ twin pairs is larger than within MZ twin pairs.

**Income.** The phenotypic regression of neuroticism on household income showed a significant negative relationship ($b$ = –0.028, $p$ < 0.001). That is, controlling for age and gender, individuals at the third quartile of earned income scored an average of 0.19 standard deviations lower on the neuroticism factor than their counterparts at the first quartile of earned income. Results from the best-fitting model mirrored those using educational attainment to predict neuroticism: The quasi-causal pathway for the regression of neuroticism on household income was reduced and nonsignificant ($b_{0E}$ = –0.012, $p$ = 0.111), and instead a significant common genetic background to education level and internalizing was present ($b_{0A}$ = –0.065, $p$ < 0.001), indicating that the genes contributing to earned income are also the same genes influencing neuroticism traits. Like what was observed with educational attainment, these results demonstrate that the relation between earned income and neuroticism is best explained by underlying genetic factors that are common to both phenotypes rather than due to systematic differences in exposure to socioeconomic factors. These results are presented in Figure 5.32. The genetic correlation between household income and neuroticism is considerably larger in magnitude than the nonshared environmental correlation, and accounts for four times as much of the total phenotypic correlation. The illustrative analysis we conducted to

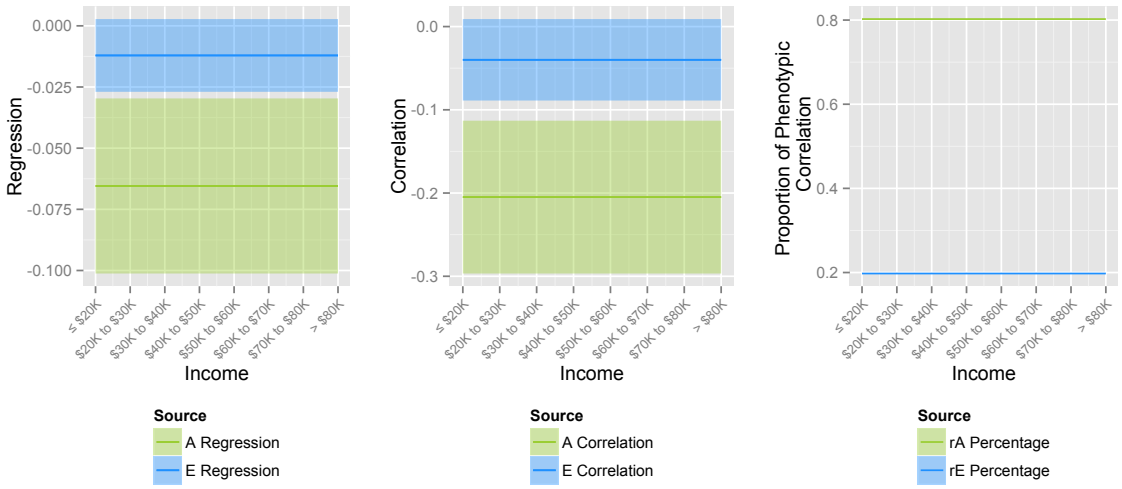


*Figure 5.32.* Main effects of household income on neuroticism in the WSTR. The left panel shows the regression of neuroticism on household income. The same relation is presented in the middle panel as genetic (green) and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic and nonshared environmental correlations. The shaded regions represent 95% confidence intervals around the regression lines.

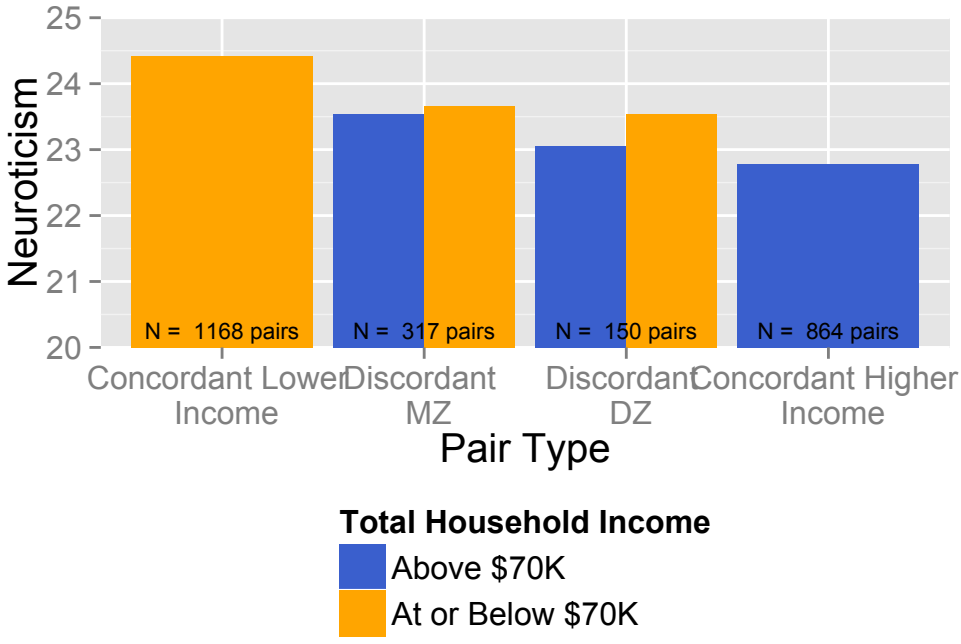


Figure 5.33. Illustrative analysis of the main effects of household income on neuroticism in the WSTR. The twin earning less (orange) is compared with the twin earning more (navy) in MZ and DZ twin pairs.

demonstrate these main effects produced results similar to those observed using educational attainment to predict neuroticism. We present this analysis in Figure 5.33.

**Area Deprivation.** At the phenotypic level, the ADI was statistically significantly associated with neuroticism ($b$ = –0.305, $p$ = 0.027), although the effect size was quite small. Individuals at the third quartile of the ADI had scores on the neuroticism factor that were 0.07 standard deviations lower than individuals at the first quartile of the ADI. By virtue of neuroticism having no influence from the common environment and the ADI having no genetic influences, this association was considered to be quasi-causal.

**Income Inequality.** Income inequality was not phenotypically associated with neuroticism ($b$ = 0.006, $p$ = 0.902).

**G×E Interaction: Effect of SES on Genetic and Environmental Etiology of Neuroticism (Dissertation Aim 2).** The moderating effects of SES on variance in neuroticism are presented in Table 5.7 under the heading *Effect of SES on Residual ACE Components of Neuroticism*. The best fitting model for each SES indicator suggested that variance in neuroticism is static with respect to socioeconomic status.

**Brief Summary.** Both compositional and contextual measures of socioeconomic status influenced mean levels of neuroticism. Education and income were correlated with neuroticism via gene-environment correlation, whereas area deprivation was correlated via the nonshared environment; income inequality was not correlated with neuroticism at the phenotypic level. Neither compositional nor contextual SES measures were correlated with residual variance in neuroticism.

## Chapter 6: Discussion – Mental Health

An abundance of research exists supporting a relationship between socioeconomic status and mental health. We summarize the extant research in detail in Chapter 1, but several highlights are noteworthy. First, the link between compositional and contextual SES and mental health is broad and spans many facets of mental well-being, including internalizing (Ansseau et al., 2007; Bøe et al., 2012; Gilman et al., 2002; Goodman, 1999; Green & Benzeval, 2013; Lemstra et al., 2008b; Lorant et al., 2003; Muramatsu, 2003; South & Krueger, 2011; Tambs et al., 2012; Zimmerman & Katon, 2005), neuroticism (Jonassaint et al, 2011; South & Krueger, 2011), externalizing (Amone-P'Olak et al., 2009; Bøe et al., 2012; Hsieh & Pugh, 1993; Huisman et al., 2010; National Center for Health Statistics, 2012), and serious mental illness and personality pathology (Hudson, 2005; Walsh et al., 2012). Second, this relationship appears to strengthen with age (Miech & Shanahan, 2000). Third, existing behavior genetics research on the SES–mental health gradient demonstrates that this relation is far more nuanced than correlational or natural experimental studies suggest. This association is rarely found to be causal and is instead attributable to family-level confounds, especially additive genetic selection factors (Behrman et al., 2015; Osler et al., 2007; South & Krueger, 2011; Strachan et al., 2016; Tambs et al., 2012). Fourth, the complexities of this phenomenon are further made evident by research demonstrating SES effects on variance in mental health. Heritability of mental illness tends to be greater in more disadvantaged environments (South & Krueger, 2011; Strachan et al. 2016; Tuvblad et al., 2006), although the mechanisms of this change varied across studies (*i.e.*, changes in raw additive genetic variance as a function of SES versus changes in heritability; causal

processes versus social selection processes). In studies using individual-level SES indicators (typically adult samples), the interaction between variance in mental health and SES appeared causal when the effect was on nonshared environmental variance (South & Krueger, 2011) and non-causal when SES acted on additive genetic factors (Strachan et al., 2016).

This dissertation study used contemporary samples of American adolescent and adult twins reared in the same household to investigate the impact of socioeconomic status on mental health more comprehensively. We examined the effect of both compositional (*e.g.*, educational attainment, household income) and contextual (*e.g.*, neighborhood socioeconomic advantage, area income inequality) measures of SES on level and variance in symptoms of psychopathology, including internalizing (*e.g.*, depression and anxiety), externalizing (*e.g.*, attention-deficit/hyperactivity disorder, delinquent behavior), and neuroticism.

**Internalizing**

We summarize the results for internalizing symptomatology in Table 6.1. We provide information regarding the direction of the main effect of SES on internalizing (column labeled *Main Effect*) as well as the approximate percentage of the SES–internalizing phenotypic correlation accounted for by the additive genetic, shared environmental, and nonshared environmental correlations (column labeled *Approx. %* r*P*). We also provide information regarding the direction of the influence of SES indicators on residual variance in internalizing (columns *Residual A*, *Residual C*, and *Residual E*). Also included in these variance columns is a summary of the changes in variance in the context of SES–internalizing ACE correlations and what those changes

Table 6.1. Summary of results for SES effects on internalizing symptomatology.

| | SES Indicator | Main Effect | Approx. % *r*P | Residual A | Residual C | Residual E |
|---|---|---|---|---|---|---|
| Depression (Add Health) | Parental Education | Protective | N/A | ∅ | ∅ | ∅ |
| | Family Income | Protective | N/A | ∅ | ∅ | ∅ |
| | Neighborhood SES | Protective | N/A | ∅ | ∅ | ∅ |
| Internalizing (WSTR) | Education | Protective (*r*A, *r*C) | A = 65%<br>C = 35%<br>E = 0% | ∅ | ∅ | ↓<br>stable *r*E<br>(causal) |
| | Income | Protective (*r*C ↓) | A = 20% → 60%<br>C = 60% → 0%<br>E = 20% → 40% | ∅ | ↓<br>lowest at low *r*C (selection) | ↓<br>lowest at high *r*E (causal) |
| | Neighborhood SES | Protective (*r*C) | —<br>C = 65%<br>E = 35% | ↓<br>no *r*A<br>(causal) | ∅ | ↓<br>stable *r*E<br>(causal) |
| | Income Inequality | ∅ | —<br>—<br>— | ∅ | ∅ | ∅ |

imply about causality versus social selection processes.

Consistent with prior research, socioeconomic status protected against symptoms of internalizing. In our study, this was true of both compositional and contextual measures of SES. Where tests of causality were possible (*i.e.*, when SES indicators were not shared by members of a twin pair, which was true only in the WSTR), results were consistent with our research hypotheses: The effect of SES on internalizing was not observed to be causal and was instead attributable to genetic and/or shared environmental selection. That is, more advantaged individuals tend to experience fewer symptoms of internalizing not because of differential exposure to socioeconomic factors, but because of a genetic and/or shared environmental background common to both SES and internalizing. Indeed, nonshared environmental correlations were not statistically significant and tended to account for less than one-third of the total phenotypic correlation. These findings also dovetail with the existing behavior genetics research suggesting a non-causal link between socioeconomic status and internalizing (Behrman et

al., 2015; Osler et al., 2007; South & Krueger, 2011; Strachan et al. 2016; Tamb et al., 2012).

In terms of variance in internalizing symptomatology, we found no evidence that family SES influenced variance in depression among adolescents. This was the first study to examine this relation in a non-adult sample, and we therefore have no basis against which we can compare these results. In adults, on the other hand, additive genetic variance in internalizing tended to be static with respect to SES (with the exception of neighborhood socioeconomic advantage, which was negatively correlated with A variance), but total phenotypic and nonshared environmental variance in internalizing decreased as a function of increasing SES. Further, we observed that these decreases in nonshared environmental variance as a function of increasing SES occurred in the context of static or decreasing nonshared environmental correlations. That is, these variance changes were causal and related to differential exposure to socioeconomic environments. These results partially support our hypotheses. We predicted that higher SES environments would constrain phenotypic variance in internalizing (supported) and that this effect would be driven by decreases in raw additive genetic variance (*i.e.*, that low socioeconomic conditions potentiate genetic vulnerabilities for poor mental health; not supported). Our results are largely consistent with previous research, however, suggesting decreases in nonshared environmental variance as a function of increasing SES (South & Krueger, 2011; Strachan et al. 2016). We also extend existing research by offering an explanation of the mechanism by which SES impacts on variance in internalizing.

That internalizing symptomatology and socioeconomic status share an underlying

genetic (and, to a lesser extent, shared environmental) pathway warrants additional attention. One interpretation of this association is that there are "genes for" being wealthy, which are the very genes that influence internalizing. A famous and insightful explanation of the relation between genes and complex behavior (television viewing, in this case) stands as a reminder for why this interpretation is far too simplistic:

> Of course, there are no genes for television viewing just as there are no genes for performance on IQ tests or for height. Complex phenotypes such as these are heritable but not inherited. We do not inherit genes that code for vocabulary words or for height, and we cannot inherit genes that code for television viewing. Genes only code for sequences of amino acids.... In other words, finding genetic influence on individual differences in children's television viewing means that some unspecified genetic differences among children indirectly affect the extent to which children watch television (Plomin, Corley, DeFries, & Fulker, 1990, p. 371).

Following Plomin et al. (1990), we assert that there are no "genes for" being rich or otherwise socioeconomically advantaged that are causing depression or anxiety. Instead, we hypothesize that traits with a strong genetic basis (*e.g.*, conscientiousness, neuroticism) predict both an individual's tendency to achieve higher academically and/or pursue more lucrative career paths *and* his or her propensity for experiencing internalizing symptomatology. That is, more advantaged individuals are not somehow "genetically superior" to less advantaged persons in terms of wealth or health, but rather are more likely to exhibit certain temperaments that directly or indirectly influence both socioeconomic position and mental health status. Such influences may be a result of passive, evocative, or active gene-environment correlation[21].

[21] Passive *r*GE refers to the relation between inherited genotypes and the environment in which an individual is raised. Evocative *r*GE refers to the association between an individual's inherited behavior and others' reactions to that behavior. Active *r*GE refers to the relation of an individual's inherited genotype and the environments that individual selects or creates (Jaffee & Price, 2008).

As an example, consider that individuals who are high on neuroticism (which is approximately 41% heritable; Jang, Livesley, & Vernon, 1996; 47% heritable in our WSTR sample) tend to be harm avoidant and may be reluctant to take risks (including financial or educational ones that may improve socioeconomic position; c.f. Kuhnen, Samanez-Larkin, & Knutson, 2013). Those same individuals may also be less likely to make uncomfortable behavioral changes that might yield psychological relief, less likely to have adequate distress tolerance coping skills (c.f. Lahey, 2009), or more likely to elicit negative interactions from others, thereby contributing to or maintaining depressive or anxious symptoms. A similar explanation could be proffered for the personality trait conscientiousness (which is approximately 44% heritable; Jang et al., 1996). Conscientious individuals may be more likely to be achievement-oriented and pursue post-secondary education, thus paving the way for better paying career opportunities. Those same characteristics may also contribute to a person's likelihood to seek mental health resources when needed, and to adhere more closely to clinician recommendations (Hill & Roberts, 2012).

Socioeconomic status does not cause internalizing symptomatology, but it does seem to cause changes in residual nonshared environmental variance in internalizing (E×E interaction in the context of stable *r*E). Bronfenbrenner & Cici's (1992) bioecological model posits that propensity for maladaptive traits will be greater in more deprived environments. We predicted that the affected propensity would be genetic in nature; what we instead found is that nonshared environmental risk for internalizing is greater in environments with fewer socioeconomic resources. Consider an environmental stressor triggering depressive symptomatology, such as job loss, academic problems or

scholarship loss, or loss of a spouse. Such stressors certainly contribute to variance in internalizing, but may disproportionately impact on those at the low end of the socioeconomic spectrum due to the financial burdens already in play (*e.g.*, being able to afford bills, struggling to support one's family). At the high end of the socioeconomic spectrum, however, such stressors may be relatively inconsequential to variance in internalizing, as these individuals may not face the same financial or socioeconomic burdens that characterize low-SES individuals. What results from such an interactive process is fewer individuals at the high end of the SES spectrum experiencing more serious symptomatology, which is broadly consistent with our data observations.

Such E×E interaction may also arise from phenotype–environment correlation (*r*PE; Beam & Turkheimer, 2013). Pathological processes are individual processes that take off on top of genes, and an individual's twin is not correlated with such processes. Phenotype-environment correlation is a transactional process by which small phenotypic differences result in differential exposure to environments (by either evocative or active means), which could then reciprocally affect phenotypic expression in a chain of interactions that eventually leads to large changes in phenotype (Beam & Turkheimer, 2013; Dickens & Flynn, 2001), and, consequently, large within-pair differences. That is to say, this type of process will induce expansions in within-family variance in a phenotype under certain conditions; our results suggest that deprived environments may be one such condition. Indeed, the fact that we observed E×E effects in adults but not in adolescents seems to support this interpretation. Variance changes in internalizing as a function of socioeconomic status is a sign that internalizing is the outcome of a process, not simply something written into one's genes.

It is noteworthy to point out that heritability (*i.e.*, the proportion of variance attributable to additive genetic influences) of internalizing appeared to increase with increasing socioeconomic status, though this is a qualitative observation (no statistical testing on standardized variance in internalizing was performed). This observation suggests that the importance of genetic factors on internalizing is higher in more advantaged environments, an observation consistent with an environmental push perspective (adapted from the "social push" perspective described by Raine, 2002). Under this theory, a genetic diathesis for internalizing symptomatology is more salient and/or likely when environmental predispositions to internalizing are minimized (Raine, 2002). Recently, Tsang, Duncan, Dinescu, and Turkheimer (2017) introduced a dual distribution hypothesis of human behavior, which postulates that the distribution of many human behavior phenotypes can actually be decomposed into two separate distributions: one that is normally distributed and under strong genetic control and another that is skewed and characterized by environmental processes operating at the individual level (see Figure 6.1). In enriched environments, there is less E×E and therefore the distribution is more heavily genetic. In deprived environments, there is more E×E (likely driven by *r*PE), which results in a distribution that is more heavily environmental (and has greater overall variance).

***Implications.*** It is important to note that, because SES and internalizing are correlated via between-family (*i.e.*, additive genetic, shared environmental) pathways, income redistribution (*e.g.*, minimum wage increases, wage increases for low-skilled workers) or post-secondary education incentives (*e.g.*, government-subsidized college tuition, tuition elimination) is unlikely to cause quantifiable shifts in *mean levels* of

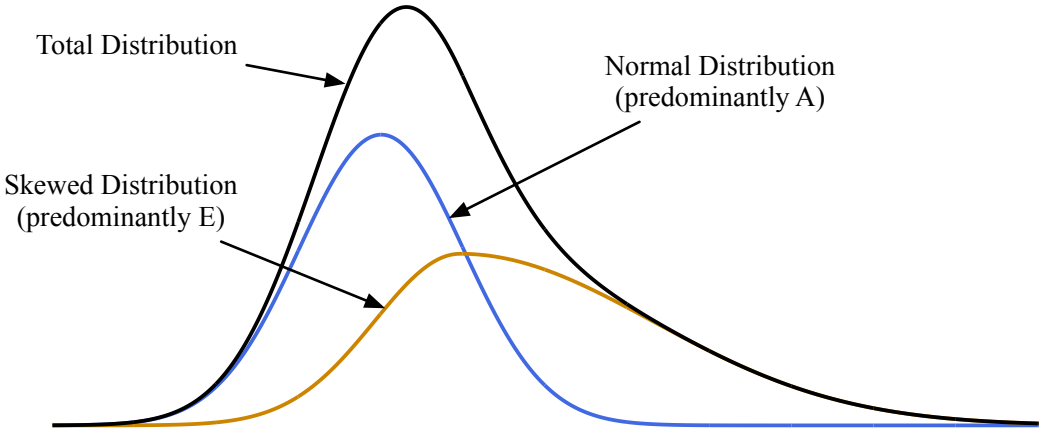


*Figure 6.1*. Illustration of the dual distribution hypothesis of human behavior (Tsang, Duncan, Dinescu, & Turkheimer, 2017). The phenotype (black distribution) is a combination of two distributions, a normal distribution under strong genetic control (navy distribution) and a skewed distribution representing nonshared environmental processes (orange distribution).

internalizing symptomatology in the general population. On the other hand, our research demonstrates that low SES exacerbates environmental risk for internalizing and suggests that this effect is causal in nature. That is, better socioeconomic conditions may help to relieve the internalizing symptomatology of some individuals in a regression-toward-the-mean fashion. Internalizing on a grand scale remains unchanged, but *the internalizing scores of some will be less severe*.

Our findings seem to underscore the importance of early interventions for individuals at risk for internalizing psychopathology. Our data show that the influence of SES on variance in internalizing is undetectable in adolescence, and rather is a process that occurs over time. Policy makers might consider implementing school- or community-based interventions for at-risk (*i.e.*, low-SES) children. In adults, healthcare providers might recommend individual, group (an effective intervention at a fraction of the cost of individual psychotherapy), or family psychotherapy for individuals from

poorer socioeconomic backgrounds in an effort to mute some of the environmental factors that may be contributing to internalizing. Of course, improving socioeconomically deprived neighborhoods through allocation of economic resources (*e.g.*, housing or general environmental improvements; increasing employment opportunities within poorer neighborhoods by, for example, offering tax incentives to businesses that open locations in low-SES areas) may be a particularly influential strategy for reducing the psychological burden for many low-SES individuals. Many clinicians operate on a sliding scale in order to best serve the needs of individuals from deprived socioeconomic backgrounds; more clinicians might consider this as an option for their practice. In addition, insurance companies might consider a sliding scale model for copays or extending the maximum number of covered psychotherapy session for individuals at the lower end of income. Psychotherapists and healthcare providers should recognize that the source of internalizing presentations may be more environmental in individuals from deprived backgrounds, whereas such presentations may be more biologically-based in individuals from higher SES backgrounds. This might help better inform patients regarding decisions about pursuing pharmacological treatment for internalizing.

**Externalizing**

A summary of the results for externalizing symptomatology is presented in Table 6.2 (ADHD) and Table 6.3 (delinquent behavior). With regard to ADHD, our results were consistent with prior research suggesting that greater socioeconomic advantage protects against symptoms and/or diagnosis of ADHD. Whereas other studies have observed this effect for global or financial measures of SES (Amone-P'Olak et al., Table

6.2. Summary of results for SES effects on ADHD symptomatology.

| | SES Indicator | Main Effect | Approx. % *r*P | Residual A | Residual C | Residual E |
|---|---|---|---|---|---|---|
| ADHD (Add Health) | Parental Education | Protective | N/A | ∅ | ∅ | ↑ |
| | Family Income | ∅ | N/A | ∅ | ∅ | ∅ |
| | Neighborhood SES | ∅ | N/A | ↑ | ∅ | ↓ |
| ADHD (WSTR) | Education | Protective (*r*E) | A = 30% → 20%<br>C = 50% → 40%<br>E = 20% → 40% | ∅ | ∅ | ↑<br>highest at low *r*E (causal) |
| | Income | ∅ | — | ∅ | ∅ | ∅ |
| | Neighborhood SES | ∅ | — | ∅ | ∅ | ∅ |
| | Income Inequality | ∅ | — | ∅ | ∅ | ∅ |

2009; Bøe et al., 2012; National Center for Health Statistics, 2012), we observed the effect only for educational attainment. Where tests of causality were possible (only true for the WSTR), results partially supported our research hypothesis that this relation is mediated by between-family factors. Although the data also supported a causal explanation, we observed that (nonsignificant) between-family factors common to both phenotypes explained a majority of the phenotypic correlation (80%) except at very high levels of educational attainment (between-family = 60%; within-family = 40%). That is to say, many of the factors that contribute to an individual pursuing higher education are also the same factors which contribute to a reduced risk of being diagnosed with ADHD.

We arrived at an unexpected conclusion when we examined the effect of socioeconomic status on variance in ADHD. In both adolescents and adults, phenotypic variance in ADHD appears to *increase* as a function of increasing SES, an effect driven by more individuals at the extreme ends of the ADHD spectrum at high SES levels. Educational attainment was related to increased nonshared environmental variance in

ADHD, a process that was causal in adults. The adolescent results for neighborhood SES diverged somewhat from this pattern, however; higher neighborhood SES was associated with lower environmental variance in ADHD but greater additive genetic variance. These differing results may reflect differences between the impact of compositional versus contextual measures of SES on variance in ADHD. On the one hand, results for educational attainment reflect an E×E process by which nonshared environmental variance is greatest in the most enriched family or individual environments. On the other hand, results for neighborhood SES reflect a G×E process by which additive genetic variance is greatest in the most enriched neighborhood environments.

It is interesting to speculate about why educational attainment—an apparently enriching environmental factor—might lead to more individuals being very high on the ADHD spectrum rather than fewer, as we observed with internalizing symptomatology. One explanation is that in families with strong educational backgrounds or in which education is highly valued, perhaps children who are showing signs of attentional difficulties or who struggle academically are more readily identified as having ADHD. Alternatively, among more educated individuals, there may be an incentive to seek an ADHD diagnosis (*e.g.*, greater productivity associated with stimulant medication use or performance benefits associated with extended time for tests; Sansone & Sansone, 2011).

It is perhaps more interesting to speculate about why neighborhood socioeconomic advantage during adolescence seems to accentuate genetic variance in ADHD. Bronfenbrenner and Cici's (1994) stress-diathesis hypothesis suggests that environmental stress potentiates expression of dysfunctional traits for which a latent predisposition exists. Why is it then that we discovered the opposite? Examination of

the MZ and DZ absolute pair differences as a function of neighborhood SES sheds light on the process at hand. At higher levels of neighborhood SES, DZ twins are more different from one another in terms of ADHD symptoms than are DZ twins at lower levels of education, which drives up additive genetic variance in ADHD as a function of neighborhood SES. This observation is consistent with what would be expected under conditions in which *r*PE was in play. The DZ twin who is less attentive or more hyperactive may, because of their phenotypic characteristics, either seek out or be placed into environments that contribute to greater expression of these traits (or vice versa for the more attentive, less hyperactive twin). Beam & Turkheimer (2013) note that *r*PE can lead to false positive G×E results.

Measures of delinquent behavior were available only in the adolescent sample (see Table 6.3). Mean level of delinquency was not observed to be related to measures of socioeconomic status. This departs from the only existing behavior genetics study on the topic, which suggested that higher family socioeconomic position (indicated by parental education and occupation) and neighborhood socioeconomic status predicted small decreases in delinquency (Tuvblad et al., 2006). Of note, the sample size of that study was roughly 5.7 times the size of the Add Health sample used in our dissertation analyses. Nevertheless, we observed main effects near zero, which suggests that the differences in these studies are not solely due to differences in power to detect effects.

Table 6.3. Summary of results for SES effects on delinquent behavior.

| | SES Indicator | Main Effect | Approx. % *r*P | Residual A | Residual C | Residual E |
|---|---|---|---|---|---|---|
| Delinquency (Add Health) | Parental Education | ∅ | N/A | — | ∅ | ∅ |
| | Family Income | ∅ | N/A | — | ↓ | ↓ |
| | Neighborhood SES | ∅ | N/A | — | ∅ | ∅ |

Instead, this difference may be attributable to the inclusion of violent and/or more serious crimes in the Tuvblad et al. (2006) study. Replication studies are needed to better understand this relationship. Further, behavior genetics studies of delinquent behavior in which socioeconomic indicators are not shared between members of a twin pair are necessary in order to make causal inferences about this relation.

Family income predicted residual variance in delinquent behavior in our study; parental education and neighborhood SES were unrelated. We observed decreases in both shared and nonshared environmental variance as a function of increasing family income, an observation that dovetails with previous research finding that environmental variance in delinquent behavior decreases with increasing SES (Tuvblad et al., 2006). In addition, this effect appears to be driven by fewer observations at the high end of delinquency in more wealthy families. Because family income was an environmental moderator shared by members of a twin pair, we were not able to make conclusions regarding social selection versus causal processes. Still, these results seem to fit with both the stress-diathesis (Bronfenbrenner & Cici, 1994) and the dual distribution hypotheses (Tsang et al., 2017): Environmental stressors accentuate environmental propensity toward delinquent behavior.

***Implications.*** Because ADHD tended to be correlated with SES via between-family pathways, it is unlikely that policy changes to redistribute income or raise education levels will result in changes in mean levels of ADHD symptomatology or diagnosis. Although our data suggest that better socioeconomic status creates more variance in ADHD symptomatology or diagnosis, we believe that this effect is an artifact and simply reflects better identification of neurodevelopmental disorder symptoms

among better educated families.

With respect to mean levels of delinquency during adolescence, changing the SES of a family will be ineffective on mean delinquency levels. Many delinquent behaviors (*e.g.*, petty theft) may be financially motivated (Sharma, Mazar, Alter, & Ariely, 2014), however, and some research suggests that individual factors (*e.g.*, morality) may interact with environmental socioeconomic deprivation to contribute to crime (Pads+ Research Team, n.d.). Our data suggest that relieving socioeconomic deprivation may help to reduce some crime by pulling in individuals on the fringes of delinquent behavior. Considered in the context of findings from the Pads+ study (Pads+ Research Team, n.d.) and the genetically informed Tuvblad study (Tuvblad et al., 2006) suggesting that neighborhood crime exacerbates genetic and nonshared environmental risk for delinquency, it seems that reducing socioeconomic burden may not be enough for many adolescents; reducing their environmental exposure to crime is critical as well. Therefore, policy makers might consider allocating resources to crime reduction within neighborhoods falling lower on the socioeconomic spectrum.

**Neuroticism**

A summary of the results for neuroticism is presented in Table 6.4. We did not observe neuroticism to be related to SES in adolescents. In adults, both compositional and contextual measures of SES were protective against neuroticism traits. Consistent with our hypotheses and with existing behavior genetics research (South & Krueger, 2011), neuroticism and compositional SES tended to be correlated via common genetic pathways. In addition, those common genetic pathways explained 80% of the total phenotypic correlation between neuroticism and compositional SES. Neighborhood

Table 6.4. Summary of results for SES effects on neuroticism.

| | SES Indicator | Main Effect | Approx. % *r*P | Residual A | Residual C | Residual E |
|---|---|---|---|---|---|---|
| Neuroticism (Add Health) | Parental Education | ∅ | N/A | ∅ | — | ∅ |
| | Family Income | ∅ | N/A | ∅ | — | ∅ |
| | Neighborhood SES | ∅ | N/A | ∅ | — | ∅ |
| Neuroticism (WSTR) | Education | Protective (*r*A) | A = 80%<br>—<br>E = 20% | ∅ | — | ∅ |
| | Income | Protective (*r*A) | A = 80%<br>—<br>E = 20% | ∅ | — | ∅ |
| | Neighborhood SES | Protective (*r*E) | —<br>—<br>E = 100% | ∅ | — | ∅ |
| | Income Inequality | ∅ | —<br>—<br>— | ∅ | — | ∅ |

socioeconomic advantage (a measure of contextual SES), on the other hand, showed a causal relationship (due to no additive genetic variance in neighborhood SES and no shared environmental variance in neuroticism).

Residual variance in neuroticism was unrelated to socioeconomic status. This departs from the only existing genetically informed G×E study of neuroticism and SES (South & Krueger, 2011), which found that nonshared environmental variance in neuroticism decreases with increasing household income. It should be noted, however, that that study included neuroticism traits along with symptom counts for depression, generalized anxiety, and panic attacks. Although neuroticism is comorbid with internalizing disorders, it is typically regarded as related to but distinct from psychopathology (Widiger, 2009). The results of our study suggest that variance in internalizing psychopathology decreases with increasing socioeconomic status, but variance in the personality trait of neuroticism does not.

That neuroticism and SES are correlated via additive genetic pathways and

variance in neuroticism is independent of socioeconomic status seems to fit with our hypothesis that SES and health are genetically correlated due to mediation by traits or temperaments that have a strong genetic basis. We hypothesize that neuroticism is a personality trait influencing achievement-related or risk-averse behaviors that confer socioeconomic advantage; we would not expect variance in neuroticism, a major player driving one's SES level, to be predicted by that which it causes.

**Summary**

Almost without exception, socioeconomic status is not causally related to mental health. Instead, these phenotypes share a common genetic (and, less commonly, shared environmental) etiology. We hypothesize that this etiology is in the form of personality characteristics or temperaments which have a strong genetic basis predicting behaviors that both interfere with upward movement on the socioeconomic spectrum *and* facilitate and/or maintain psychopathology. In terms of real-world implications, these results imply that eliminating socioeconomic burdens in society will not influence mean mental health levels. Yet, our results also had much more to say about this complex relation.

With the exception of externalizing, socioeconomic indicators showed no evidence of influence on additive genetic, shared environmental, or nonshared environmental variance in the mental health of adolescents. Instead, the effects of SES on mental health seem to result from an interactive, reciprocally causal process which occurs over time. In adults, SES predicted reduced variance in internalizing symptomatology, and increased variance in externalizing disorders. In addition, these variance changes were the result of E×E interactions that were pulling individuals from (internalizing, delinquency) or pushing individuals toward (ADHD) the tail of the

distribution marking greater psychopathology. While income redistribution or widespread availability of educational opportunities may not affect mean levels of psychopathology, it may reduce this burden for some, particularly those at greatest environmental risk.

# Chapter 7: Results – Physical Health

The role of socioeconomic status on physical health was evaluated using several indicators of physical well-being. These indicators included an overall estimate of one's health (considered the gold standard for estimating health status); the presence of various health conditions (*e.g.*, diabetes, asthma); body mass index; and immune functioning (*e.g.*, inflammatory protein levels, antibody levels). We discuss the influence of socioeconomic status on each of these physical health and well-being indicators in turn.

## General Health

***Self-Rated Health—Add Health.*** As noted above, general health in the Add Health sample was assessed using a single-item measure of self-rated health (see Chapter 3). We fit the model presented in Figure 7.1 to the data separately for each socioeconomic status indicator, partialling for the linear effects of age and gender on level and variance in self-rated general health. We used likelihood ratio tests to determine the best fitting model. Biometric decomposition of both the SES indicators and general health are presented in Table 4.1, and parameter estimates for each of these models are presented in Table 7.1. Family household income was log-transformed to correct for positive skew. General health showed no evidence of influence from shared environmental factors; therefore, we did not include C in any of the models fit to the data.

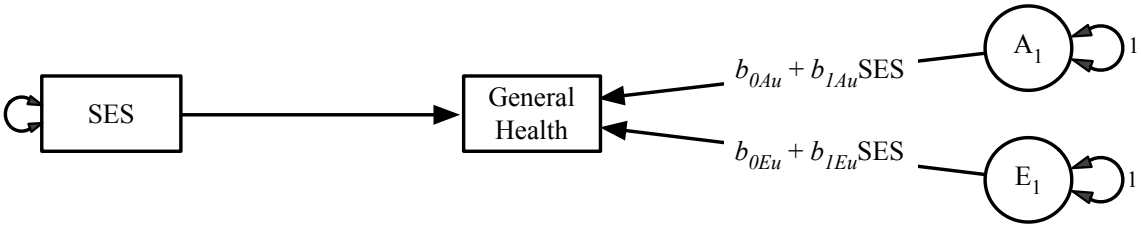


*Figure 7.1*. Path diagram G×SES model fit to self-rated general health in the Add Health sample (only one twin shown for clarity).

Table 7.1. Parameter estimates and model fit statistics for G×SES models, self-rated general health in the Add Health sample.

| | Parameter | Phenotypic Model (No Moderation) | Moderation of Residual Variance | Best-Fitting Model |
|---|---|---|---|---|
| **Parental Education** | Main Effect of Parental Education on Self-Rated Health | | | |
| | $b_1$ | ***.124 (.025)*** | ***.126 (.025)*** | ***.127 (.025)*** |
| | Effect of Education on Residual ACE Components of Self-Rated Health | | | |
| | $b_{0Au}$ | .331 (.478) | ***1.953 (.404)*** | .342 (.479) |
| | $b_{1Au}$ | — | .050 (.028) | — |
| | $b_{0Eu}$ | .413 (.300) | .433 (.252) | .523 (.310) |
| | $b_{1Eu}$ | — | –.032 (.020) | –.038 (.020) |
| | Model Fit | | | |
| | *-2LL* | 5915.846 | 5908.378 | 5912.020 |
| | *Δ-2LL (Δdf)* | — | 7.468 (+2) | 3.642 (–1) |
| | *p* | — | .024† | .056 |
| **Family Household Income** | Main Effect of Family Income on Self-Rated Health | | | |
| | $b_1$ | .066 (.024) | .125 (.042) | .123 (.041) |
| | Effect of Family Income on Residual ACE Components of Self-Rated Health | | | |
| | $b_{0Au}$ | ***.288 (.494)*** | ***2.063 (.447)*** | .244 (.489) |
| | $b_{1Au}$ | — | –.040 (.051) | — |
| | $b_{0Eu}$ | .442 (.299) | ***.622 (.287)*** | **.791 (.327)** |
| | $b_{1Eu}$ | — | **–.076 (.034)** | ***–.084 (.032)*** |
| | Model Fit | | | |
| | *-2LL* | 5292.412 | 5284.708 | 5285.378 |
| | *Δ-2LL (Δdf)* | — | 7.704 (+2) | .670 (–1) |
| | *p* | — | .021† | .413 |
| **Neighborhood Socioeconomic Advantage** | Main Effect of Neighborhood SES on Self-Rated Health | | | |
| | $b_1$ | .067 (.043) | .067 (.043) | .067 (.043) |
| | Effect of Neighborhood SES on Residual ACE Components of Self-Rated Health | | | |
| | $b_{0Au}$ | ***1.723 (.396)*** | .256 (.488) | ***1.723 (.396)*** |
| | $b_{1Au}$ | — | .111 (.063) | — |
| | $b_{0Eu}$ | .297 (.259) | .515 (.321) | .297 (.259) |
| | $b_{1Eu}$ | — | **–.082 (.037)** | — |
| | Model Fit | | | |
| | *-2LL* | 4850.546 | 4845.134 | 4850.546 |
| | *Δ-2LL (Δdf)* | — | 5.412 (+2) | — |
| | *p* | —† | .067 | — |

*Note:* Standard errors presented within parentheses. Estimates p < .05 **bolded**. Estimates p < .01 *italicized*. Baseline model denoted by †.

**Main Effects of SES on Self-Rated Health (Dissertation Aim 1).** The main effects of SES on self-rated general health are presented in Table 7.1 under the heading *Main Effect of Moderator on Self-Rated Health*. We discuss the main effects results for each moderator below.

**Parental Education.** Parental education was significantly associated with greater self-rated health ($b$ = 0.124, $p$ < 0.001). Adolescents with a parent who had earned at least a college degree had depression scores that were 0.29 standard deviations higher

than their counterparts whose parent holds a high school degree.

**Family Income.** Self-rated health was also positively associated with family household income ($b$ = 0.066, $p$ = 0.006). Adolescents living with a household income at the first quartile ($20,000/year) reported general health scores that were 0.09 standard deviations lower than adolescents living with a household income at the third quartile ($56,000/year).

**Neighborhood Socioeconomic Advantage.** Neighborhood-level SES was not statistically significantly associated with self-rated health ($b$ = 0.067, $p$ = 0.120).

**G×E Interaction: Effect of SES on Genetic and Environmental Etiology of Self-Rated Health.** The moderating effects of SES on variance in self-rated general health are presented in Table 7.1 under the heading *Effect of SES on Residual ACE Components of Self-Rated Health.*

**Parental Education.** The best-fitting model suggested that residual variance in self-rated general health decreased as a function of increasing parental educational attainment, an effect driven by decreasing nonshared environmental variance ($b_{1Eu}$ = –0.039, $p$ = 0.043). These model results are illustrated in Figure 7.2, which shows residual AE variance components of self-rated general health as a function of parental educational attainment, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure clearly demonstrates how residual phenotypic and nonshared environmental variance in general health decreases with increasing parental education. Considered in the context of the protective effect of parental education on mean level of general health, these results suggest that high-SES environments also protect against environmental risk for poorer health.

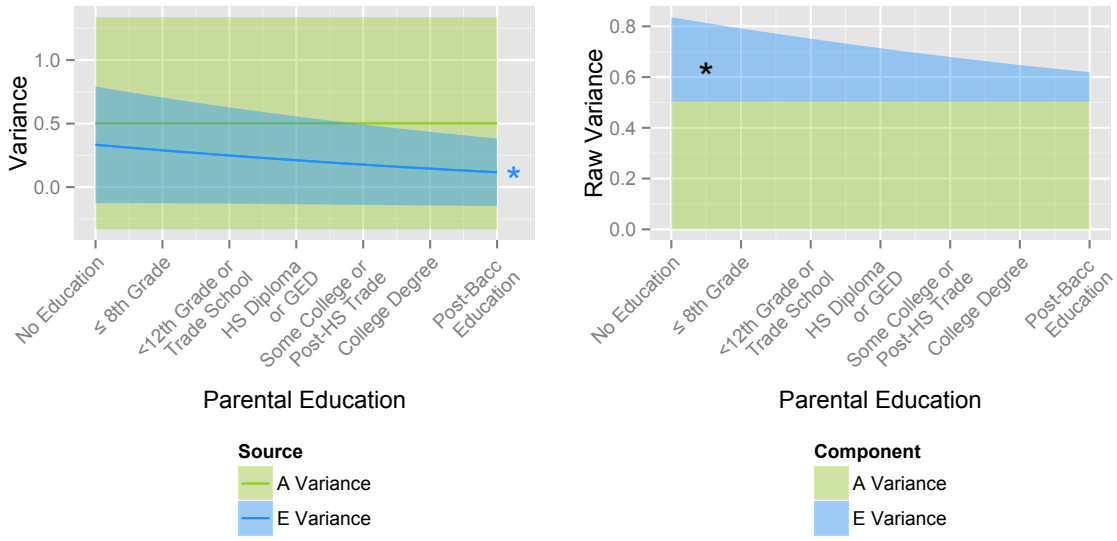


*Figure 7.2.* Gene-by-environment interaction between self-rated general health and parental educational attainment in the Add Health sample. The left panel shows additive genetic (green) and nonshared environmental (blue) variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in general health decreases as a function of increasing parental education. The asterisks (*) indicate that variance in general health depends on level of parental education.

To illustrate these results, we plotted absolute pair differences in self-rated health for MZ and DZ twins against parental educational attainment (see the left panel of Figure 7.3). In this plot, the gap between the MZ and DZ regression lines (*i.e.*, the tendency for MZ pairs to be more similar than DZ pairs) represents additive genetic variance in self-rated health. Increases in this gap with respect to education would suggest that additive genetic variance in self-rated health increases with increasing parental educational attainment; decreases would suggest the opposite. The location of the MZ line reflects nonshared environmental variance in self-rated health. A decreasing slope represents decreasing nonshared environmental variance in self-rated health. Evident in this plot is that the slope of the MZ regression lines is negative, reflecting decreasing nonshared environmental variance in self-rated health with increasing parental educational

attainment. The distance between these lines remains stable with respect to educational attainment, consistent with additive genetic variance being static as a function of education level.

This heteroscedasticity is further illustrated in the right panel of Figure 7.3, which shows box plots overlaid with violin plots (which show the probability density of the data) of self-rated health by quartile of parental educational attainment. This plot shows that the distribution of self-rated health becomes less platykurtic at higher levels of parental educational attainment, demonstrating that overall variance in self-rated health is more constrained at higher SES levels. This effect appears to be driven primarily by the presence of fewer individuals in the lower tail of the self-rated health distribution (*i.e.*, worse self-rated health) at higher SES levels.

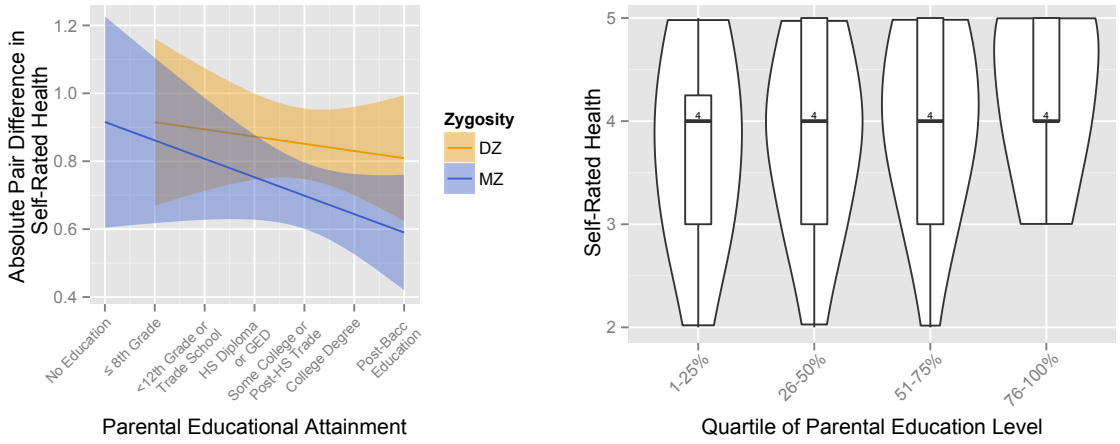


*Figure 7.3.* Illustrative analysis of the effects of parental educational attainment on variance in self-rated health in the Add Health sample. The left panel shows absolute pair differences in self-rated health as a function of parental educational attainment. The distance between the MZ and DZ lines represents the additive genetic variance in self-rated health. The location of the MZ line represents nonshared environmental variance in self-rated health. The right panel shows box plots overlaid with violin plots, self-rated health as a function of quartile of parental educational attainment.

**Family Income.** The best-fitting model suggested that residual variance in self-rated general health decreased as a function of increasing family income. Like education, this effect appeared to be driven primarily by decreasing nonshared environmental variance ($b_{1Eu}$ = –0.084, $p$ = 0.008). These model results are illustrated in Figure 7.4. These results, like those for parental educational attainment, suggest that high-SES environments protect against environmental risk for poor physical health.

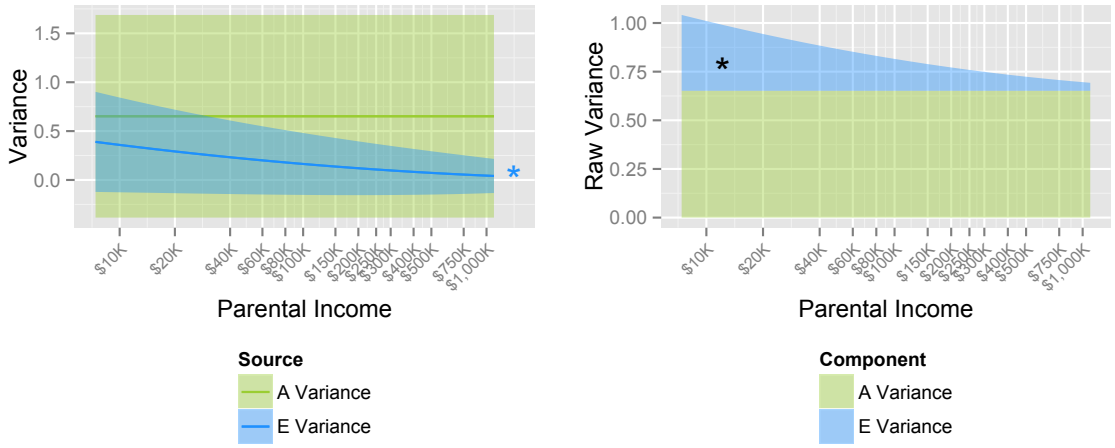


*Figure 7.4.* Gene-by-environment interaction between self-rated general health and family income in the Add Health sample. The left panel shows additive genetic (green) and nonshared environmental (blue) variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in general health decreases as a function of increasing family income. The asterisks (*) indicate that variance in general health depends on level of family income.

These results are further illustrated in Figure 7.5. As we observed with parental education, nonshared environmental variance (but not additive genetic variance) in self-rated health decreases as a function of increasing family income. The violin plots show that this effect appears to be the result of fewer observations in the lower (*i.e.*, worse) tail of self-rated health when family income level is high.

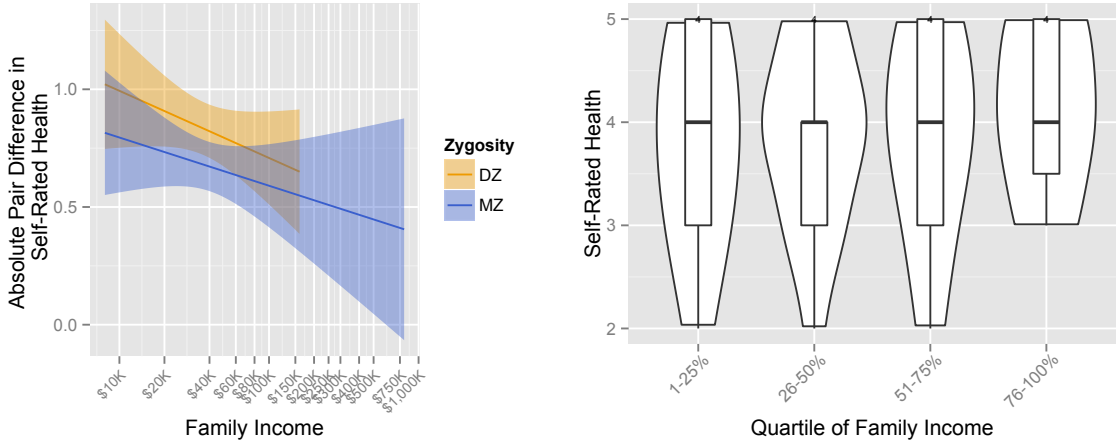


*Figure 7.5.* Illustrative analysis of the effects of family income on variance in self-rated health in the Add Health sample. The left panel shows absolute pair differences in self-rated health as a function of family income. The distance between the MZ and DZ lines represents the additive genetic variance in self-rated health. The location of the MZ line represents nonshared environmental variance in self-rated health. The right panel shows box plots overlaid with violin plots, self-rated health as a function of quartile of family income.

**Neighborhood Socioeconomic Advantage.** The best-fitting model did not suggest that residual variance in self-rated general health is related to neighborhood-level socioeconomic advantage. However, when residual variance was allowed to vary as a function of neighborhood-level SES, nonshared environmental variance tended to decrease with increasing neighborhood-level SES ($b_{1Eu}$ = –0.082, $p$ = 0.027[22]). These model results are illustrated in Figure 7.6. Again, it seems that high-SES environments protect against environmental risk for poor physical health. Illustrative analyses further illustrate these results (see Figure 7.7). Like we observed with parental education and family income and consistent with decreasing within-family variance as a function of neighborhood SES, MZ and DZ twins grow more similar as a function of increasing

[22] This moderated model itself fell just shy of statistical significance when compared with the unmoderated model ($p$ = .067).

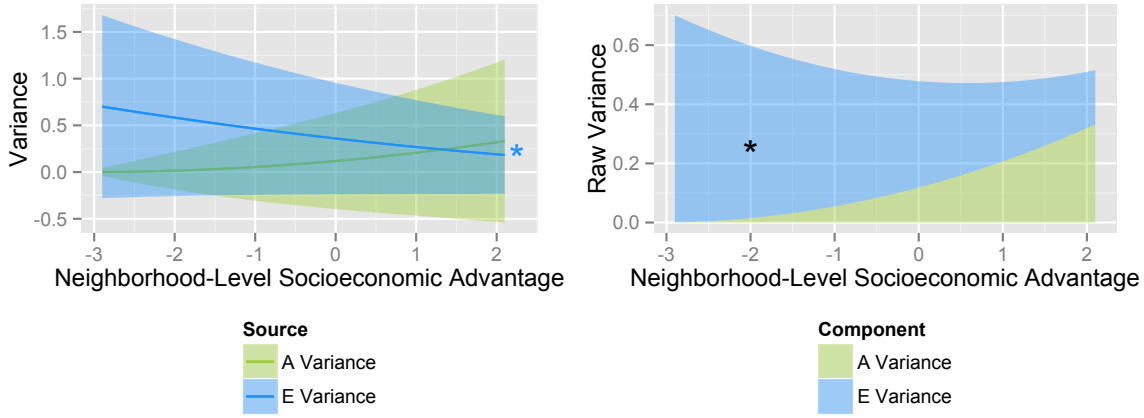


*Figure 7.6.* Gene-by-environment interaction between self-rated general health and neighborhood-level socioeconomic advantage in the Add Health sample The left panel shows additive genetic (green) and nonshared environmental (blue) variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in general health increases as a function of increasing neighborhood-level SES. The asterisks (*) indicate that variance in general health depends on level of neighborhood-level SES.

neighborhood SES. Further, the differences between the MZ and DZ regression lines remains approximately stable across all levels of neighborhood SES. The violin plots show that this constraint in variance at high neighborhood SES is driven primarily by fewer observations in the lower (*i.e.*, worse) tail of self-rated health.

**Brief Summary.** Compositional measures of socioeconomic status were related to better self-rated general health; contextual SES trended in this same direction, although did not reach statistical significance. Both compositional and contextual SES predicted decreased nonshared environmental variance in self-rated general health. That is, socioeconomic status tended to protect against poor physical health as well as against environmental risk for poor health.

***Self-Rated Health—WSTR.*** A similar measure of subjective general health was used in the WSTR sample (see Chapter 3). We fit the model presented in Figure 7.8 to

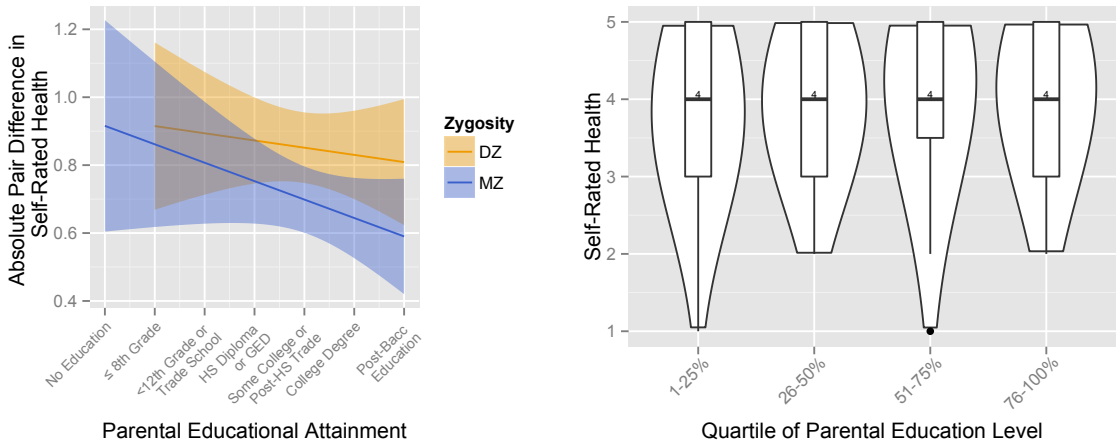


*Figure 7.7.* Illustrative analysis of the effects of neighborhood socioeconomic advantage on variance in self-rated health in the Add Health sample. The left panel shows absolute pair differences in self-rated health as a function of neighborhood SES. The distance between the MZ and DZ lines represents the additive genetic variance in self-rated health. The location of the MZ line represents nonshared environmental variance in self-rated health. The right panel shows box plots overlaid with violin plots, self-rated health as a function of quartile of neighborhood SES.

the data separately for each socioeconomic status indicator, partialling for the linear effects of age and gender on level and variance in self-rated health. We used likelihood ratio tests to determine the best fitting model. We first established a baseline model by conducting omnibus tests for the moderation by SES of both the residual variance in general health and the main effects of SES on health. We then systematically eliminated parameters from the baseline model to evaluate statistical significance of moderation parameters. Biometric decomposition of both the SES indicators and self-rated health are presented in Table 4.2, and parameter estimates and model fit statistics for the baseline and best-fitting models are presented in Table 7.2. The Area Deprivation Index was log-transformed to correct for positive skew, and the Gini Index was scaled by a factor of 10 to facilitate model convergence. These transformed values were used in all analyses in which the ADI or Gini Index was used as the indicator of contextual socioeconomic

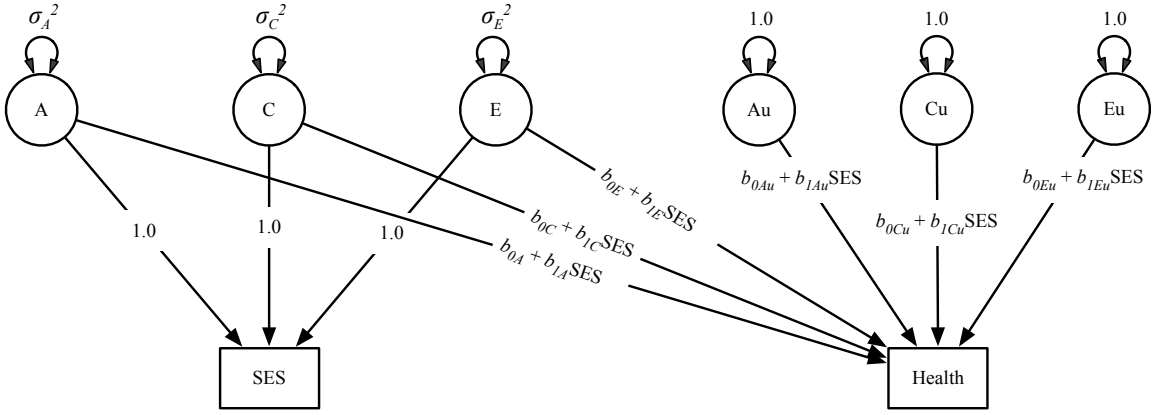


*Figure 7.8.* Path diagram G×SES model fit to self-rated health in the WSTR (only one twin shown for clarity). In the fully saturated model, the main effects of SES on self-rated health and the ACE variances of self-rated health vary as a function of SES.

status. We did not include A for the ADI since there was no evidence of genetic influences on area deprivation in our univariate model.

**Causal Pathways vs. rGE: Main Effects of SES on Self-Rated Health (Dissertation Aim 1).** The main effects of SES on self-rated health are presented in Table 7.2 under the heading *Main Effect of Moderator on Self-Rated Health*. We discuss the main effects results for each moderator below.

**Education.** Education demonstrated a phenotypic association with self-rated health ($b = 0.126$, $p < 0.001$). Controlling for age and gender, individuals with a graduate or professional degree rated their health an average of 0.61 standard deviations higher than individuals with a high school degree. The best-fitting model suggested that this association is at least partially mediated by a common shared environmental background. The quasi-causal pathway is large at low educational attainment ($b_{0E} = 0.418$, $p < 0.001$), but grows weaker with increasing educational attainment ($b_{1E} = –0.058$, $p < 0.001$). As illustrated in the left panel of Figure 7.9, which shows the magnitude of the genetic,

Table 7.2. Parameter estimates and model fit statistics for G×SES models, self-rated health in the WSTR.

| | Parameter | Phenotypic Model | Quasi-Causal Model (No Moderation) | Moderation of Residual Variance | Moderation of Main Effects | Best-Fitting Model |
|---|---|---|---|---|---|---|
| **Education** | Main Effect of Education on Self-Rated Health | | | | | |
| | $b_{0A}$ | ***.126 (.008)*** | .090 (.063) | .083 (.064) | .282 (.233) | .107 (.060) |
| | $b_{1A}$ | — | — | — | –.031 (.034) | — |
| | $b_{0C}$ | ***.126 (.008)*** | ***.238 (.073)*** | ***.251 (.075)*** | –.255 (.235) | –.071 (.141) |
| | $b_{1C}$ | — | — | — | **.080 (.034)** | **.048 (.020)** |
| | $b_{0E}$ | ***.126 (.008)*** | ***.071 (.020)*** | ***.066 (.020)*** | ***.399 (.102)*** | ***.418 (.094)*** |
| | $b_{1E}$ | — | — | — | ***–.055 (.017)*** | ***–.058 (.015)*** |
| | Effect of Education on Residual ACE Components of Self-Rated Health | | | | | |
| | $b_{0Au}$ | ***.505 (.152)*** | ***.513 (.140)*** | ***.628 (.228)*** | ***.809 (.270)*** | ***.430 (.104)*** |
| | $b_{1Au}$ | — | — | –.030 (.040) | –.052 (.037) | — |
| | $b_{0Cu}$ | .410 (.479) | .346 (.521) | .515 (.348) | .229 (.665) | ***.741 (.172)*** |
| | $b_{1Cu}$ | | | ***–.095 (.032)*** | –.061 (.049) | ***–.104 (.021)*** |
| | $b_{0Eu}$ | ***.601 (.038)*** | ***.596 (.038)*** | ***.612 (.057)*** | ***.597 (.057)*** | ***.593 (.037)*** |
| | $b_{1Eu}$ | — | — | –.003 (.008) | –.002 (.007) | — |
| | Model Fit | | | | | |
| | *-2LL* | — | 32893.900 | 32862.244 | 32844.774 | 32847.852 |
| | *Δ-2LL (Δdf)* | — | — | 31.656 (+3) | 17.470 (+3) | 3.078 (–3) |
| | *p* | — | — | <.001 | <.001† | .380 |
| **Household Income** | Main Effect of Income on Self-Rated Health | | | | | |
| | $b_{0A}$ | ***.064 (.005)*** | **.078 (.037)** | .063 (.036) | **.152 (.065)** | .064 (.036) |
| | $b_{1A}$ | — | — | — | –.017 (.009) | — |
| | $b_{0C}$ | ***.064 (.005)*** | ***.209 (.079)*** | ***.238 (.082)*** | .091 (.107) | ***.235 (.082)*** |
| | $b_{1C}$ | — | | | .027 (.015) | — |
| | $b_{0E}$ | ***.064 (.005)*** | **.020 (.008)** | **.021 (.008)** | .025 (.022) | **.020 (.008)** |
| | $b_{1E}$ | — | — | — | –.001 (.004) | — |
| | Effect of Income on Residual ACE Components of Self-Rated Health | | | | | |
| | $b_{0Au}$ | ***.505 (.131)*** | ***.490 (.145)*** | ***.433 (.083)*** | ***.581 (.157)*** | ***.436 (.097)*** |
| | $b_{1Au}$ | — | — | –.009 (.024) | –.018 (.016) | — |
| | $b_{0Cu}$ | .278 (.619) | .252 (.408) | .022 (.221) | .222 (.395) | .007 (.213) |
| | $b_{1Cu}$ | | | ***.048 (.017)*** | .027 (.019) | **.047 (.019)** |
| | $b_{0Eu}$ | ***.618 (.038)*** | ***.596 (.039)*** | ***.635 (.041)*** | ***.628 (.041)*** | ***.637 (.041)*** |
| | $b_{1Eu}$ | — | — | ***–.014 (.005)*** | ***–.014 (.005)*** | ***–.015 (.004)*** |
| | Model Fit | | | | | |
| | *-2LL* | — | 38587.122 | 38553.932 | 38551.488 | 38554.074 |
| | *Δ-2LL (Δdf)* | — | — | 33.190 (+3) | 2.444 (+3) | 2.586 (–4) |
| | *p* | — | — | <.001† | .485 | .629 |
| **Area Deprivation** | Main Effect of Area Deprivation on Self-Rated Health | | | | | |
| | $b_{0C}$ | ***.626 (.105)*** | ***1.208 (.239)*** | ***1.260 (.248)*** | ***.316 (.102)*** | ***1.255 (.247)*** |
| | $b_{1C}$ | — | — | — | **–.017 (.008)** | — |
| | $b_{0E}$ | ***.626 (.105)*** | .302 (.158) | **.250 (.170)** | .050 (.065) | **.351 (.170)** |
| | $b_{1E}$ | — | — | — | .000 (.005) | — |
| | Effect of Area Deprivation on Residual ACE Components of Self-Rated Health | | | | | |
| | $b_{0Au}$ | ***.428 (.155)*** | ***.450 (.145)*** | .065 (1.773) | **.502 (.219)** | ***.460 (.086)*** |
| | $b_{1Au}$ | — | — | –.115 (.387) | –.005 (.026) | — |
| | $b_{0Cu}$ | .319 (.389) | .282 (.411) | ***5.369 (.986)*** | .523 (.380) | ***5.349 (.994)*** |
| | $b_{1Cu}$ | — | | ***–1.182 (.204)*** | ***–.077 (.018)*** | ***–1.187 (.201)*** |
| | $b_{0Eu}$ | ***.603 (.051)*** | ***.596 (.050)*** | ***1.690 (.462)*** | ***.787 (.090)*** | ***1.634 (.428)*** |
| | $b_{1Eu}$ | — | — | **–.240 (.099)** | ***–.019 (.007)*** | **–.227 (.092)** |
| | Model Fit | | | | | |
| | *-2LL* | — | 4726.366 | 4706.902 | 4700.702 | 4706.990 |
| | *Δ-2LL (Δdf)* | — | — | 19.464 (+3) | 6.200 (+2) | 6.288 (–3) |
| | *p* | — | — | <.001 | .045† | .098 |
| **Income Inequality** | Main Effect of Income Inequality on Self-Rated Health | | | | | |
| | $b_{0A}$ | **.097 (.043)** | .933 (1.744) | .952 (1.716) | | .933 (1.744) |
| | $b_{1A}$ | — | — | — | | — |
| | $b_{0C}$ | **.097 (.043)** | .207 (.530) | .197 (.516) | | .207 (.530) |
| | $b_{1C}$ | — | — | — | | — |
| | $b_{0E}$ | **.097 (.043)** | –.027 (.070) | –.023 (.071) | | –.027 (.070) |
| | $b_{1E}$ | — | — | — | | — |
| | Effect of Income Inequality on Residual ACE Components of Self-Rated Health | | | | | |
| | $b_{0Au}$ | ***.457 (.126)*** | ***.452 (.130)*** | **.802 (.397)** | | ***.452 (.130)*** |
| | $b_{1Au}$ | — | — | .089 (.098) | | — |
| | $b_{0Cu}$ | .260 (.463) | .259 (.467) | .573 (7124) | | .259 (.467) |
| | $b_{1Cu}$ | — | — | –.210 (.137) | | — |
| | $b_{0Eu}$ | ***.615 (.042)*** | ***.614 (.042)*** | ***.615 (.198)*** | | ***.614 (.042)*** |
| | $b_{1Eu}$ | — | — | .000 (.044) | | — |
| | Model Fit | | | | | |
| | *-2LL* | — | 13646.588 | 13644.602 | | 13646.588 |
| | *Δ-2LL (Δdf)* | — | — | 1.986 (+3) | | — |
| | *p* | — | —† | .575 | | — |

*Note:* Standard errors presented within parentheses. Estimates $p < .05$ **bolded**. Estimates $p < .01$ *italicized*. Baseline model denoted by †.

shared environmental, and nonshared environmental regressions of self-rated health on educational attainment, this effect becomes indistinguishable from zero at higher levels of educational attainment (*i.e.*, Bachelor's degree or higher). On the other hand, the shared environmental pathway is negligible at low levels of educational attainment ($b_{0C}$ = –0.071, $p$ = 0.614) and grows stronger with increasing educational attainment ($b_{1E}$ = 0.048, $p$ = 0.017). Notably, the shared environmental pathway becomes influential on the mean level of self-rated health at the same education level where the nonshared pathway ceases to be influential. These results suggest that systematic differences in educational attainment are important for self-rated health, but only among less educated individuals. Among more educated individuals, family environmental factors are the most important contributors to self-rated health. There was no evidence of education level-dependent decreased genetic risk for low self-rated health ($b_{0A}$ = 0.107, $p$ = 0.076). These regression lines are re-represented as genetic, shared environmental, and nonshared environmental correlations in the middle panel of Figure 7.9. We also present the proportions of the total phenotypic correlation accounted for by the genetic, shared environmental, and nonshared environmental correlations, shown in the right panel of Figure 7.9.

To demonstrate what these effects look like within and between twin pairs, we conducted an illustrative analysis of the main effect of educational attainment on self-rated health and how it varies according to the level of educational attainment, which we present in Figure 7.10. We identified twin pairs concordant for lower education (up to Associate's degree) and pairs concordant for higher education (Bachelor's degree or

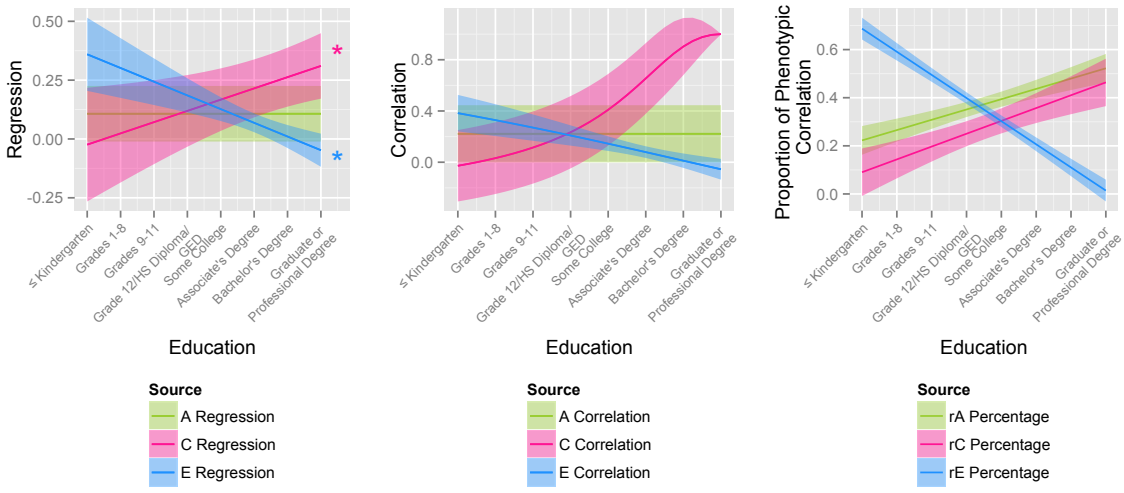


*Figure 7.9.* Main effects of educational attainment on self-rated health in the WSTR. The left panel shows the regression of self-rated health on educational attainment. The same relation is presented in the middle panel as genetic (green), shared environmental (pink), and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic, shared environmental, and nonshared environmental correlations. The asterisks (*) indicate that the magnitude of the regression of self-rated health on educational attainment depend on level of education (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). The shaded regions represent 95% confidence intervals around the regression lines.

higher). Within these groups, we then compared the mean self-rated health of the less educated twin (orange) with that of the more educated twin (navy). Overall, there is a main effect of educational attainment on self-rated health such that higher education is associated with greater self-rated health on average (this is evident comparing the height of the bars in the left panel to those in the right panel). Examining the relation at the within-pair level, it is evident that this main effect persists at lower levels of education (left panel); the more highly educated twin reported better health relative to his or her co-twin, demonstrating the presence of a causal effect of educational attainment on self-rated health operating at lower levels of education. On the other hand, this main effect

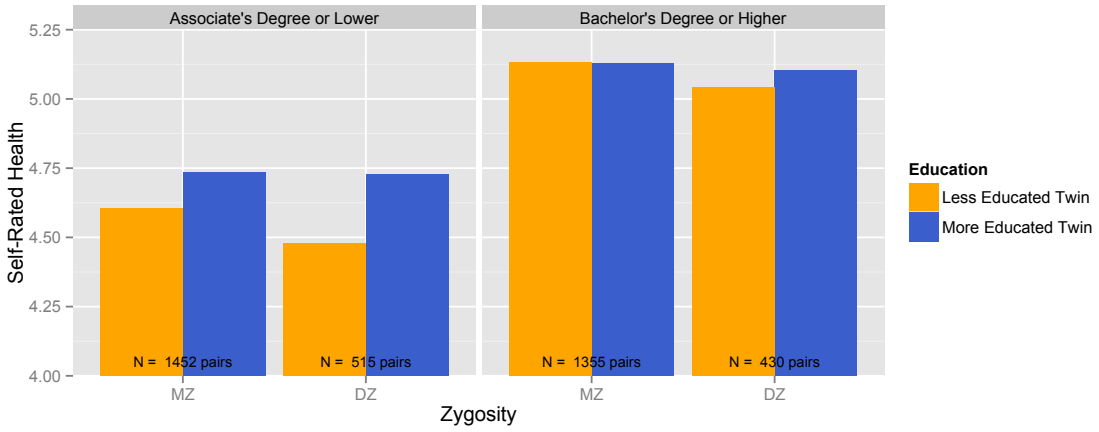


*Figure 7.10.* Illustrative analysis of the main effects of educational attainment on self-rated health in the WSTR. The left panel shows self-rated health at lower levels of education (up to an Associate's degree), and the right panel at higher levels of education (Bachelor's degree or higher). Within each panel, the less educated twin (dark gray) is compared with the more educated twin (light gray) in MZ and DZ twin pairs.

disappears at higher levels of education (right panel). Among twin pairs concordant for higher educational attainment, there are virtually no education-based differences in self-rated health among MZ or DZ twins, demonstrating the presence of a shared environmental selection effect operating at high levels of education.

**Income.** The phenotypic regression of self-rated health on household income showed a significant positive relationship ($b$ = 0.064, $p < 0.001$). That is, controlling for age and gender, individuals at the third quartile of earned income rated their health, on average, 0.39 standard deviations higher than their counterparts at the first quartile of earned income. The best-fitting model suggested that this association is partially mediated by a common shared environmental background ($b_{0C}$ = 0.235, $p$ = 0.004). The genetic and environmental regressions of self-rated health on household income did not depend on income level ($b_{0A}$ = 0.064, $p$ = 0.077; $b_{0E}$ = 0.020, $p$ = 0.014). As is evident in the left panel of Figure 7.8, the between-family contributions to this association are

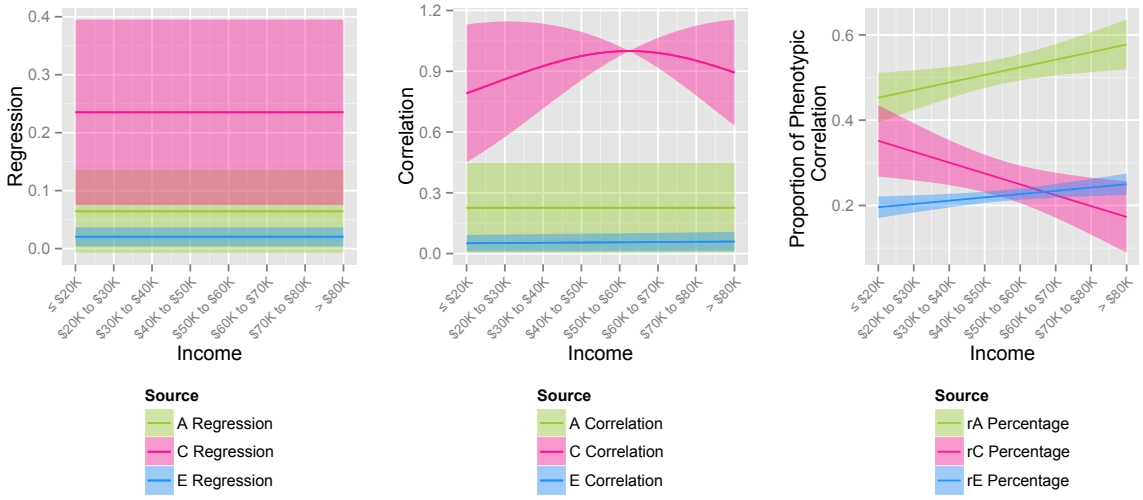


*Figure 7.11.* Main effects of household income on self-rated health in the WSTR. The left panel shows the regression of self-rated health on household income. The same relation is presented in the middle panel as genetic (green), shared environmental (pink), and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic, shared environmental, and nonshared environmental correlations. The asterisks (*) indicate that the magnitude of the regression of self-rated health on household income depends on level of education (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). The shaded regions represent 95% confidence intervals around the regression lines.

larger than the within-family contribution, although there is a small but statistically significant quasi-causal effect. Additionally, between-family confounds collectively account for much more of the total phenotypic correlation than within-family factors, evident in the right panel of Figure 7.11.

Illustrative analyses further highlight this shared environmental correlation. In Figure 7.12, we show pair differences in self-rated health as a function of pair differences in household income within randomly paired individuals (unrelated relation; blue line) and within MZ (red line) and DZ (green line) pairs. If the protective effect of household income on self-rated health was causal, the slopes of these lines would closely

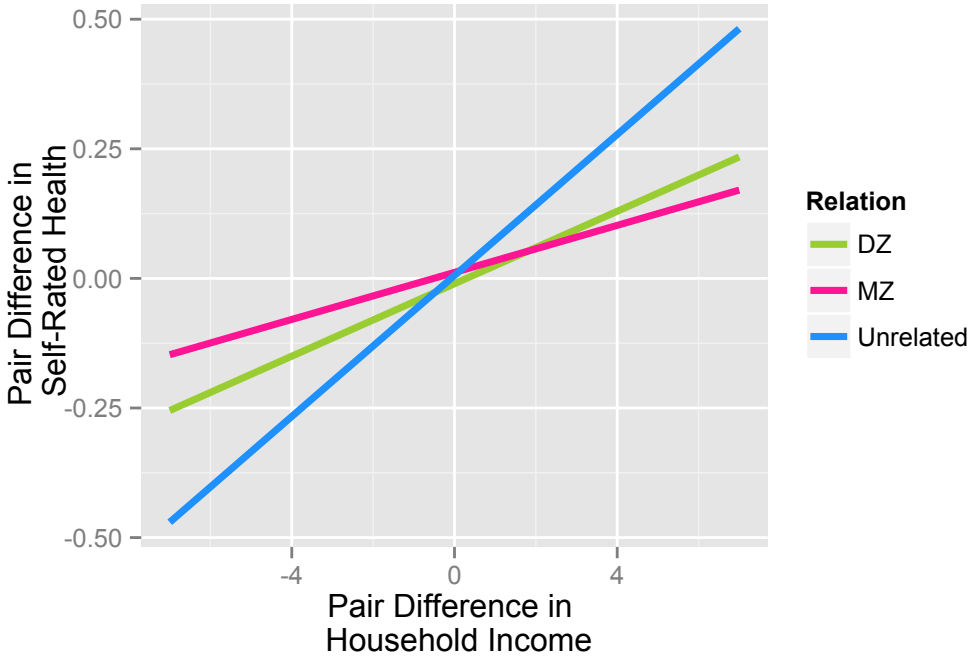


*Figure 7.12*. Illustrative analysis of the main effects of household income on self-rated health in the WSTR. This plot shows pair differences in self-rated health as a function of pair differences in household income in the population (referred to as the ‘‘Unrelated Relation’’) and within pairs of MZ and DZ twins.

approximate one another. Comparison of these lines suggests, however, that differences in household income do not predict differences in self-rated health as well within families as they do between them. This effect is instead attenuated within families. The slopes of the MZ and DZ lines are quite close, consistent with a common shared environmental pathway between household income and self-rated health.

**Area Deprivation.** At the phenotypic level, the ADI was statistically significantly associated with better self-rated health ($b = 0.626$, $p < 0.001$). Individuals at the third quartile of the ADI rated their health 0.13 standard deviations higher than individuals at the first quartile of the ADI. The best-fitting model suggested that this association is partially mediated by a common shared environmental background (as a

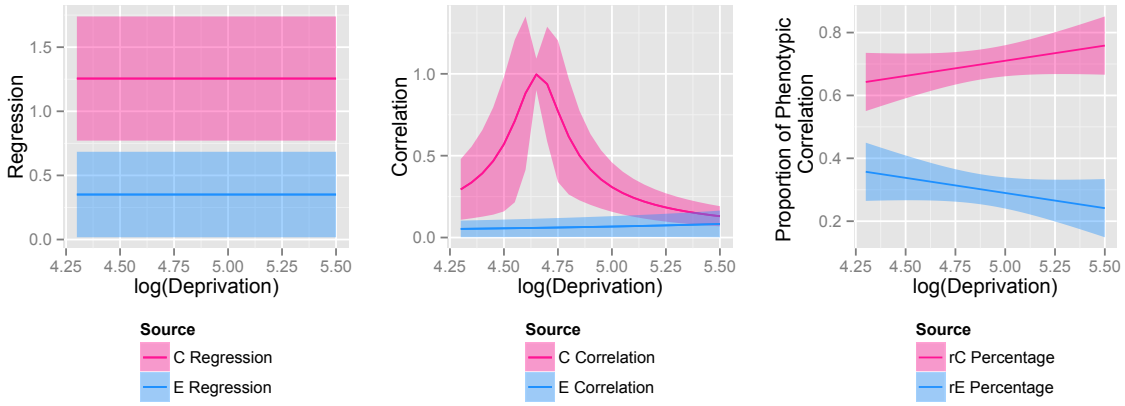


*Figure 7.13*. Main effects of area deprivation on self-rated health in the WSTR. The left panel shows the regression of self-rated health on the (log transformed) ADI. The same relation is presented in the middle panel as shared (pink) and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic, shared environmental, and nonshared environmental correlations. The asterisks (*) indicate that the magnitude of the regression of self-rated health on the ADI depends on level of area deprivation (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). The shaded regions represent 95% confidence intervals around the regression lines.

note, we did not observe genetic contributions to the ADI, and therefore no common genetic pathway was estimated). The shared and nonshared environmental regressions of self-rated health on the ADI did not depend on level of area deprivation ($b_{0C}$ = 1.255, $p$ < 0.001; $b_{0E}$ = 0.351, $p$ = 0.039). As is evident in the left panel of Figure 7.13, the between-family contributions to this association are larger than the within-family contribution, although there is a statistically significant quasi-causal effect. Also evident is that shared environmental confounds account for much more of the total phenotypic correlation than within-family factors, as illustrated in the right panel of Figure 7.13.

Illustrative analyses further highlight this shared environmental correlation. In Figure 7.14, we show pair differences in self-rated health as a function of pair differences in the log-transformed ADI within randomly paired individuals (unrelated

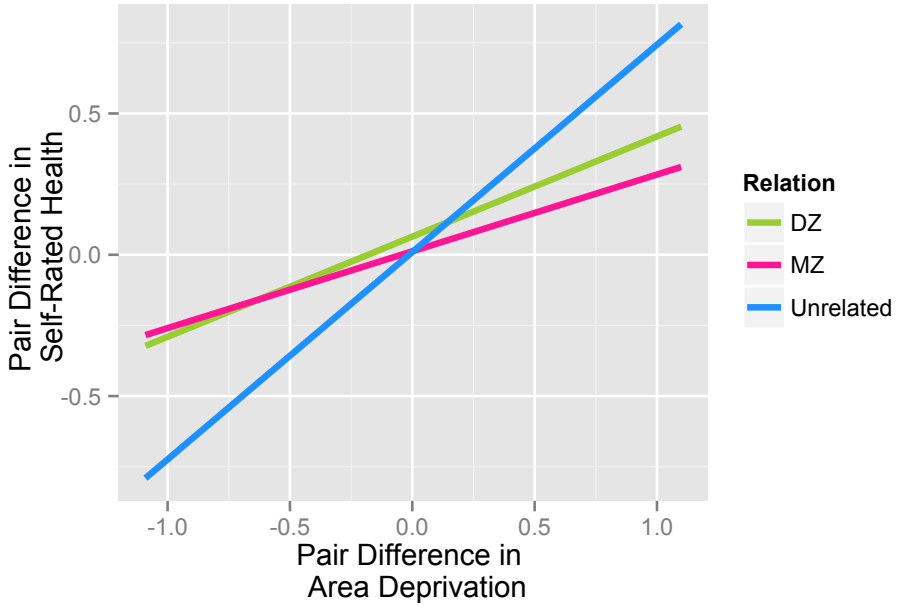


*Figure 7.14.* Illustrative analysis of the main effects of area deprivation on self-rated health in the WSTR. This plot shows pair differences in self-rated health as a function of pair differences in log-transformed ADI in the population (referred to as the ‘‘Unrelated Relation’’) and within pairs of MZ and DZ twins.

relation; blue line) and within MZ (red line) and DZ (green line) pairs. If the protective effect of lower area deprivation on self-rated health was causal, the slopes of these lines would closely approximate one another. Comparison of these lines suggests, however, that differences in the ADI do not predict differences in self-rated health as well within families as they do between them; this effect is instead attenuated within families. The slopes of the MZ and DZ lines are quite close, consistent with a common shared environmental pathway between area deprivation and self-rated health.

**Income Inequality.** At the phenotypic level, income inequality was statistically significantly associated with better overall self-rated health ($b$ = 0.097, $p$ = 0.024), but the effect size was negligible. Individuals at the third quartile of the Gini Index rated their health 0.05 standard deviations higher than individuals at the first quartile of the

Gini Index. The best fitting model suggested that self-rated health was not quasi-causally associated with the ADI ($b_{0E}$ = –0.027, $p$ = 0.695); the genetic ($b_{0A}$ = 0.933, $p$ = 0.593) and shared environmental ($b_{0C}$ = 0.207, $p$ = 0.695) pathways were also not statistically significant, which likely represents lack of power to differentiate between these sources of covariation. When estimating the total between-family effect (achieved by constraining $b_{0A}$ and $b_{0C}$ to be equal), however, there was significant positive between-family confounding between income inequality and self-rated health ($b_{0A}$ = $b_{0C}$ = 0.384, $p$ = 0.003; $b_{0E}$ = –0.018, $p$ = 0.776; results from this model not presented in Table 7.2). These results suggest that the relation between earned income and self-rated health is best explained by between-family factors that are common to both phenotypes rather than by systematic differences in exposure to socioeconomic factors. Figure 7.15 illustrates

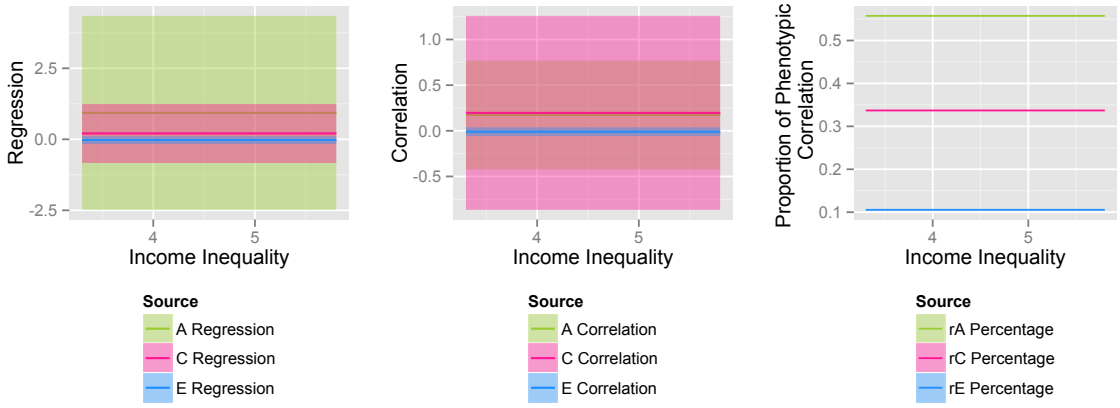


*Figure 7.15.* Main effects of income inequality on self-rated health in the WSTR. The left panel shows the regression of self-rated health on county-level income inequality. The same relation is presented in the middle panel as additive genetic (green), shared environmental (pink), and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic, shared environmental, and nonshared environmental correlations. The asterisks (*) indicate that the magnitude of the regression of self-rated health on income inequality depends on level of income inequality (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). The shaded regions represent 95% confidence intervals around the regression lines.

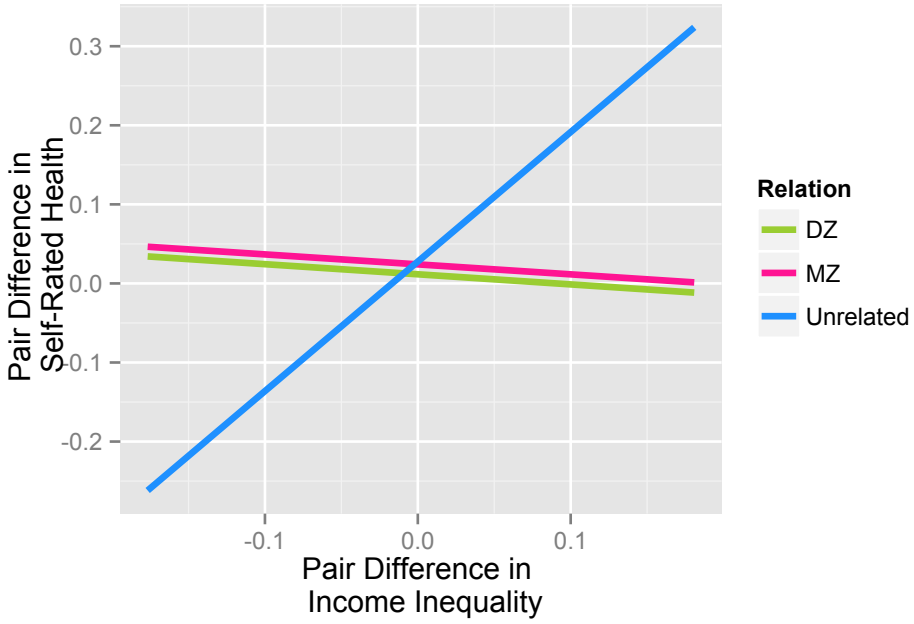


*Figure 7.16.* Illustrative analysis of the main effects of income inequality on self-rated health in the WSTR. This plot shows pair differences in self-rated health as a function of pair differences in log-transformed ADI in the population (referred to as the ‘‘Unrelated Relation’’) and within pairs of MZ and DZ twins.

these results; evident in this figure is that the between-family contributions to the association between income inequality and self-rated health are larger than the within-family contribution. The illustrative analysis in Figure 7.16 further illustrates the main effect of income inequality on self-rated health.

**G×E Interaction: Effect of SES on Genetic and Environmental Etiology of Self-Rated Health (Dissertation Aim 2).** The moderating effects of SES on variance in self-rated health are presented in Table 7.2 under the heading *Effect of SES on Residual ACE Components of Self-Rated Health.* We discuss the interactive effect of each SES indicator on variance in self-rated health below.

**Education.** The best fitting model suggested that residual phenotypic variance in self-rated health decreased with increasing educational attainment. This effect was

driven by decreasing shared environmental variance ($b_{1Cu}$ = –0.104, $p < 0.001$); there was no moderation of the genetic or nonshared environmental variance as a function of educational attainment. These model results are illustrated in Figure 7.17, which shows residual ACE variance components of self-rated health as a function of educational attainment, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure clearly demonstrates how residual phenotypic and shared environmental variance in self-rated health decreases with increasing educational attainment. Considered within the context of the dynamics of the ACE correlations, it does not appear that the presence of factors contributing to self-rated health depends on level of education, suggesting that education is acting to restrict shared environmental variation in self-rated health. Stated differently, high-SES environments appear to protect against shared environmental risk for low general health.

To illustrate these results, we plotted absolute pair differences in self-rated health for MZ and DZ twins against pair-average educational attainment (see the left panel of Figure 7.18). Evident in this plot is that the distance between the MZ and DZ regression lines remains stable across all levels of education, consistent with stable additive genetic variance in self-rated health as a function of educational attainment. The slope of the MZ line is approximately zero, consistent with nonshared environmental variance that is static with respect to educational attainment. The violin plot in the right panel of Figure 7.18 shows that total phenotypic variance in self-rated health decreases with increasing education. Notably, this decrease in variance appears to be due to fewer observations in the lower tail of the self-rated health distribution at higher SES levels.

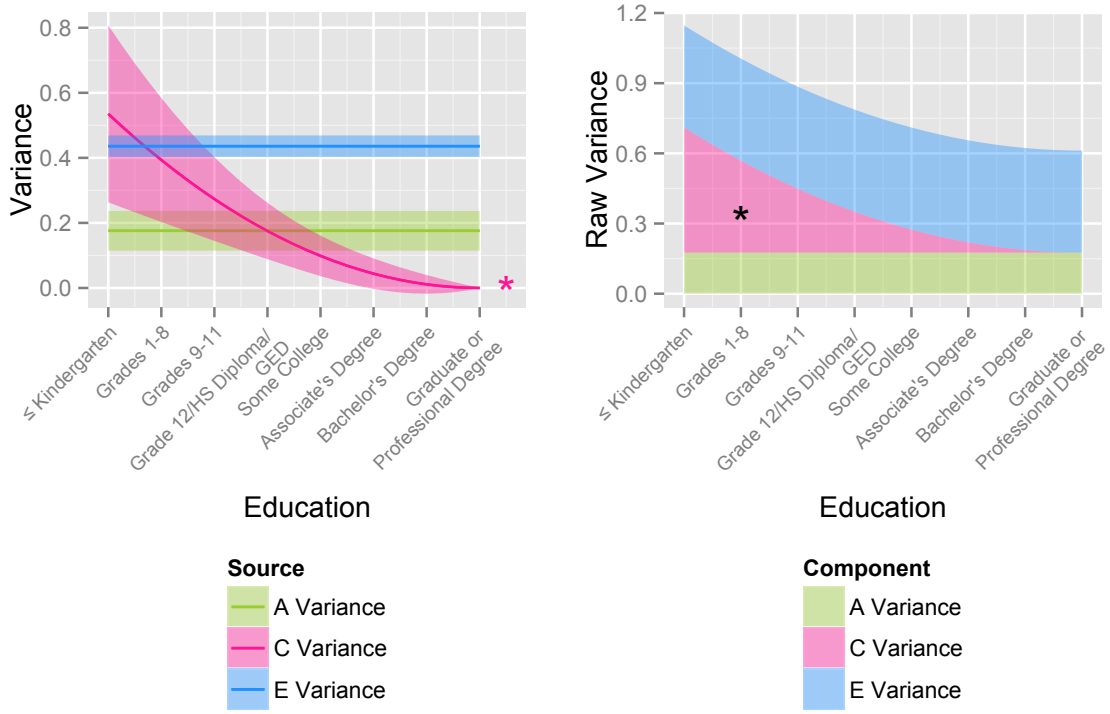


*Figure 7.17.* Gene-by-environment interaction between self-rated health and educational attainment in the WSTR. The left panel shows ACE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in self-rated health decreases as a function of increasing educational attainment. The asterisks (*) indicate that variance in self-rated health depends on level of education.

**Income.** Like with education, the best fitting model suggested that residual phenotypic variance in self-rated health decreased with increasing household income. This effect was driven by decreasing shared ($b_{1Cu}$ = –0.047, $p$ = 0.013) and nonshared environmental variance ($b_{1Eu}$ = –0.015, $p$ < 0.001). There was no moderation of the genetic variance as a function of educational attainment. These model results are illustrated in Figure 7.19, which shows residual ACE variance components of self-rated health as a function of household income, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure clearly demonstrates how residual phenotypic and nonshared environmental variance in self-rated health decreases

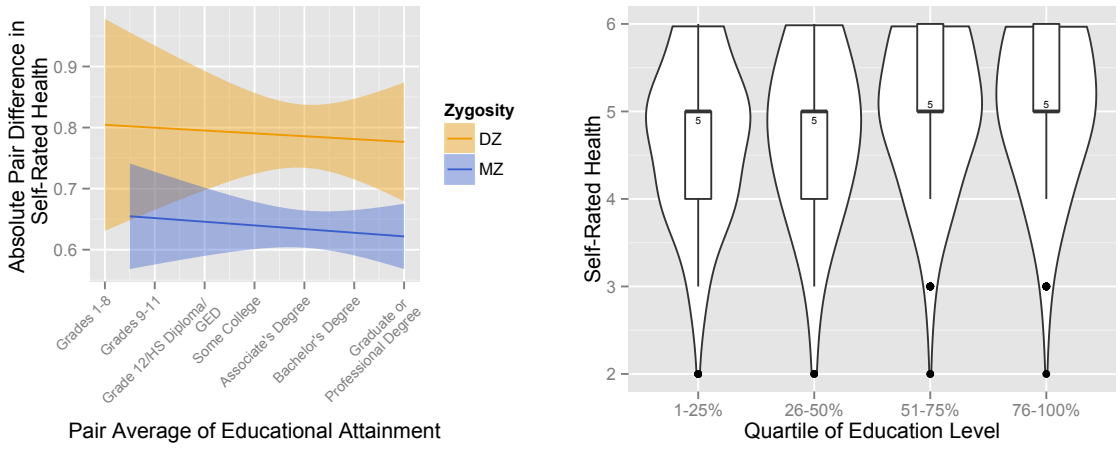


*Figure 7.18.* Illustrative analysis of the effects of educational attainment on variance in self-rated health in the WSTR. The left panel shows absolute pair differences in self-rated health as a function of pair-average educational attainment. The distance between the MZ and DZ lines represents the additive genetic variance in self-rated health. The location of the MZ line represents nonshared environmental variance in self-rated health. The right panel shows box plots overlaid with violin plots, self-rated health as a function of quartile of educational level.

with increasing household income. Although shared environmental variance also decreased with increasing income, the overall contribution of the shared environment did not appear to be distinguishable from zero across the household income spectrum. The nonshared environmental correlation was static with respect to household income, suggesting that low household income potentiates environmental risk for poor health.

These results are further illustrated in Figure 7.20. As we observed with educational attainment, total variance in self-rated health decreases as a function of increasing household income. Consistent with decreasing nonshared environmental variance, the slope of the MZ regression line (left panel of Figure 7.20) is negative. The violin plots (right panel of Figure 7.20) show that this constrained variance effect appears to be the result of fewer observations at the lower end of self-rated health when income level is high.

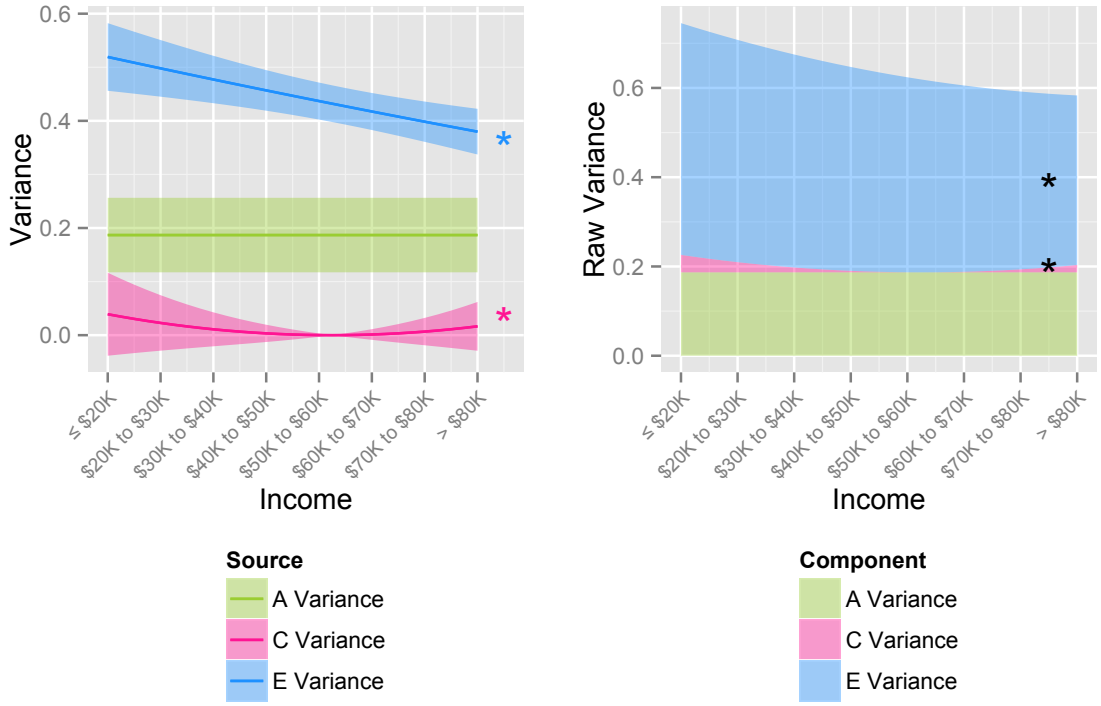


*Figure 7.19.* Gene-by-environment interaction between self-rated health and household income in the WSTR. The left panel shows ACE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in self-rated health decreases as a function of increasing household income. The asterisks (*) indicate that variance in self-rated health depends on income level.

**Area Deprivation.** As was the case with compositional measures of SES, the best fitting model suggested that residual phenotypic variance in self-rated health decreased with decreasing area deprivation. This effect was driven by decreasing shared ($b_{1Cu}$ = –1.187, $p$ < 0.001; this effect reversed at higher levels of the log-transformed ADI) and nonshared environmental variance ($b_{1Eu}$ = –0.227, $p$ = 0.013). There was no moderation of the genetic variance as a function of area deprivation. These model results are illustrated in Figure 7.21, which shows residual ACE variance components of self-rated health as a function of household income, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure illustrates the decrease in

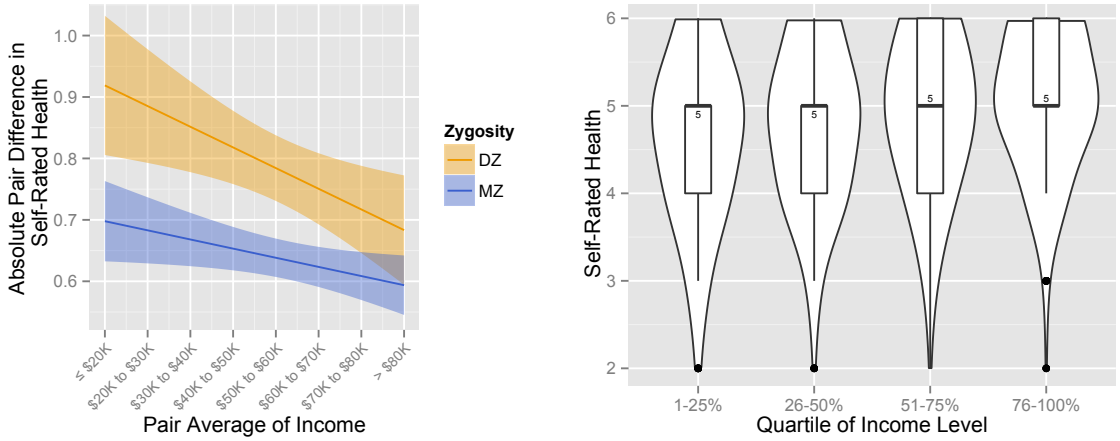


*Figure 7.20.* Illustrative analysis of the effects of household income on variance in self-rated health in the WSTR. The left panel shows absolute pair differences in self-rated health as a function of pair-average household income. The distance between the MZ and DZ lines represents the additive genetic variance in self-rated health. The location of the MZ line represents within-family variance in self-rated health. The right panel shows box plots overlaid with violin plots, self-rated health as a function of quartile of income level.

residual phenotypic and environmental variance in self-rated health as a function of decreasing area deprivation. Although the shared environmental variance actually increased at the lowest levels of area deprivation, its contribution to total phenotypic variance remained small (and mostly indistinguishable from zero) throughout the range of ADI scores. The nonshared environmental correlation was static with respect to household income, suggesting that high area deprivation potentiates environmental risk for poor self-rated health.

These results are further illustrated in Figure 7.22. As we observed with educational attainment and household income, total variance in self-rated health decreases as a function of increasing neighborhood SES. Consistent with decreasing nonshared environmental variance, the slope of the MZ regression line (left panel of Figure 7.22) is negative. The violin plots (right panel of Figure 7.22) show that this

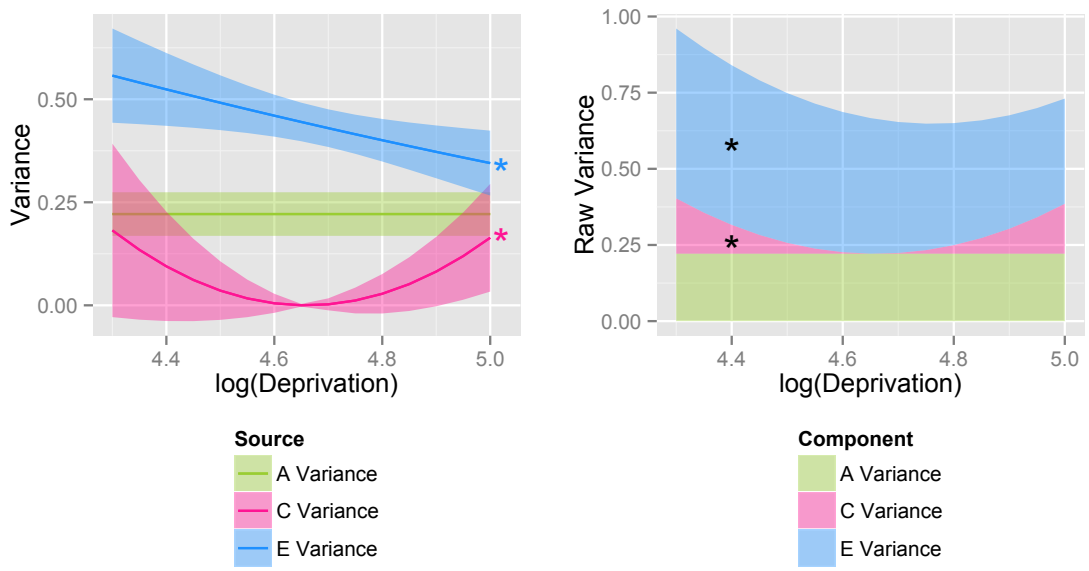


*Figure 7.21.* Gene-by-environment interaction between self-rated health and area deprivation in the WSTR. The left panel shows ACE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in self-rated health decreases as a function of decreasing area deprivation. The asterisks (*) indicate that variance in self-rated health depends on deprivation level. *Note:* Approximately 96% of individuals in the WSTR sample had log-transformed ADI scores under 5.0; therefore, we opted to plot he model-predicted results within the range of the majority of individuals in our sample in order to keep the plots representative of the data.

constrained variance effect appears to be the result of fewer observations at the lower end of self-rated health when income level is high.

**Income Inequality.** The best fitting model did not support county-level income inequality as a moderator of residual phenotypic variance (or residual ACE variances) in self-rated health. It does not appear that income inequality affects perceived health status.

**Brief Summary.** Both compositional and contextual measures of socioeconomic status influenced mean levels of self-rated health, and operated through both genetic and environmental pathways. Additive genetic and shared environmental regressions tended

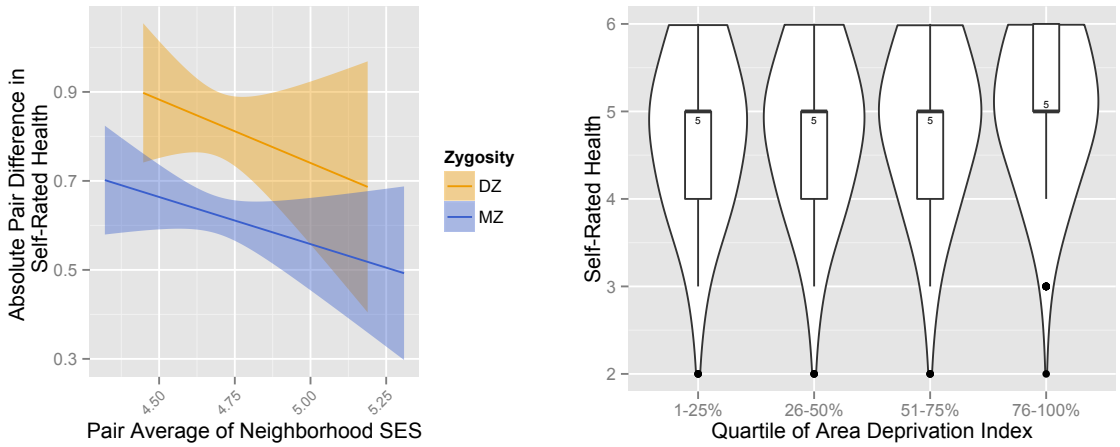


*Figure 7.22*. Illustrative analysis of the effects of neighborhood socioeconomic status on variance in self-rated health in the WSTR. The left panel shows absolute pair differences in self-rated health as a function of pair-average neighborhood SES. The distance between the MZ and DZ lines represents the additive genetic variance in self-rated health. The location of the MZ line represents nonshared environmental variance in self-rated health. The right panel shows box plots overlaid with violin plots, self-rated health as a function of quartile of neighborhood SES.

to have the greatest magnitude, and between-family factors accounted for a much larger proportion of the total phenotypic correlation than did within-family factors. Residual variance in self-rated health was also influenced by compositional and contextual measures of SES. Residual phenotypic variance decreased with increasing socioeconomic status, and this decrease was driven primarily by decreases in shared and nonshared environmental variances. Finally, decreases in residual environmental variances appeared to be potentiated by socioeconomic status, and not by social selection factors.

***Health Conditions—WSTR.*** Individuals in the WSTR sample indicated whether they experience a number of physical health conditions (see Chapter 3). We fit the model presented in Figure 7.23 to the data separately for each socioeconomic status indicator, partialling for the linear effects of age and gender on level of and variance in health

conditions. We used likelihood ratio tests to determine the best fitting model. We first established a baseline model by conducting omnibus tests for the moderation by SES of both the residual variance in health conditions and the main effects of SES on health conditions. We then systematically eliminated parameters from the baseline model to evaluate statistical significance of moderation parameters. Biometric decomposition of both the SES indicators and health conditions are presented in Table 4.2, and parameter estimates and model fit statistics for the baseline and best-fitting models are presented in Table 7.3. The Area Deprivation Index was log-transformed to correct for positive skew, and the Gini Index was scaled by a factor of 10 to facilitate model convergence. These transformed values were used in all analyses in which the ADI or Gini Index was used as the indicator of contextual socioeconomic status. We did not include A for the ADI since there was no evidence of genetic influences on area deprivation in our univariate model. Likewise, C for health conditions was not included in any of the models.

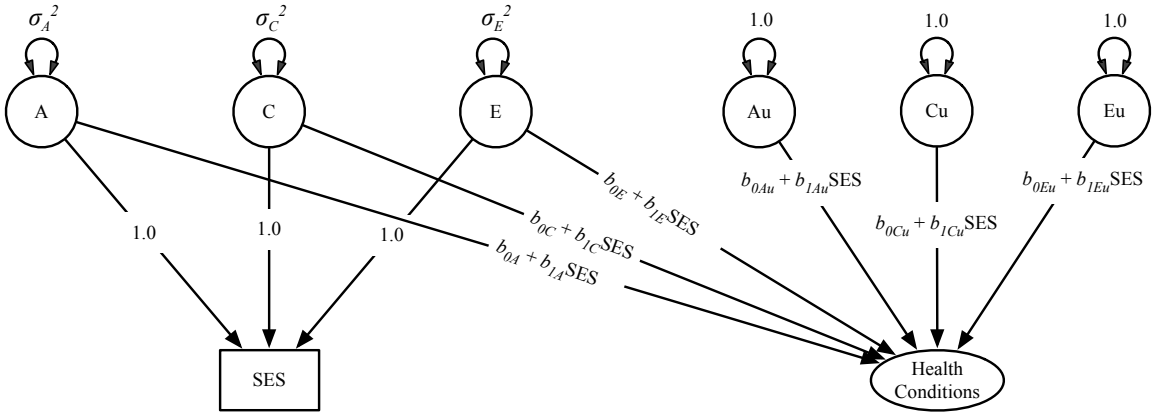


*Figure 7.23.* Path diagram G×SES model fit to the health conditions factor in the WSTR. (only one twin shown for clarity). In the fully saturated model, the main effects of SES on health conditions and the ACE variances of self-rated health vary as a function of SES. The residual variances for the health conditions items were permitted to correlate across twins, and were estimated freely according to zygosity.

Table 7.3. Parameter estimates and model fit statistics for G×SES models, health conditions in the WSTR.

| | Parameter | Phenotypic Model | Quasi-Causal Model (No Moderation) | Moderation of Residual Variance | Moderation of Main Effects | Best-Fitting Model |
|---|---|---|---|---|---|---|
| **Education** | Main Effect of Education on Health Conditions | | | | | |
| | $b_{0A}$ | **–.028 (.014)** | ***–.099 (.023)*** | ***–.101 (.023)*** | .071 (.091) | ***–.100 (.023)*** |
| | $b_{1A}$ | — | — | — | –.030 (.015) | — |
| | $b_{0E}$ | **–.028 (.014)** | .034 (.020) | .034 (.020) | .030 (.095) | .033 (.020) |
| | $b_{1E}$ | — | — | — | .001 (.015) | — |
| | Effect of Education on Residual ACE Components of Health Conditions | | | | | |
| | $b_{0Au}$ | .041 (.078) | .040 (.078) | .181 (.110) | .204 (.111) | .045 (.078) |
| | $b_{1Au}$ | — | — | –.024 (.014) | –.027 (.014) | — |
| | $b_{0Eu}$ | ***.327 (.063)*** | ***.336 (.063)*** | .161 (.090) | .159 (.090) | .112 (.086) |
| | $b_{1Eu}$ | — | — | **–.032 (.012)** | –.032 (.012) | ***–.040 (.011)*** |
| | Model Fit | | | | | |
| | *-2LL* | — | 562259.622 | 562242.890 | 562238.428 | 562245.864 |
| | *Δ-2LL (Δdf)* | — | — | 16.732 (+2) | 4.462 (+1) | 2.974 (–1) |
| | *p* | — | — | <.001† | .107 | .085 |
| **Household Income** | Main Effect of Income on Health Conditions | | | | | |
| | $b_{0A}$ | –.012 (.006) | ***–.040 (.013)*** | ***–.039 (.013)*** | **–.055 (.029)** | ***–.039 (.013)*** |
| | $b_{1A}$ | — | — | — | .003 (.005) | — |
| | $b_{0E}$ | –.012 (.006) | .003 (.008) | .002 (.008) | .008 (.019) | .002 (.008) |
| | $b_{1E}$ | — | — | — | –.001 (.004) | — |
| | Effect of Income on Residual ACE Components of Health Conditions | | | | | |
| | $b_{0Au}$ | .031 (.080) | .030 (.081) | .121 (.083) | .119 (.083) | .121 (.083) |
| | $b_{1Au}$ | — | — | ***–.021 (.007)*** | ***–.021 (.007)*** | ***–.021 (.007)*** |
| | $b_{0Eu}$ | ***.333 (.066)*** | ***.337 (.065)*** | ***.285 (.068)*** | ***.286 (.068)*** | ***.285 (.068)*** |
| | $b_{1Eu}$ | — | — | ***–.016 (.007)*** | **–.016 (.007)** | ***–.016 (.007)*** |
| | Model Fit | | | | | |
| | *-2LL* | — | 544080.414 | 544053.718 | 544053.280 | 544053.718 |
| | *Δ-2LL (Δdf)* | — | — | 26.696 (+2) | .438 (+2) | — |
| | *p* | — | — | <.001† | .803 | — |
| **Area Deprivation** | Main Effect of Area Deprivation on Health Conditions | | | | | |
| | $b_{0E}$ | –.061 (.173) | | –.250 (.170) | 1.127 (2.588) | .144 (.169) |
| | $b_{1E}$ | — | | — | –.266 (.536) | — |
| | Effect of Area Deprivation on Residual ACE Components of Health Conditions | | | | | |
| | $b_{0Au}$ | .010 (.108) | | .065 (1.773) | .785 (.764) | .022 (.109) |
| | $b_{1Au}$ | — | | –.115 (.387) | –.176 (.167) | — |
| | $b_{0Eu}$ | ***.445 (.081)*** | | ***1.690 (.462)*** | **1.471 (.683)** | **1.475 (.651)** |
| | $b_{1Eu}$ | — | | **–.240 (.099)** | ***–.417 (.146)*** | ***–.416 (.139)*** |
| | Model Fit | | | | | |
| | *-2LL* | 280139.802 | | 280130.210 | 280129.964 | 280131.514 |
| | *Δ-2LL (Δdf)* | — | | 9.592 (+2) | .246 (+1) | 1.304 (–1) |
| | *p* | — | | .008† | .620 | .253 |
| **Income Inequality** | Main Effect of Income Inequality on Health Conditions | | | | | |
| | $b_{0A}$ | –.027 (.066) | –.471 (.291) | –.466 (.291) | | –.467 (.291) |
| | $b_{1A}$ | — | — | — | | — |
| | $b_{0E}$ | –.027 (.066) | .025 (.072) | .018 (.072) | | .018 (.072) |
| | $b_{1E}$ | — | — | — | | — |
| | Effect of Income Inequality on Residual ACE Components of Health Conditions | | | | | |
| | $b_{0Au}$ | .099 (.088) | .098 (.088) | .374 (.309) | | .083 (.089) |
| | $b_{1Au}$ | — | — | –.066 (.067) | | — |
| | $b_{0Eu}$ | ***.302 (.069)*** | ***.304 (.069)*** | .197 (.273) | | .306 (.248) |
| | $b_{1Eu}$ | — | — | –.115 (.060) | | **–.138 (.054)** |
| | Model Fit | | | | | |
| | *-2LL* | — | 439381.292 | 439373.974 | | 439374.942 |
| | *Δ-2LL (Δdf)* | — | — | 7.318 (+2) | | .968 (–1) |
| | *p* | — | — | .026† | | .325 |

*Note:* Standard errors presented within parentheses. Estimates p < .05 **bolded**. Estimates p < .01 *italicized*. Baseline model denoted by †.

**Causal Pathways vs. rGE: Main Effects of SES on Health Conditions (Dissertation Aim 1).** The main effects of SES on health conditions are presented in

Table 7.3 under the heading *Main Effect of Moderator on Health Conditions*. We discuss the main effects results for each moderator below.

**Education.** Education demonstrated a phenotypic association with the health conditions factor ($b$ = –0.028, $p$ = 0.044). Controlling for age and gender, individuals with a graduate or professional degree had scores on the health conditions factor that averaged 0.18 standard deviations lower than individuals with a high school degree. The best-fitting model suggested that this association is at least partially mediated by a common genetic background ($b_{0A}$ = –0.100, $p < 0.001$); the quasi-causal pathway was not statistically significant ($b_{0E}$ = 0.033, $p$ = 0.094). As illustrated in the left panel of Figure 7.24, which shows the magnitude of the genetic and nonshared environmental regressions

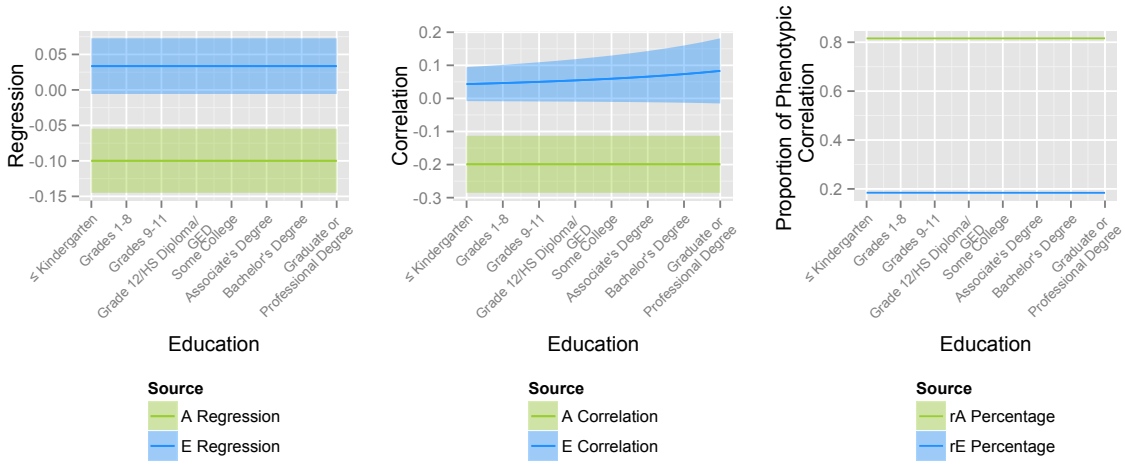


*Figure 7.24*. Main effects of educational attainment on health conditions in the WSTR. The left panel shows the regression of health conditions on educational attainment. The same relation is presented in the middle panel as genetic (green) and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic and nonshared environmental correlations. Asterisks (*) indicate that the magnitude of the regression of health conditions on educational attainment depends on level of education (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). Shaded regions represent 95% confidence intervals around regression lines.

of health conditions on educational attainment, the between-family regression is larger than the within-family regression. These regression lines are re-represented as genetic and nonshared environmental correlations in the middle panel of Figure 7.24. We also present the proportions of the total phenotypic correlation accounted for by the genetic and nonshared environmental correlations in the right panel of Figure 7.24.

To demonstrate what these effects look like within and between twins, we conducted an illustrative analysis of the main effect of educational attainment on health conditions and how it varies according to the level of educational attainment, which we present in Figure 7.25. We identified twin pairs concordant for lower education (up to Associate's degree; orange), pairs concordant for higher education (Bachelor's degree or higher; navy), and pairs discordant for educational status, and compared the mean number of health conditions endorsed by members of each of these groups. Overall, there is a main effect of educational attainment on health conditions such that higher education is associated with fewer health conditions on average (this is evident comparing the height of the bars in the left panel to those in the right panel). Among MZ twin pairs concordant for higher educational attainment, however, there are virtually no education-based differences in number of health conditions endorsed; DZ twins show a larger, but similarly small, difference, consistent with the presence of genetic-based selection effect.

**Income.** The phenotypic regression of health conditions on household income was small and did not reach statistical significance ($b$ = –0.012, $p$ = 0.053). The best-fitting model suggested that this association (although phenotypically nonsignificant) is driven by a common genetic background ($b_{0A}$ = –0.040, $p$ = 0.003; $b_{0E}$ = 0.002, $p$ =

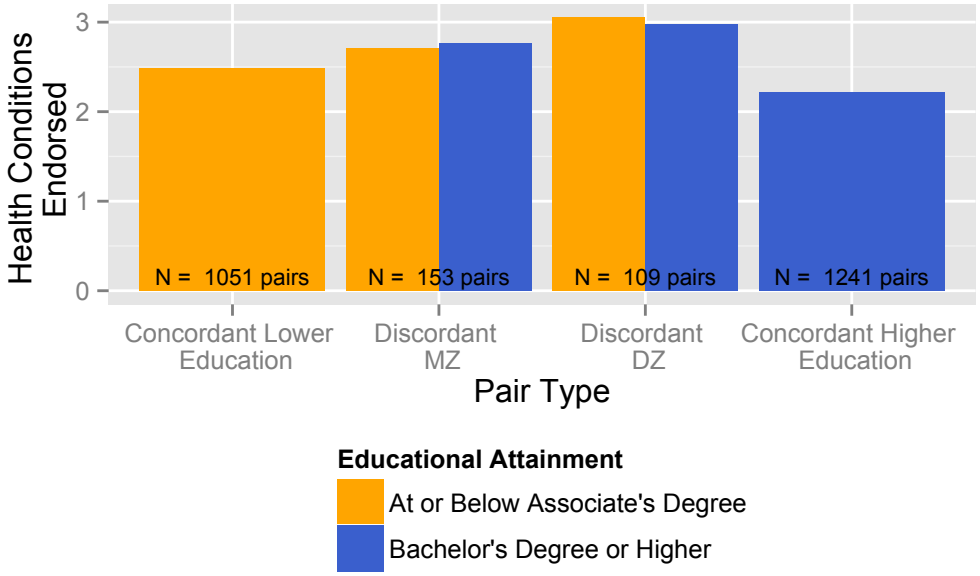


*Figure 7.25*. Illustrative analysis of the main effects of educational attainment on health conditions in the WSTR. The twin with less education (orange) is compared with the twin with more education (navy) in MZ and DZ twin pairs.

0.801). As is evident in the left panel of Figure 7.18, the between-family contributions to this association are larger than the within-family contribution. Between-family confounds collectively account for nearly all of the total phenotypic correlation (see the right panel of Figure 7.26). Illustrative analyses further highlight this shared environmental correlation. In Figure 7.27, we compare mean health conditions endorsed among twin pairs concordant for lower income (at or below $70K; orange), pairs concordant for higher income (above $70K; navy), and pairs discordant for income level. Differences in health conditions endorsed among MZ twins were minimal; DZ twins showed larger differences, consistent with genetic selection.

**Area Deprivation.** At the phenotypic level, the ADI was not statistically significantly associated with the health conditions factor ($b = –0.082$, $p = 0.624$).

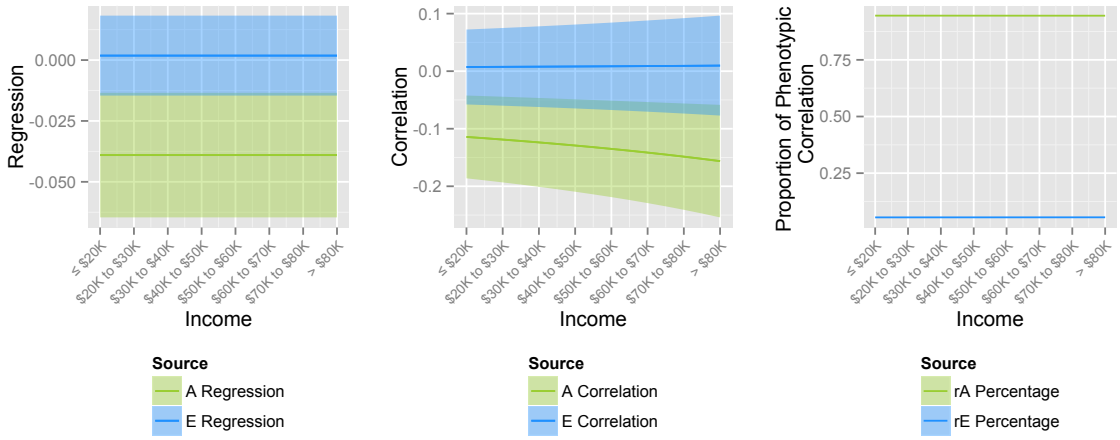


*Figure 7.26*. Main effects of household income on health conditions in the WSTR. The left panel shows the regression of health conditions on household income. The same relation is presented in the middle panel as genetic (green) and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic and nonshared environmental correlations. The asterisks (*) indicate that the magnitude of the regression of health conditions on household income depends on level of education (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). The shaded regions represent 95% confidence intervals around the regression lines.

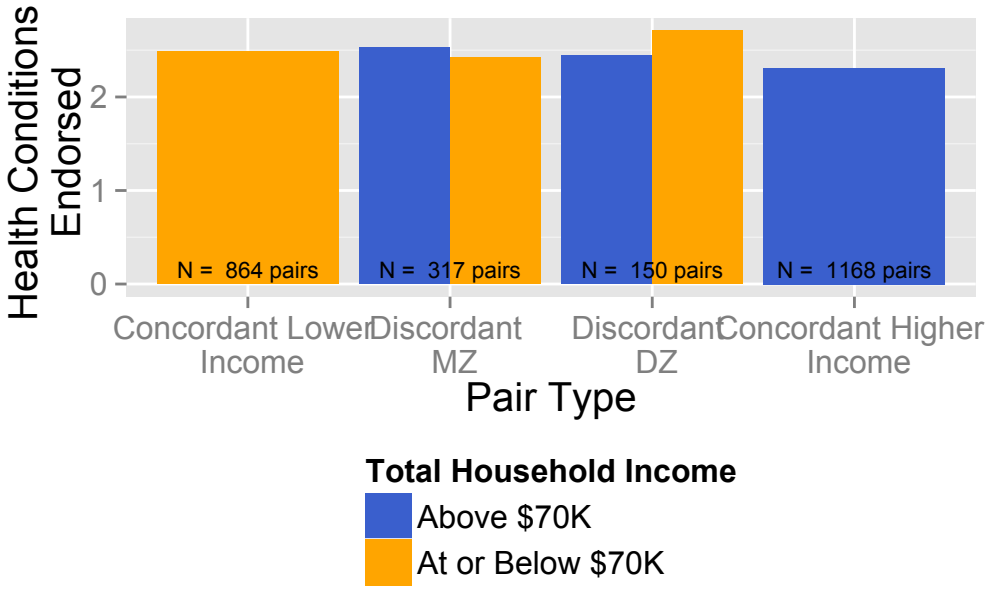


*Figure 7.27*. Illustrative analysis of the main effects of household income on health conditions in the WSTR. The twin with less income (orange) is compared with the twin earning more (navy) in MZ and DZ twin pairs.

**Income Inequality.** At the phenotypic level, income inequality also was not statistically significantly associated with the health conditions factor ($b$ = –0.027, $p$ = 0.675).

**G×E Interaction: Effect of SES on Genetic and Environmental Etiology of Health Conditions (Dissertation Aim 2).** The moderating effects of SES on variance in self-rated health are presented in Table 7.3 under the heading *Effect of SES on Residual ACE Components of Health Conditions.* We discuss the interactive effect of each SES indicator on variance in the health conditions factor below.

**Education.** The best fitting model suggested that residual phenotypic variance in the health conditions factor decreased with increasing educational attainment. This effect was driven by decreasing nonshared environmental variance ($b_{1Eu}$ = –0.040, $p < 0.001$); there was no moderation of the genetic variance as a function of educational attainment. These model results are illustrated in Figure 7.28, which shows residual ACE variance components of the health conditions factor as a function of educational attainment, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure clearly demonstrates how residual phenotypic and nonshared environmental variance in health conditions decreases with increasing educational attainment. Considered within the context of the dynamics of the ACE correlations, it does not appear that the presence of factors contributing to health conditions depends on level of education, suggesting that education is indeed acting to restrict nonshared environmental variation in health conditions. Stated differently, high-SES environments appear to protect against nonshared environmental risk for endorsing health conditions.

To illustrate these results, we plotted absolute pair differences in total health

conditions endorsed for MZ and DZ twins against pair-average educational attainment (see the left panel of Figure 7.29). Evident in this plot is that the distance between the MZ and DZ regression lines decreases as a function of education level, an effect driven by DZ twins being more similar at higher levels of education. This is consistent with decreasing between-family variance as a function of educational attainment. Our model results showed decreased nonshared environmental variance in health conditions as a function of education, however, which may be attributable to the fact that the analysis was on variance residual to specific health factors. The violin plot in the right panel of Figure 7.29 illustrates decreased total phenotypic variance in health conditions as a

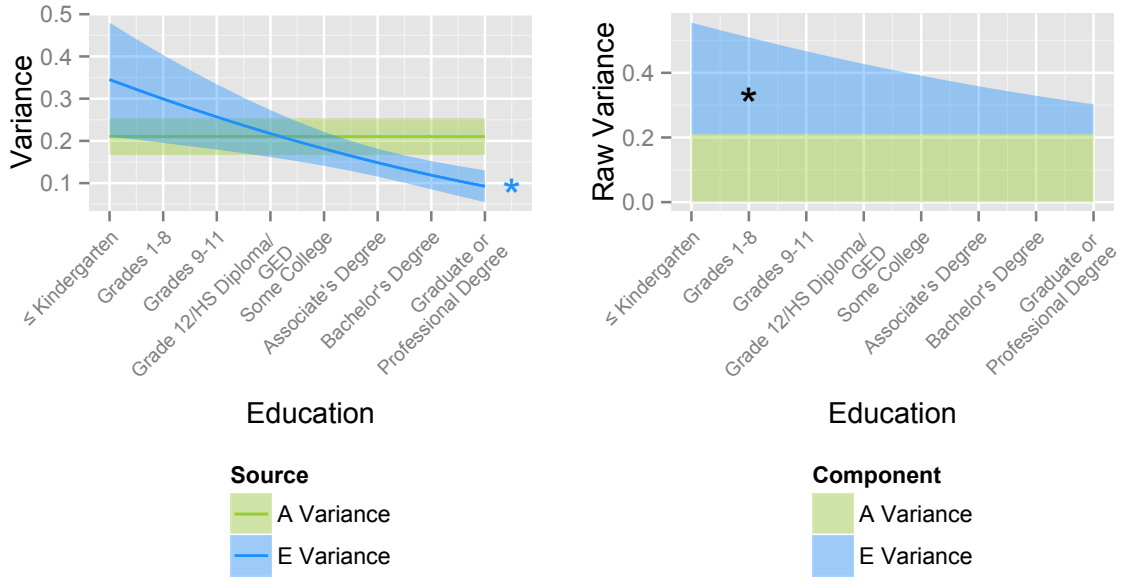


*Figure 7.28*. Gene-by-environment interaction between the health conditions factor and educational attainment in the WSTR. The left panel shows AE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in health conditions decreases as a function of increasing educational attainment. The asterisks (*) indicate that variance in the health conditions factor depends on level of education.

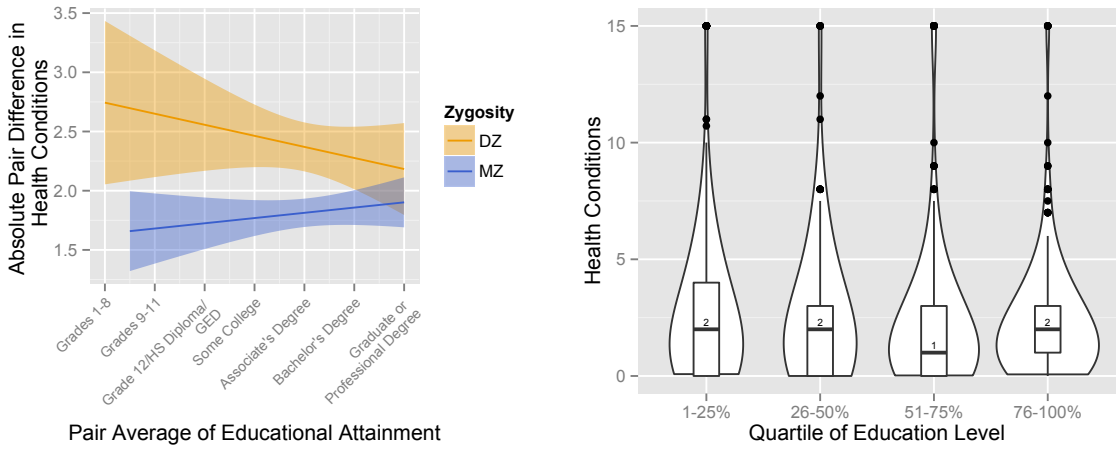


*Figure 7.29.* Illustrative analysis of the effects of educational attainment on variance in health conditions in the WSTR. The left panel shows absolute pair differences in total health conditions endorsed as a function of pair-average educational attainment. The distance between the MZ and DZ lines represents the additive genetic variance in health conditions. The location of the MZ line represents nonshared environmental variance in health conditions. The right panel shows box plots overlaid with violin plots, health conditions as a function of quartile of educational level.

function of increasing education. Notably, this decrease in variance appears to be due to fewer observations in the upper tail of the health conditions distribution at higher SES levels.

**Income.** Like with education, the best fitting model suggested that residual phenotypic variance in the health conditions factor decreased with increasing household income. This effect was driven by decreasing genetic ($b_{1Au}$ = –0.021, $p$ = 0.002) and nonshared environmental variance ($b_{1Eu}$ = 0.016[23], $p$ = 0.014). There was no moderation of the genetic variance as a function of educational attainment. These model results are illustrated in Figure 7.30. The genetic and nonshared environmental correlations were static with respect to household income, suggesting that low household income

[23] Although this coefficient is positive, nonshared environmental variance in fact was decreasing as a function of income level, holding gender and age constant.

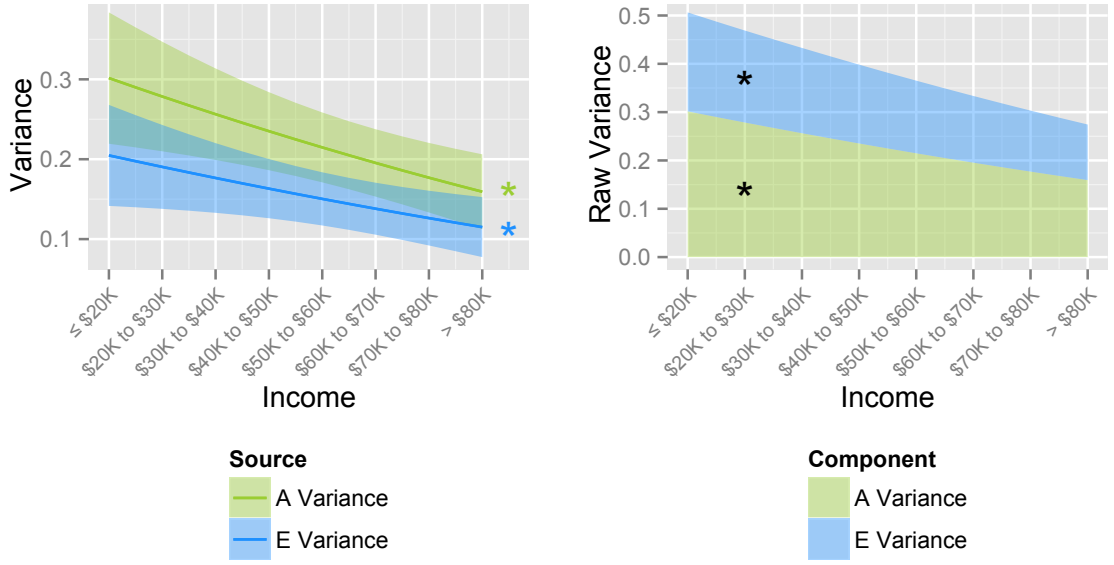


*Figure 7.30*. Gene-by-environment interaction between health conditions and household income in the WSTR. The left panel shows AE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in the health conditions factor decreases as a function of increasing household income. The asterisks (*) indicate that variance in health conditions depends on income level.

potentiates genetic and environmental risk for certain health conditions.

These results are further illustrated in Figure 7.31. Evident in this plot is that the distance between the MZ and DZ regression lines decreases as a function of income level, an effect driven by MZ twins being less similar at higher levels of income. This is consistent with decreasing additive genetic variance as a function of income. Our model results also showed decreased nonshared environmental variance in health conditions as a function of income, however, which may be attributable to the fact that the analysis was on variance residual to specific health factors. The violin plots show that this constrained variance effect appears to be the result of fewer observations at the upper tail of health conditions when income level is high.

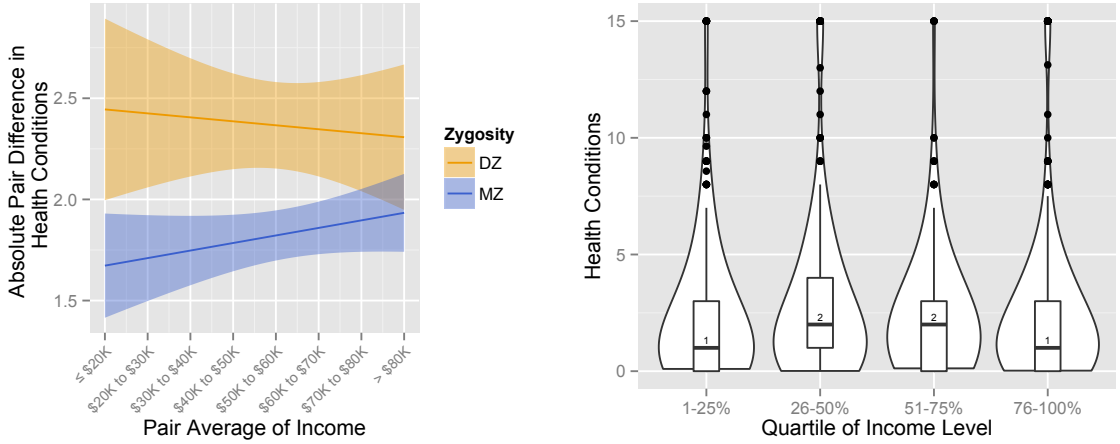


*Figure 7.31.* Illustrative analysis of the effects of household income on variance in health conditions in the WSTR. The left panel shows absolute pair differences in total health conditions endorsed as a function of pair-average household income. The distance between the MZ and DZ lines represents the additive genetic variance in health conditions. The location of the MZ line represents nonshared environmental variance in health conditions. The right panel shows box plots overlaid with violin plots, health conditions as a function of quartile of income level.

**Area Deprivation.** As was the case with compositional measures of SES, the best fitting model suggested that residual phenotypic variance in the health conditions factor decreased with decreasing area deprivation, an effect driven by decreasing nonshared environmental variance ($b_{1Eu}$ = –0.416, $p$ = 0.003). There was no moderation of the genetic variance as a function of area deprivation. These model results are illustrated in Figure 7.32. The nonshared environmental correlation was static with respect to household income, suggesting that high area deprivation potentiates environmental risk for certain health conditions.

These results are further illustrated in Figure 7.33. Evident in this plot is that the distance between the MZ and DZ regression lines decreases as a function of neighborhood SES, an effect driven by DZ twins being less similar at higher levels of SES. This is consistent with decreasing additive genetic variance as a function of

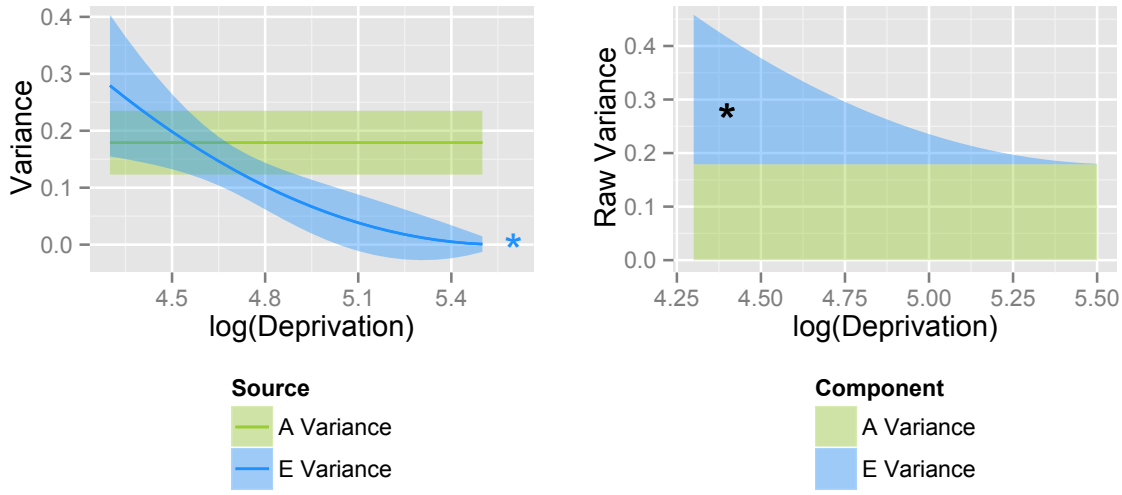


*Figure 7.32*. Gene-by-environment interaction between health conditions and area deprivation in the WSTR. The left panel shows AE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in the health conditions factor decreases as a function of decreasing area deprivation. The asterisks (*) indicate that variance in health conditions depends on income level.

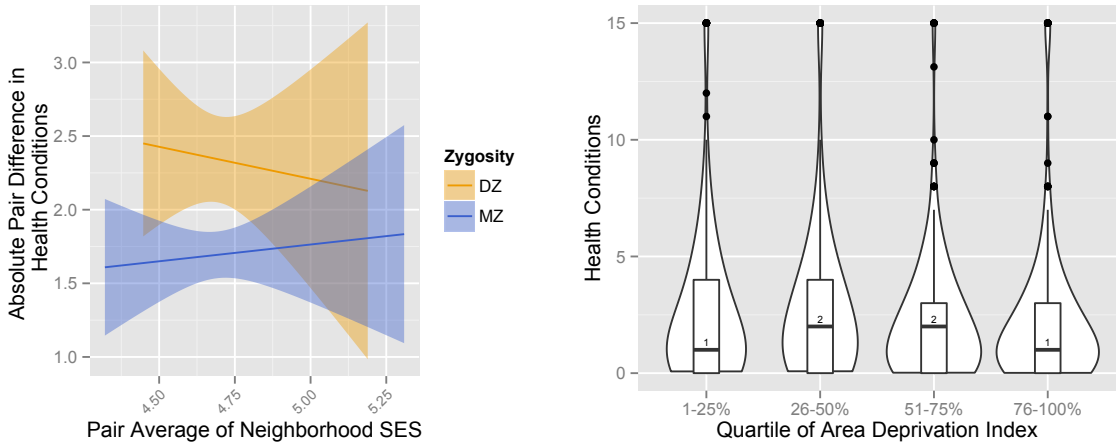


*Figure 7.33*. Illustrative analysis of the effects of neighborhood socioeconomic advantage on variance in health conditions in the WSTR. The left panel shows absolute pair differences in total health conditions endorsed as a function of pair-average neighborhood SES. The distance between the MZ and DZ lines represents the additive genetic variance in health conditions. The location of the MZ line represents nonshared environmental variance in health conditions. The right panel shows box plots overlaid with violin plots, health conditions as a function of quartile of neighborhood SES.

neighborhood SES. Our model results showed decreased nonshared environmental variance in health conditions as a function of neighborhood SES, however, which may be attributable to the fact that the analysis was on variance residual to specific health factors. The violin plots show that this constrained variance effect appears to be the result of fewer observations at the upper tail of health conditions when neighborhood SES is high.

**Income Inequality.** The best fitting model suggested that residual phenotypic variance in health conditions decreased with increasing income inequality. As with the ADI, this effect was driven by decreasing nonshared environmental variance ($b_{1Eu}$ = –0.138, $p$ = 0.011). There was no moderation of the genetic variance as a function of income inequality. These model results are illustrated in Figure 7.34. The nonshared environmental correlation was static with respect to household income, suggesting that high area deprivation potentiates environmental risk for certain health conditions.

These results are further illustrated in Figure 7.35. Nonshared environmental variance (but not additive genetic variance) in health conditions decreases as a function of increasing income inequality. The violin plots show that total phenotypic variance in health conditions decreases with increasing income inequality, an effect that appears to be the result of fewer observations in the upper tail of health conditions at high levels of income inequality.

**Brief Summary.** Although compositional measures of socioeconomic status appeared to influence frequencies of health conditions endorsed, contextual SES measures were not related. Compositional SES measures operated through common genetic pathways; that is, between-family factors accounted for a much larger proportion of the total phenotypic correlation than did within-family factors. Residual variance in

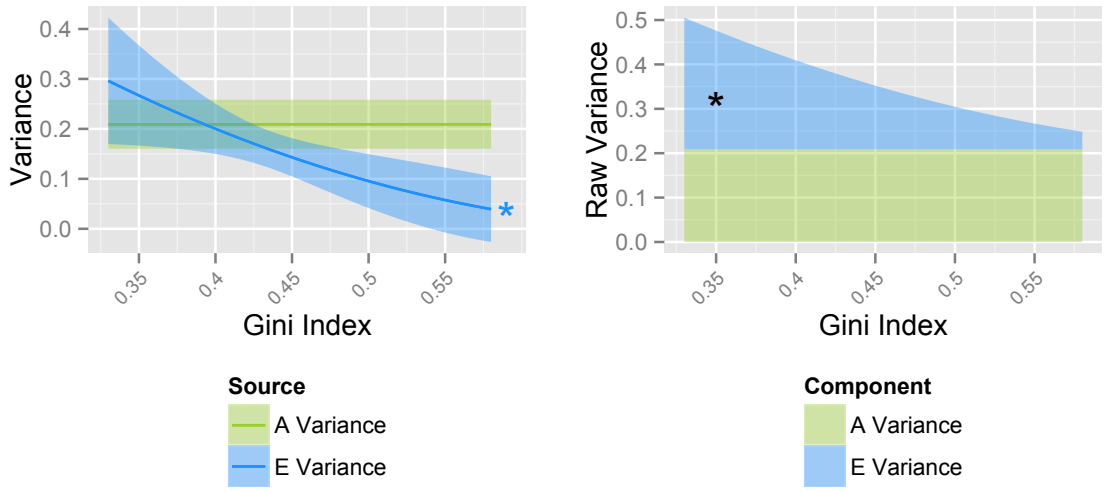


*Figure 7.34*. Gene-by-environment interaction between health conditions and income inequality in the WSTR. The left panel shows AE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in the health conditions factor decreases as a function of increasing income inequality. The asterisks (*) indicate that variance in health conditions depends on income inequality.

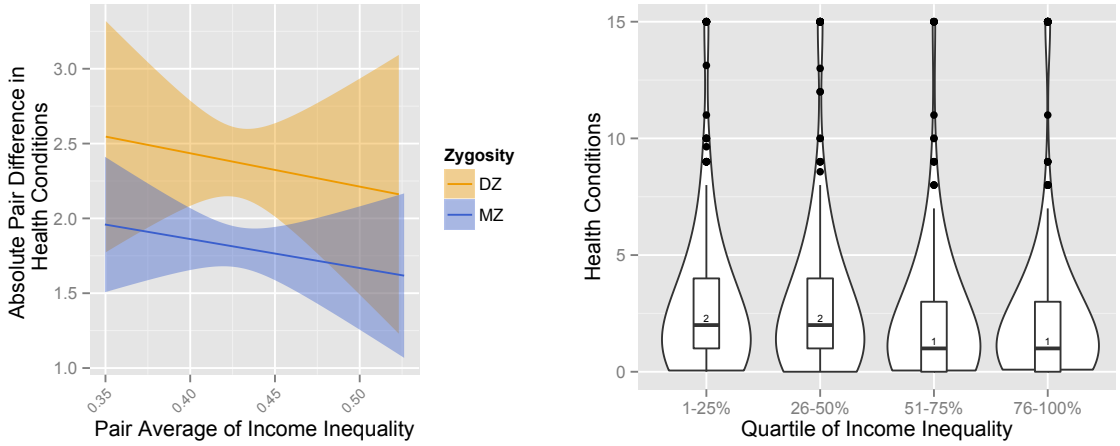


*Figure 7.35*. Illustrative analysis of the effects of income inequality on variance in health conditions in the WSTR. The left panel shows absolute pair differences in total health conditions endorsed as a function of pair-average income inequality. The distance between the MZ and DZ lines represents the additive genetic variance in health conditions. The location of the MZ line represents nonshared environmental variance in health conditions. The right panel shows box plots overlaid with violin plots, health conditions as a function of quartile of income inequality.

self-rated health was influenced by both compositional and contextual measures of SES. Residual phenotypic variance tended to decrease with increasing socioeconomic status, an effect driven primarily by decreases nonshared environmental variance. Finally, decreases in these residual environmental variances appeared to be potentiated by socioeconomic status, and not by social selection factors.

**Body Mass Index**

***Body Mass Index—Add Health.*** Respondents in the Add Health sample reported on their height at weight, from which BMI was calculated. We fit the model presented in Figure 7.36 to the data separately for each socioeconomic status indicator, partialling for the linear effects of age and gender on level and variance in BMI. We used likelihood ratio tests to determine the best fitting model. Biometric decomposition of both the SES indicators and general health are presented in Table 4.1, and parameter estimates for each of these models are presented in Table 7.4. Parental household income was log-transformed to correct for positive skew. Body mass index showed no evidence of influence from shared environmental factors; therefore, we did not include C in any of the models fit to the data.

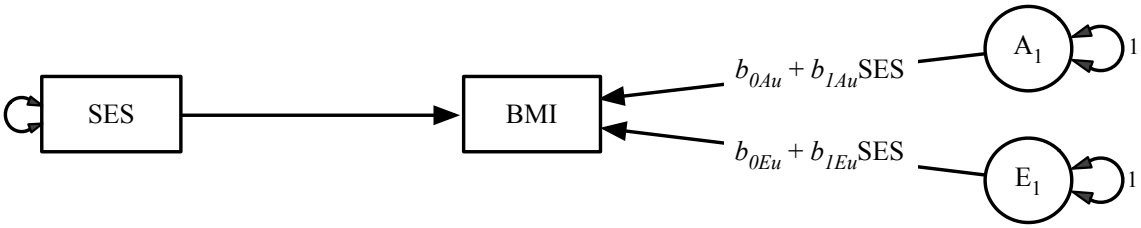


*Figure 7.36*. Path diagram G×SES model fit to body mass index in the Add Health sample (only one twin shown for clarity).

Table 7.4. Parameter estimates and model fit statistics for G×SES models, body mass index in the Add Health sample.

| | Parameter | Phenotypic Model (No Moderation) | Moderation of Residual Variance | Best-Fitting Model |
|---|---|---|---|---|
| **Parental Education** | Main Effect of Parental Education on BMI | | | |
| | $b_1$ | **–.338 (.133)** | ***–.359 (.135)*** | ***–.361 (.135)*** |
| | Effect of Education on Residual ACE Components of BMI | | | |
| | $b_{0Au}$ | **3.972 (1.536)** | ***4.554 (1.536)*** | ***4.708 (1.537)*** |
| | $b_{1Au}$ | — | ***–.323 (.106)*** | ***–.364 (.099)*** |
| | $b_{0Eu}$ | ***3.179 (.910)*** | ***3.442 (.986)*** | ***3.178 (.912)*** |
| | $b_{1Eu}$ | — | –.083 (.079) | — |
| | Model Fit | | | |
| | *-2LL* | 8232.500 | 8217.330 | 8218.464 |
| | *Δ-2LL (Δdf)* | — | 15.170 (+2) | 1.134 (–1) |
| | *p* | — | <.001† | .287 |
| **Family Household Income** | Main Effect of Family Income on BMI | | | |
| | $b_1$ | –.046 (.133) | –.068 (.179) | –.046 (.133) |
| | Effect of Income on Residual ACE Components of BMI | | | |
| | $b_{0Au}$ | ***4.685 (1.744)*** | ***4.207 (1.848)*** | ***4.685 (1.744)*** |
| | $b_{1Au}$ | — | .031 (.197) | — |
| | $b_{0Eu}$ | ***3.488 (.976)*** | ***4.614 (1.598)*** | ***3.488 (.976)*** |
| | $b_{1Eu}$ | — | –.191 (.216) | — |
| | Model Fit | | | |
| | *-2LL* | 7320.578 | 7320.342 | 7320.578 |
| | *Δ-2LL (Δdf)* | — | .236 (+2) | — |
| | *p* | —† | .889 | — |
| **Neighborhood Socioeconomic Advantage** | Main Effect of Neighborhood SES on BMI | | | |
| | $b_1$ | –.304 (.220) | –.312 (.224) | –.304 (.220) |
| | Effect of Neighborhood SES on Residual ACE Components of BMI | | | |
| | $b_{0Au}$ | 2.590 (1.760) | 2.260 (1.781) | 2.590 (1.760) |
| | $b_{1Au}$ | — | –.046 (.172) | — |
| | $b_{0Eu}$ | ***4.049 (1.030)*** | ***4.088 (1.130)*** | ***4.049 (1.030)*** |
| | $b_{1Eu}$ | — | **–.225 (.108)** | — |
| | Model Fit | | | |
| | *-2LL* | 6781.426 | 6775.842 | 6781.426 |
| | *Δ-2LL (Δdf)* | — | 5.584 (+2) | — |
| | *p* | —† | .061 | — |

*Note:* Standard errors presented within parentheses. Estimates p < .05 **bolded**. Estimates p < .01 *italicized*. Baseline model denoted by †.

**Main Effects of SES on Body Mass Index (Dissertation Aim 1).** The main effects of SES on BMI are presented in Table 7.4 under the heading *Main Effect of Moderator on BMI*. We discuss the main effects results for each moderator below.

**Parental Education.** Parental education was significantly associated with lower body mass index ($b$ = –0.338, $p$ = 0.011). Adolescents with a parent who had earned at least a college degree had BMIs that were 0.16 standard deviations lower than their

counterparts whose parent holds a high school degree.

**Family Income.** Family household income was unrelated to body mass index ($b$ = –0.046, $p$ = 0.730).

**Neighborhood Socioeconomic Advantage.** Neighborhood-level socioeconomic status was not statistically significantly associated with BMI ($b$ = –0.304, $p$ = 0.167).

**G×E Interaction: Effect of SES on Genetic and Environmental Etiology of Body Mass Index (Dissertation Aim 2).** The moderating effects of SES on variance in body mass index health are presented in Table 7.4 under the heading *Effect of SES on Residual ACE Components of BMI.*

**Parental Education.** The best-fitting model suggested that residual variance in BMI decreased as a function of increasing parental educational attainment, an effect driven by decreasing additive genetic variance ($b_{1Au}$ = –0.364, $p < 0.001$). These model results are illustrated in Figure 7.37, which shows residual AE variance components of BMI as a function of parental educational attainment, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure clearly demonstrates how residual phenotypic and additive genetic variance in BMI decreases with increasing parental education. It seems that high-SES environments reduce genetic risk for high BMI.

To illustrate these results, we plotted absolute pair differences in BMI for MZ and DZ twins against parental educational attainment (see the left panel of Figure 7.38). In this plot, the gap between the MZ and DZ regression lines (*i.e.*, the tendency for MZ pairs to be more similar than DZ pairs) represents additive genetic variance in BMI. Increases

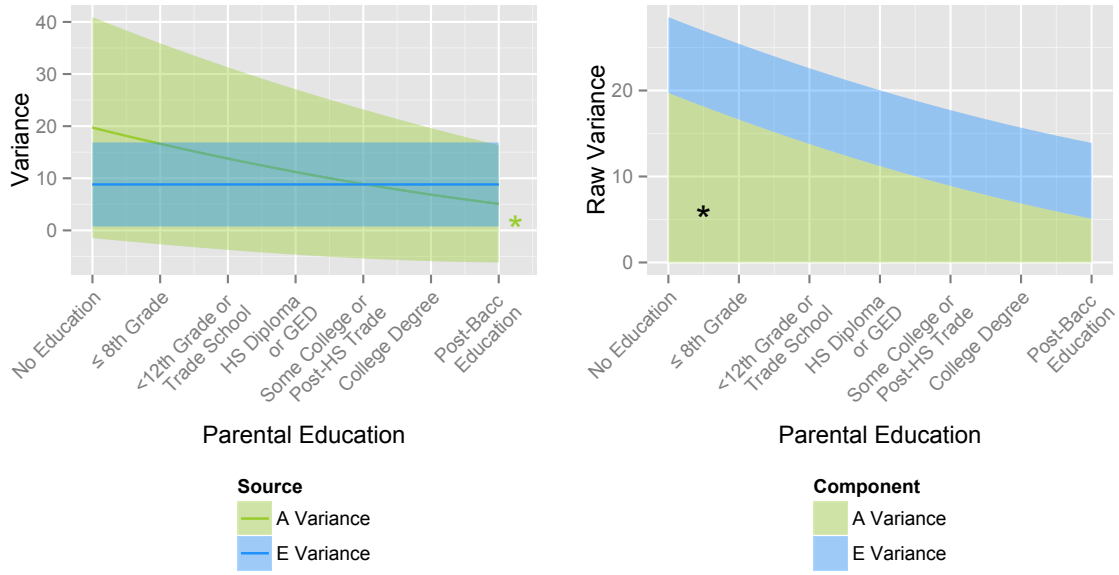


*Figure 7.37*. Gene-by-environment interaction between body mass index and parental educational attainment in the Add Health sample. The left panel shows AE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in BMI decreases as a function of increasing parental education. The asterisks (*) indicate that variance in BMI depends on level of parental education.

in this gap with respect to education would suggest that additive genetic variance in BMI increases with increasing parental educational attainment; decreases would suggest the opposite. The location of the MZ line reflects nonshared environmental variance in BMI. A decreasing slope represents decreasing nonshared environmental variance in BMI. Evident in this plot is that the slope of the MZ regression line is roughly zero while the distance between the lines appears to decrease slightly at lower levels of parental education. These observations are consistent with decreasing additive genetic variance in BMI as a function of increasing parental educational attainment.

This heteroscedasticity is further illustrated in the right panel of Figure 7.38, which shows box plots overlaid with violin plots (which show the probability density of

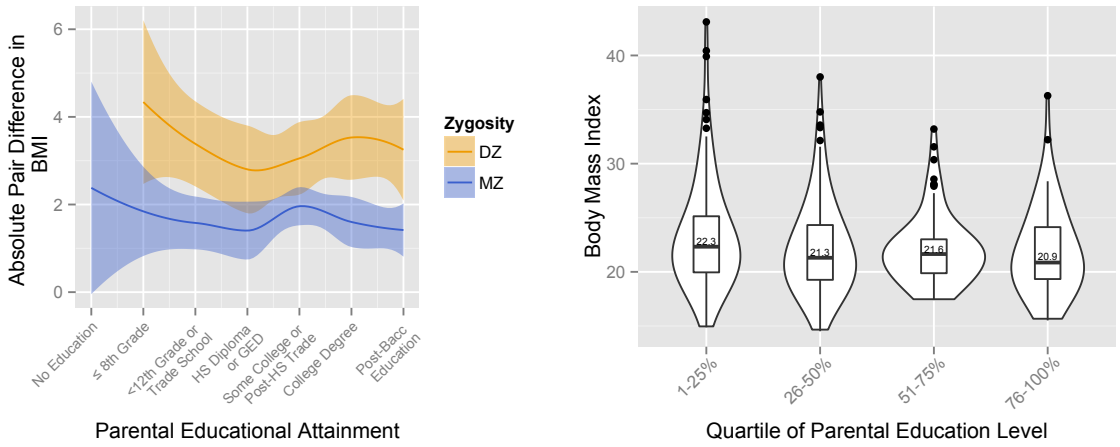


*Figure 7.38.* Illustrative analysis of the effects of parental educational attainment on variance in body mass index in the Add Health sample. The left panel shows absolute pair differences in BMI as a function of parental educational attainment. The distance between the MZ and DZ lines represents the additive genetic variance in BMI. The location of the MZ line represents nonshared environmental variance in BMI. The right panel shows box plots overlaid with violin plots, BMI as a function of quartile of parental educational attainment.

the data) of BMI by quartile of parental educational attainment. This plot shows that the distribution of BMI becomes less platykurtic at higher levels of parental educational attainment, 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 lower tail of the BMI distribution at higher SES levels. The violin plot also shows the main effect of parental educational attainment on BMI; the median BMI decreases across each quartile of parental education.

**Family Income.** The best-fitting model suggested that residual variance in BMI is not related to family household income.

**Neighborhood Socioeconomic Advantage.** The best-fitting model did not suggest that residual variance in BMI is related to neighborhood-level socioeconomic advantage. However, when residual variance in BMI was allowed to vary as a function

of neighborhood-level SES, nonshared environmental variance tended to decrease with increasing neighborhood-level SES ($b_{1Eu}$ = –0.225, $p$ = 0.037[24]). These model results are illustrated in Figure 7.39, and demonstrate how residual nonshared environmental variance in BMI decreases with increasing neighborhood socioeconomic advantage. It seems that high-SES environments protect against environmental risk for physical activity.

These results are further illustrated in Figure 7.40. Nonshared environmental variance (but not between-family variance) in BMI decreases as a function of increasing neighborhood socioeconomic status. The violin plots show that this effect appears to be the result of fewer observations at the lower end of BMI when neighborhood SES is high.

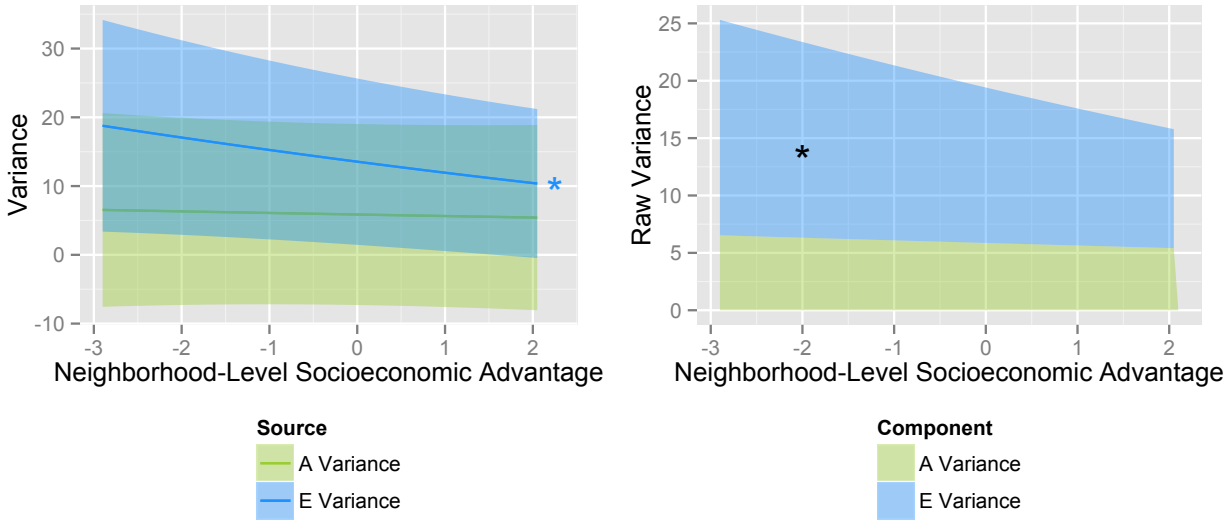


Figure 7.39. Gene-by-environment interaction between body mass index and neighborhood-level socioeconomic advantage in the Add Health sample. The left panel shows AE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in BMI decreases as a function of increasing neighborhood-level SES. The asterisks (*) indicate that variance in BMI depends on level of neighborhood-level SES.

[24] This moderated model itself fell just shy of statistical significance when compared with the unmoderated model ($p$ = .067).

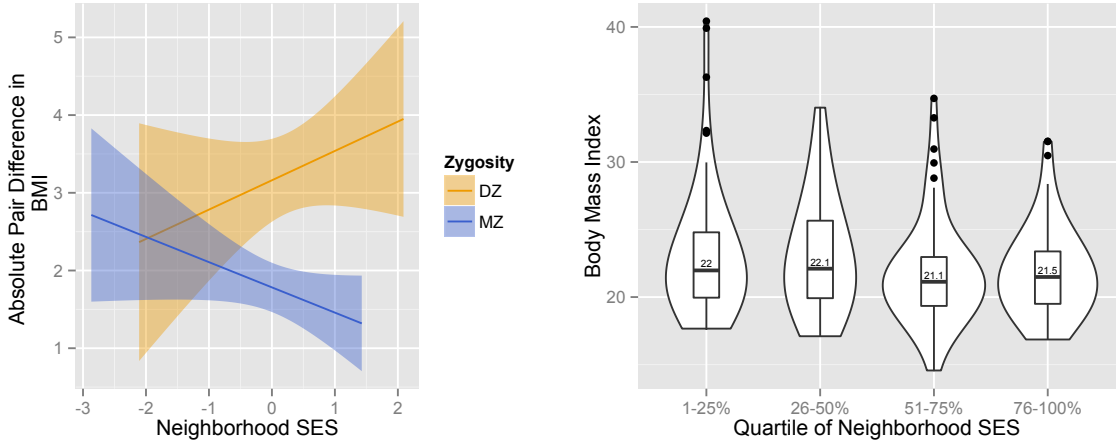


*Figure 7.40.* Illustrative analysis of the effects of neighborhood socioeconomic advantage on variance in body mass index in the Add Health sample. The left panel shows absolute pair differences in BMI as a function of neighborhood SES. The distance between the MZ and DZ lines represents the additive genetic variance in BMI. The location of the MZ line represents nonshared environmental variance in BMI. The right panel shows box plots overlaid with violin plots, BMI as a function of quartile of neighborhood SES.

**Brief Summary.** Higher parental educational attainment was related to lower BMI. Parental education also predicted decreased additive genetic variance in BMI. Conversely, neighborhood-level socioeconomic advantage was associated with decreased nonshared environmental variance in BMI. That is, it appears that compositional SES may protect against genetic risk for high BMI, whereas contextual SES may protect against environmental risk for high BMI.

***Body Mass Index—WSTR.*** Individuals in the WSTR sample provided height and weight measurements, which were used to derive body mass index. We fit the model presented in Figure 7.41 to the data separately for each socioeconomic status indicator, partialling for the linear effects of age and gender on level and variance in self-rated health. We used likelihood ratio tests to determine the best fitting model. We first established a baseline model by conducting omnibus tests for the moderation by SES of

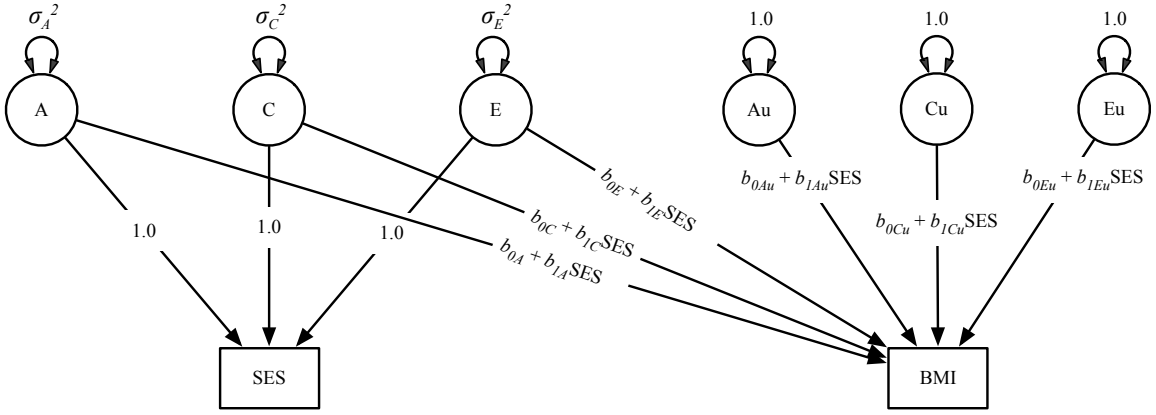


*Figure 7.41*. Path diagram G×SES model fit to body mass index in the WSTR (only one twin shown for clarity). In the fully saturated model, the main effects of SES on BMI and the ACE variances of BMI vary as a function of SES.

both the residual variance in body mass index and the main effects of SES on BMI. We then systematically eliminated parameters from the baseline model to evaluate statistical significance of moderation parameters. Biometric decomposition of both the SES indicators and BMI are presented in Table 4.2, and parameter estimates and model fit statistics for the baseline and best-fitting models presented in Table 7.5. The Area Deprivation Index was log-transformed to correct for positive skew, and the Gini Index was scaled by a factor of 10 to facilitate model convergence. These transformed values were used in all analyses in which the ADI or Gini Index was used as the indicator of contextual socioeconomic status. We did not include A for the ADI since there was no evidence of genetic influences on area deprivation in our univariate model.

**Causal Pathways vs. rGE: Main Effects of SES on Body Mass Index (Dissertation Aim 1).** The main effects of SES on body mass index are presented in Table 7.5 under the heading *Main Effect of Moderator on Body Mass Index*. We discuss the main effects results for each moderator below.

Table 7.5. Parameter estimates and model fit statistics for G×SES models, body mass index in the WSTR.

| | Parameter | Phenotypic Model | Quasi-Causal Model | Moderation of Residual Variance | Moderation of Main Effects | Best-Fitting Model |
|---|---|---|---|---|---|---|
| **Education** | Main Effect of Education on BMI | | | | | |
| | $b_{0A}$ | ***–.237 (.049)*** | –.238 (.336) | –.287 (.318) | .340 (1.570) | –.442 (.324) |
| | $b_{1A}$ | — | — | — | –.123 (.235) | — |
| | $b_{0C}$ | ***–.237 (.049)*** | ***–.917 (.406)*** | ***–.991 (.372)*** | 1.662 (1.632) | **1.999 (.849)** |
| | $b_{1C}$ | — | — | — | –.413 (.241) | ***–.462 (.123)*** |
| | $b_{0E}$ | ***–.237 (.049)*** | .036 (.080) | .062 (.084) | –.462 (.534) | .049 (.084) |
| | $b_{1E}$ | — | — | — | .083 (.086) | — |
| | Effect of Education on Residual ACE Components of BMI | | | | | |
| | $b_{0Au}$ | ***6.595 (.479)*** | ***6.649 (.452)*** | ***7.728 (.699)*** | ***7.948 (.725)*** | ***7.947 (.718)*** |
| | $b_{1Au}$ | — | — | **–.179 (.090)** | **–.206 (.093)** | **–.209 (.091)** |
| | $b_{0Cu}$ | 1.982 (2.373) | 1.582 (2.538) | ***4.741 (1.343)*** | **4.445 (1.479)** | ***4.323 (1.459)*** |
| | $b_{1Cu}$ | | | ***–.627 (.110)*** | ***–.562 (.143)*** | ***–.538 (.133)*** |
| | $b_{0Eu}$ | ***2.243 (.166)*** | ***2.234 (.165)*** | ***2.792 (.263)*** | ***2.782 (.263)*** | ***2.772 (.262)*** |
| | $b_{1Eu}$ | — | — | ***–.100 (.031)*** | ***–.098 (.030)*** | ***–.096 (.030)*** |
| | Model Fit | | | | | |
| | *-2LL* | — | 52053.862 | 51972.442 | 51955.030 | 51956.006 |
| | *Δ-2LL (Δdf)* | — | — | 81.420 (+3) | 17.412 (+3) | .976 (–2) |
| | *p* | — | — | <.001 | <.001† | .614 |
| **Household Income** | Main Effect of Income on BMI | | | | | |
| | $b_{0A}$ | ***–.062 (.024)*** | .054 (.201) | –.057 (.206) | **.149 (.310)** | –.087 (.206) |
| | $b_{1A}$ | — | — | — | –.044 (.046) | — |
| | $b_{0C}$ | ***–.062 (.024)*** | ***–1.135 (.413)*** | **–.911 (.376)** | –.985 (.547) | **–.892 (.373)** |
| | $b_{1C}$ | — | | | .010 (.068) | — |
| | $b_{0E}$ | ***–.062 (.024)*** | .031 (.032) | .021 (.033) | **.251 (.112)** | ***.313 (.096)*** |
| | $b_{1E}$ | — | — | — | **–.050 (.022)** | ***–.061 (.019)*** |
| | Effect of Income on Residual ACE Components of BMI | | | | | |
| | $b_{0Au}$ | ***6.511 (.525)*** | ***6.566 (.303)*** | ***6.407 (.083)*** | ***6.358 (.515)*** | ***5.843 (.423)*** |
| | $b_{1Au}$ | — | — | –.064 (.033) | –.055 (.035) | — |
| | $b_{0Cu}$ | 2.324 (2.742) | .003 (4.724) | **3.666 (1.424)** | ***3.802 (1.401)*** | ***4.039 (1.206)*** |
| | $b_{1Cu}$ | | | ***–.175 (.063)*** | .178 (.061) | ***–.211 (.055)*** |
| | $b_{0Eu}$ | ***2.227 (.171)*** | ***2.296 (.170)*** | ***2.682 (.192)*** | ***2.672 (.192)*** | ***2.693 (.193)*** |
| | $b_{1Eu}$ | — | — | ***–.087 (.018)*** | ***–.087 (.018)*** | ***–.093 (.017)*** |
| | Model Fit | | | | | |
| | *-2LL* | — | 56945.788 | 56891.532 | 56880.366 | 56884.320 |
| | *Δ-2LL (Δdf)* | — | — | 54.256 (+3) | 11.166 (+3) | 3.954 (–3) |
| | *p* | — | — | <.001 | .011† | .266 |
| **Area Deprivation** | Main Effect of Area Deprivation on BMI | | | | | |
| | $b_{0C}$ | ***–2.120 (.532)*** | ***–11.438 (1.494)*** | ***–10.586 (1.347)*** | ***–58.910 (28.941)*** | ***–10.191 (1.302)*** |
| | $b_{1C}$ | — | — | — | 9.986 (5.950) | — |
| | $b_{0E}$ | ***–2.120 (.532)*** | .446 (.637) | –.482 (.600) | 23.555 (16.108) | –.336 (.603) |
| | $b_{1E}$ | — | — | — | –5.054 (3.348) | — |
| | Effect of Area Deprivation on Residual ACE Components of BMI | | | | | |
| | $b_{0Au}$ | ***6.361 (.665)*** | ***6.418 (.641)*** | ***21.802 (2.721)*** | ***21.392 (2.714)*** | ***24.048 (2.821)*** |
| | $b_{1Au}$ | — | — | ***3.289 (.553)*** | ***–3.210 (.556)*** | ***–3.741 (.624)*** |
| | $b_{0Cu}$ | **3.990 (1.789)** | 3.251 (1.869) | **9.833 (4.815)** | 9.165 (4.828) | 3.335 (1.722) |
| | $b_{1Cu}$ | — | | –1.482 (1.039) | –1.328 (1.021) | — |
| | $b_{0Eu}$ | ***2.163 (.215)*** | ***2.196 (.213)*** | ***11.583 (1.140)*** | ***11.521 (1.149)*** | ***11.567 (1.170)*** |
| | $b_{1Eu}$ | — | — | ***–2.053 (.245)*** | ***–2.041 (.247)*** | ***–2.056 (.252)*** |
| | Model Fit | | | | | |
| | *-2LL* | — | 14564.880 | 14456.138 | 14453.418 | 14458.170 |
| | *Δ-2LL (Δdf)* | — | — | 108.742 (+3) | 2.720 (+2) | 2.032 (–1) |
| | *p* | — | — | <.001† | .257 | .154 |
| **Income Inequality** | Main Effect of Income Inequality on BMI | | | | | |
| | $b_{0A}$ | ***–.794 (.222)*** | –11.628 (11.537) | –11.340 (11.332) | –19.402 (52.797) | –11.635 (11.371) |
| | $b_{1A}$ | — | — | — | 1.893 (11.699) | — |
| | $b_{0C}$ | ***–.794 (.222)*** | –1.384 (2.772) | –1.549 (2.690) | 4.869 (20.357) | –1.483 (2.068) |
| | $b_{1C}$ | — | — | — | –1.481 (4.653) | — |
| | $b_{0E}$ | ***–.794 (.222)*** | –.090 (.280) | –.129 (.274) | –.079 (4.886) | –.104 (.286) |
| | $b_{1E}$ | — | — | — | –.001 (1.122) | — |
| | Effect of Income Inequality on Residual ACE Components of BMI | | | | | |
| | $b_{0Au}$ | ***6.296 (.596)*** | ***6.192 (.615)*** | ***8.781 (1.642)*** | ***8.532 (2.107)*** | ***5.907 (.565)*** |
| | $b_{1Au}$ | — | — | –.620 (.338) | –.563 (.445) | — |
| | $b_{0Cu}$ | 2.797 (2.174) | 2.799 (2.085) | 3.690 (6.334) | 3.934 (6.426) | ***8.752 (2.585)*** |
| | $b_{1Cu}$ | — | — | –.204 (1.270) | –.258 (1.303) | **–1.325 (.526)** |
| | $b_{0Eu}$ | ***2.431 (.188)*** | ***2.426 (.187)*** | ***4.698 (.352)*** | ***4.704 (.744)*** | ***5.099 (.721)*** |
| | $b_{1Eu}$ | — | — | ***–.513 (.159)*** | ***–.514 (.160)*** | ***–.599 (.154)*** |
| | Model Fit | | | | | |
| | *-2LL* | — | 29004.824 | 28977.758 | 28977.444 | 28980.440 |
| | *Δ-2LL (Δdf)* | — | — | 27.066 (+3) | .314 (+3) | 2.682 (–1) |
| | *p* | — | — | <.001† | .957 | .101 |

*Note:* Standard errors presented within parentheses. Estimates $p < .05$ **bolded**. Estimates $p < .01$ *italicized*. Baseline model denoted by †.

**Education.** Education demonstrated a phenotypic association with BMI ($b$ = –0.237, $p$ < 0.001). Controlling for age and gender, individuals with a graduate or professional degree had BMIs that were an average of 0.95 kg/m$^2$ (an equivalent of 0.16 standard deviations) lower than individuals with just a high school degree. The best-fitting model showed nonsignificant genetic ($b_{0A}$ = –0.441, $p$ = 0.173) and quasi-causal pathways ($b_{0E}$ = 0.049, $p$ = 0.559), and suggested that this association is instead mediated by a common shared environmental pathway that grows more negative with increasing educational attainment ($b_{0C}$ = 1.996, $p$ = 0.019; $b_{1C}$ = –0.461, $p$ < 0.001). As illustrated in the left panel of Figure 7.42, which shows the magnitude of the genetic, shared environmental, and nonshared environmental regressions of BMI on educational attainment, this effect is indistinguishable from zero except at higher levels of educational attainment (*i.e.*, Bachelor's degree or higher). In sum, it appears that educational attainment influences BMI, but only at high levels of education, and operates through shared environmental factors that are common to both educational attainment and BMI. That is, family environmental factors are the most important contributors to the BMI–SES association; systematic differences in educational attainment do not appear to influence BMI. These regression lines are re-represented as genetic, shared environmental, and nonshared environmental correlations in the middle panel of Figure 7.42. We also present the proportions of the total phenotypic correlation accounted for by the genetic, shared environmental, and nonshared environmental correlations, shown in the right panel of Figure 7.42. Evident in this figure is that the between-family effects of educational attainment on BMI tend to be larger than the within-family effects, and

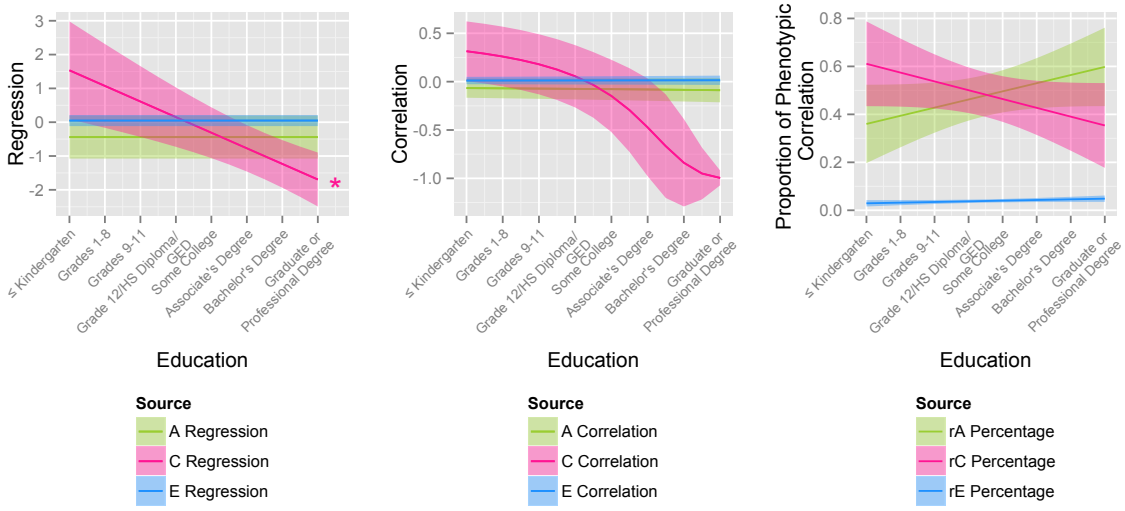


*Figure 7.42*. Main effects of educational attainment on body mass index in the WSTR. The left panel shows the regression of BMI on educational attainment. The same relation is presented in the middle panel as genetic (green), shared environmental (pink), and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic, shared environmental, and nonshared environmental correlations. The asterisks (*) indicate that the magnitude of the regression of BMI on educational attainment depends on level of education (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). The shaded regions represent 95% confidence intervals around the regression lines.

account for nearly all of the total phenotypic correlation between education and BMI.

To demonstrate what these effects look like within and between twins, we conducted an illustrative analysis of the main effect of educational attainment on BMI, which we present in Figure 7.43. We identified twin pairs concordant for lower education (up to Associate's degree; orange), pairs concordant for higher education (Bachelor's degree or higher; navy), and pairs discordant for educational status, and compared the mean BMI of each of these groups. Overall, there is a main effect of educational attainment on BMI such that higher education is associated with lower adiposity (this is evident comparing the outer bars in this figure). Examining the relation at the within-pair

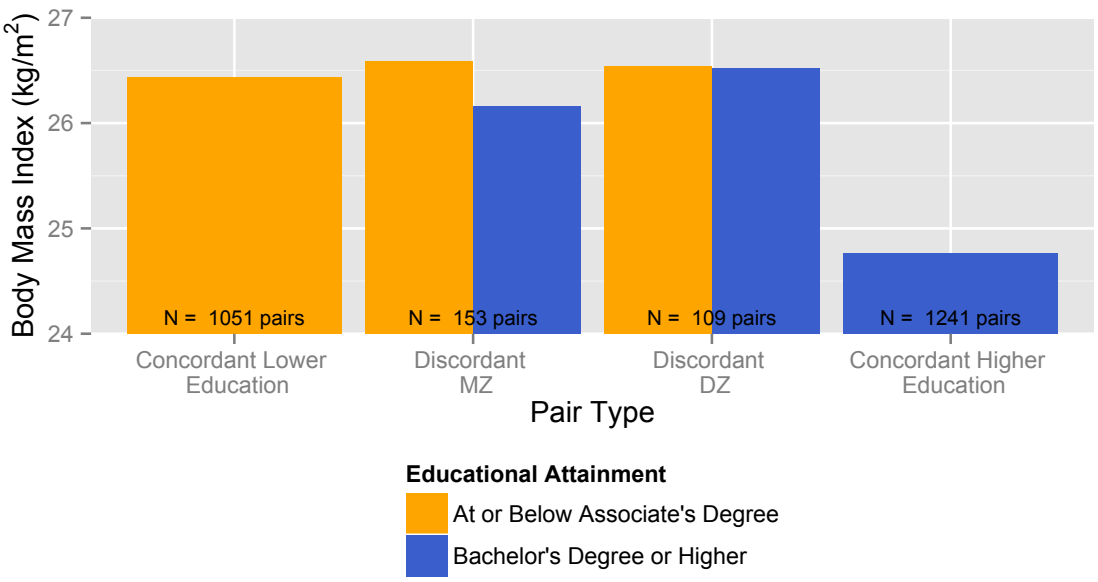


*Figure 7.43*. Illustrative analysis of the main effects of educational attainment on body mass index in the WSTR. The twin with less education (orange) is compared with the twin with more education (navy) in MZ and DZ twin pairs.

level, however, shows that it does not exist within pairs of twins (neither MZ nor DZ), consistent with shared environmental selection.

**Income.** The phenotypic regression of self-rated health on household income showed a significant positive relationship ($b$ = –0.794, $p$ < 0.001). Controlling for age and gender, individuals at the third quartile of earned income rated had BMIs that were on average 3.97 kg/m$^2$ (equivalent to 0.66 standard deviations) lower than their counterparts at the first quartile of earned income. The best-fitting model suggested that this association is partially mediated by a common shared environmental background ($b_{0C}$ = 4.039, $p$ = 0.001). The genetic regression was not statistically significant ($b_{0A}$ = –0.089, $p$ = 0.665), and the nonshared environmental regression of BMI on household income depended on income level ($b_{0E}$ = 0.313, $p$ = 0.001; $b_{1E}$ = –0.061, $p$ = 0.001). As is evident in the left panel of Figure 7.44, the between-family contributions to this

association tended to be larger than the within-family contributions, although there is a small but statistically significant quasi-causal effect that was positive at low levels of education (high school diploma or less) and negative at very high levels (Bachelor's degree or higher). Additionally, between-family confounds collectively account for more of the total phenotypic correlation than within-family factors, evident in the right panel of Figure 7.44.

Illustrative analyses further highlight this shared environmental correlation. In Figure 7.45, we show pair differences in BMI as a function of pair differences in household income within randomly paired individuals (unrelated relation; blue line) and

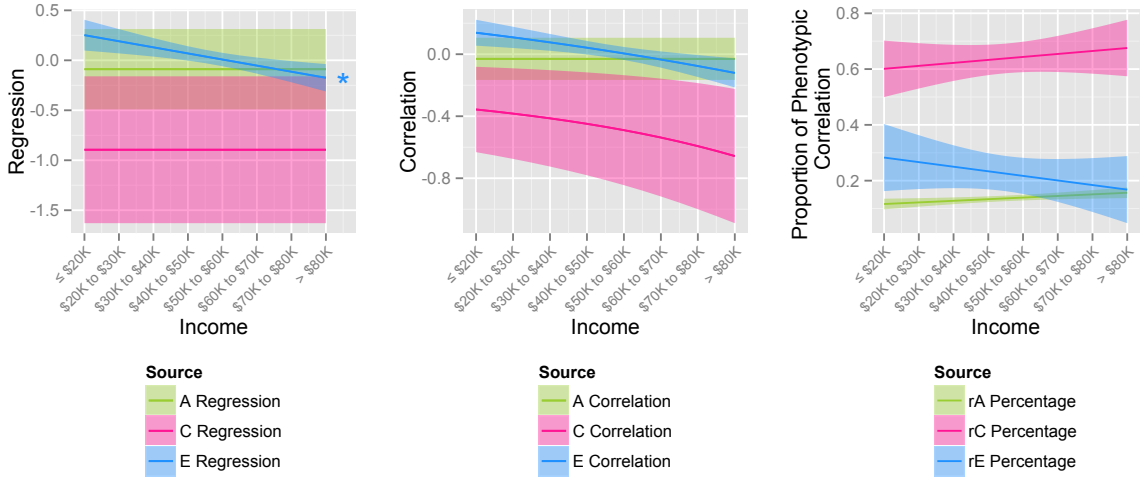


*Figure 7.44*. Main effects of household income on body mass index in the WSTR. The left panel shows the regression of BMI on household income. The same relation is presented in the middle panel as genetic (green), shared environmental (pink), and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic, shared environmental, and nonshared environmental correlations. The asterisks (*) indicate that the magnitude of the regression of BMI on household income depends on level of education (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). The shaded regions represent 95% confidence intervals around the regression lines.

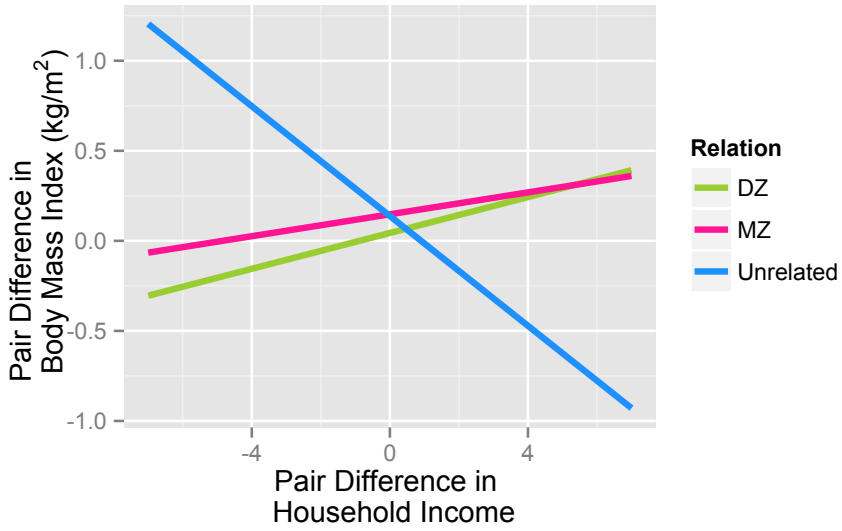


*Figure 7.45*. Illustrative analysis of the main effects of household income on body mass index in the WSTR. This plot shows pair differences in BMI as a function of pair differences in household income in the population (referred to as the ‘‘Unrelated Relation’’) and within pairs of MZ and DZ twins.

within MZ (red line) and DZ (green line) pairs. If the protective effect of household income on self-rated health was causal, the slopes of these lines would closely approximate one another. Comparison of these lines suggests, however, that differences in household income do not predict differences in BMI within families, only between them. The slopes of the MZ and DZ lines are quite close, consistent with a common shared environmental pathway between household income and BMI.

**Area Deprivation.** At the phenotypic level, the ADI was statistically significantly associated with better self-rated health ($b$ = –2.120, $p$ < 0.001). Individuals at the third quartile of the ADI had BMIs that were on average 2.01 kg/m$^2$ (equivalent to 0.34 standard deviations) lower than individuals at the first quartile of the ADI. The best-fitting model suggested that this association is mediated by a common shared environmental background ($b_{0C}$ = –10.191, $p$ < 0.001; as a note, we did not observe

genetic contributions to the ADI, and therefore no common genetic pathway was estimated). The nonshared environmental regression of BMI on the ADI was not statistically significant and was significantly reduced in magnitude relative to the phenotypic effect ($b_{0E}$ = –0.336, $p$ = 0.578). As is evident in the left panel of Figure 7.46, the between-family contribution to this association is larger than the within-family contribution. Also evident is that shared environmental confounds account for nearly all of the total phenotypic correlation, as illustrated in the right panel of Figure 7.46.

Illustrative analyses further highlight this shared environmental correlation. In Figure 7.47, we show pair differences in body mass index as a function of pair differences in the log-transformed ADI within randomly paired individuals (unrelated relation; blue line) and within MZ (red line) and DZ (green line) pairs. If the protective effect of lower area deprivation on BMI were causal, the slopes of these lines would

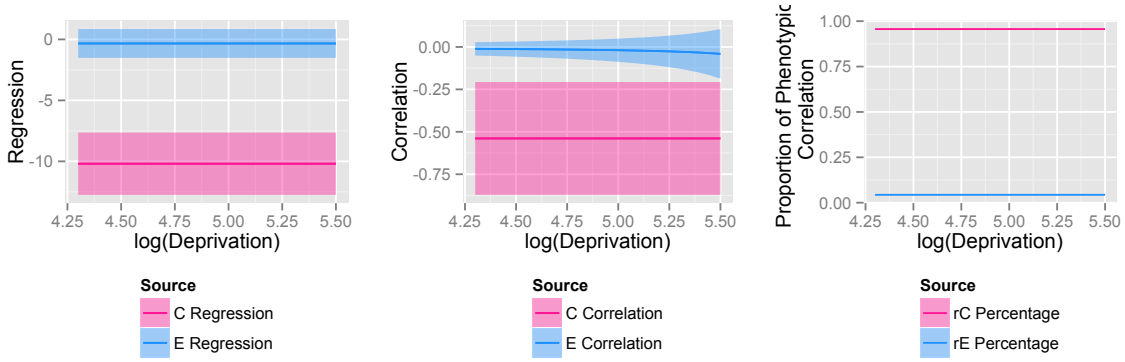


*Figure 7.46*. Main effects of area deprivation on body mass index in the WSTR. The left panel shows the regression of BMI on the (log transformed) ADI. The same relation is presented in the middle panel as shared environmental (pink) and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by shared environmental and nonshared environmental correlations. The asterisks (*) indicate that the magnitude of the regression of BMI on the ADI depends on level of area deprivation (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). The shaded regions represent 95% confidence intervals around the regression lines.

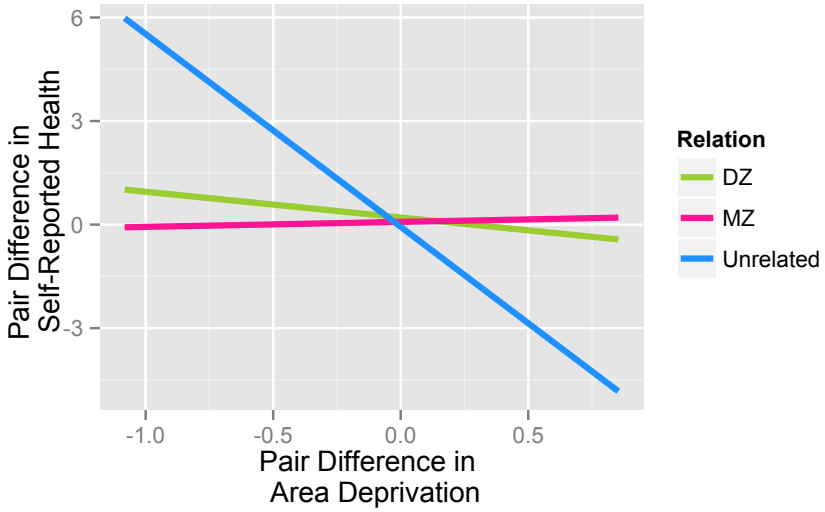


*Figure 7.47*. Illustrative analysis of the main effects of area deprivation on body mass index in the WSTR. This plot shows pair differences in BMI as a function of pair differences in log-transformed ADI in the population (referred to as the ‘‘Unrelated Relation’’) and within pairs of MZ and DZ twins.

closely approximate one another. Comparison of these lines suggests, however, that differences in the ADI do not predict differences in BMI within families, only between them. The slopes of the MZ and DZ lines are quite close, consistent with a common shared environmental pathway between area deprivation and BMI.

**Income Inequality.** At the phenotypic level, income inequality was statistically significantly associated with body mass index ($b$ = –0.794, $p < 0.001$), but the effect size was negligible: Individuals at the third quartile of the Gini Index had BMIs that were approximately 0.90 kg/m$^2$ (equivalent to 0.02 standard deviations) lower on average than individuals at the first quartile of the Gini Index. The best fitting model suggested that BMI was not quasi-causally associated with income inequality ($b_{0E}$ = –0.104, $p$ = 0.717); the genetic ($b_{0A}$ = –11.635, $p$ = 0.306) and shared environmental ($b_{0C}$ = –1.483, $p$ = 0.570) pathways were also not statistically significant, which likely represents lack of

power to differentiate between these sources of covariation. When estimating the total between-family effect (achieved by constraining $b_{0A}$ and $b_{0C}$ to be equal), however, there was significant positive between-family confounding between income inequality and BMI ($b_{0A} = b_{0C} = -3.967$, $p < 0.001$; $b_{0E} = -0.183$, $p = 0.506$; results from this model not presented in Table 7.5). These results suggest that the relation between income inequality and BMI is best explained by between-family factors that are common to both phenotypes rather than by systematic differences in exposure to socioeconomic factors. As is evident in the left panel of Figure 7.48, the between-family contributions to this association are larger than the within-family contribution. Also evident is that the between-family contributions account for nearly all of the total phenotypic correlation, as illustrated in the right panel of Figure 7.48.

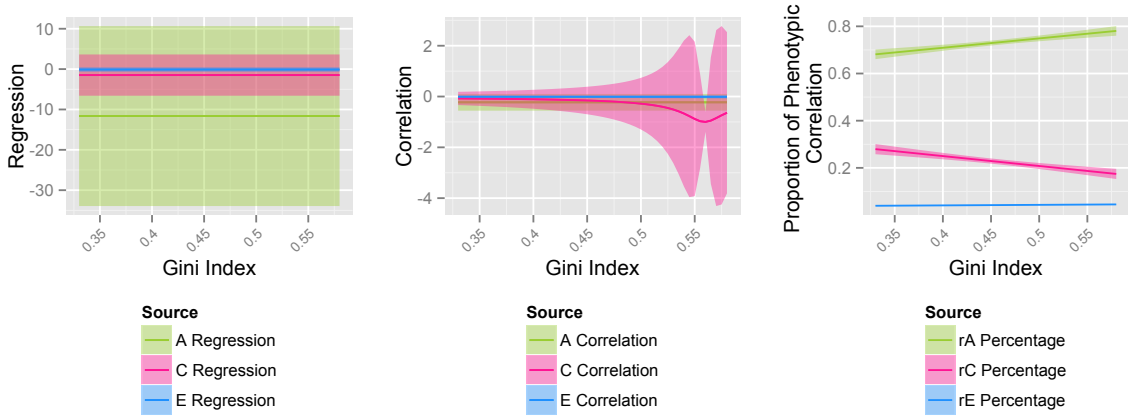


*Figure 7.48*. Main effects of income inequality on body mass index in the WSTR. The left panel shows the regression of BMI on income inequality (scaled by a factor of 10). The same relation is presented in the middle panel as genetic (green), shared environmental (pink), and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic, shared environmental, and nonshared environmental correlations. The asterisks (*) indicate that the magnitude of the regression of BMI on income inequality depends on level of income inequality (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). The shaded regions represent 95% confidence intervals around the regression lines.

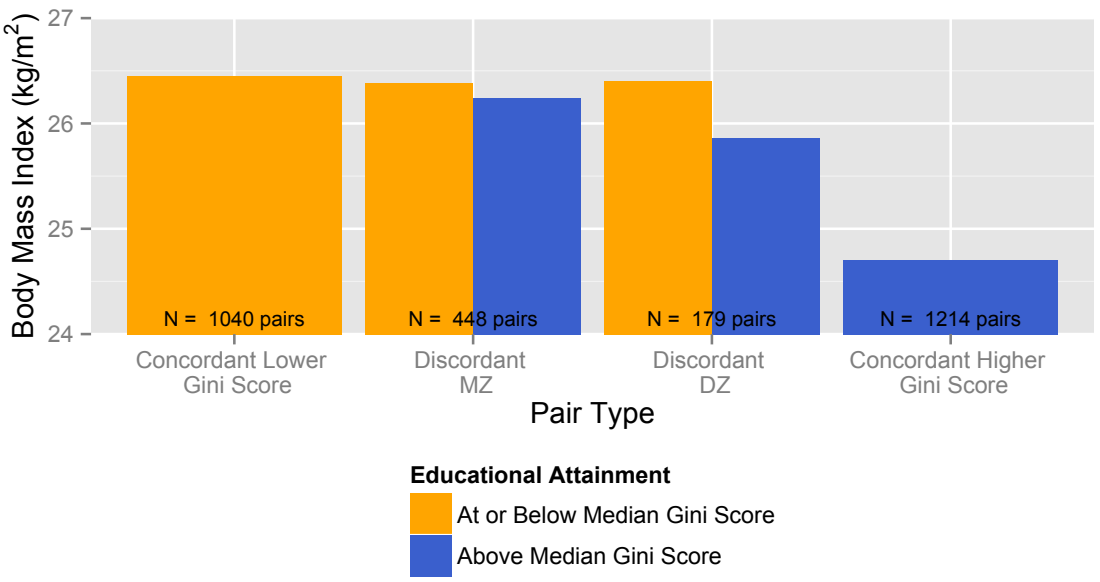


*Figure 7.49*. Illustrative analysis of the main effects of income inequality on body mass index in the WSTR. The twin living in a county with less income inequality (dark gray) is compared with the twin living in a county with greater income inequality (light gray) in MZ and DZ twin pairs.

Illustrative analyses mirror those observed for the other socioeconomic indicators. In Figure 7.49, we show pair differences in body mass index as a function of pair differences in the Gini Index within randomly paired individuals (unrelated relation; blue line) and within MZ (red line) and DZ (green line) pairs. If the protective effect of higher income inequality on BMI were causal, the slopes of these lines would closely approximate one another. Comparison of these lines suggests, however, that differences in income inequality do not predict differences in BMI within families, only between them. The slopes of the MZ and DZ lines are quite close, consistent with a common shared environmental pathway between income inequality and BMI.

**G×E Interaction: Effect of SES on Genetic and Environmental Etiology of Body Mass Index (Dissertation Aim 2).** The moderating effects of SES on variance in BMI are presented in Table 7.5 under the heading *Effect of SES on Residual ACE*

*Components of Body Mass Index.* We discuss the interactive effect of each SES indicator on variance in BMI below.

**Education.** The best fitting model suggested that residual phenotypic variance in BMI decreased with increasing educational attainment. This effect was driven by decreases in all ACE variance components ($b_{1Au}$ = –0.209, $p$ = 0.022; $b_{1Cu}$ = –0.537, $p$ < 0.001; $b_{1Eu}$ = –0.096, $p$ = 0.002). These model results are illustrated in Figure 7.50, which shows residual ACE variance components of BMI as a function of educational attainment, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure clearly demonstrates how residual phenotypic (and ACE) variance(s) in BMI decreases with increasing educational attainment. The genetic and nonshared environmental correlations were static with respect to educational attainment, suggesting that low education potentiates genetic and environmental risk for high BMI. Similarly, the shared environmental correlation was lowest where shared environmental variance in BMI was highest, also consistent with low educational attainment potentiating family environmental risk for high BMI.

To illustrate these results, we plotted absolute pair differences in BMI for MZ and DZ twins against pair-average educational attainment (see the left panel of Figure 7.51). In this plot, the gap between the MZ and DZ regression lines (*i.e.*, the tendency for MZ pairs to be more similar than DZ pairs) represents additive genetic variance in BMI. Increases in this gap with respect to education would suggest that additive genetic variance in BMI increases with increasing educational attainment; decreases would suggest the opposite. The location of MZ line in reflects nonshared environmental variance in BMI. A decreasing slope represents decreasing nonshared environmental

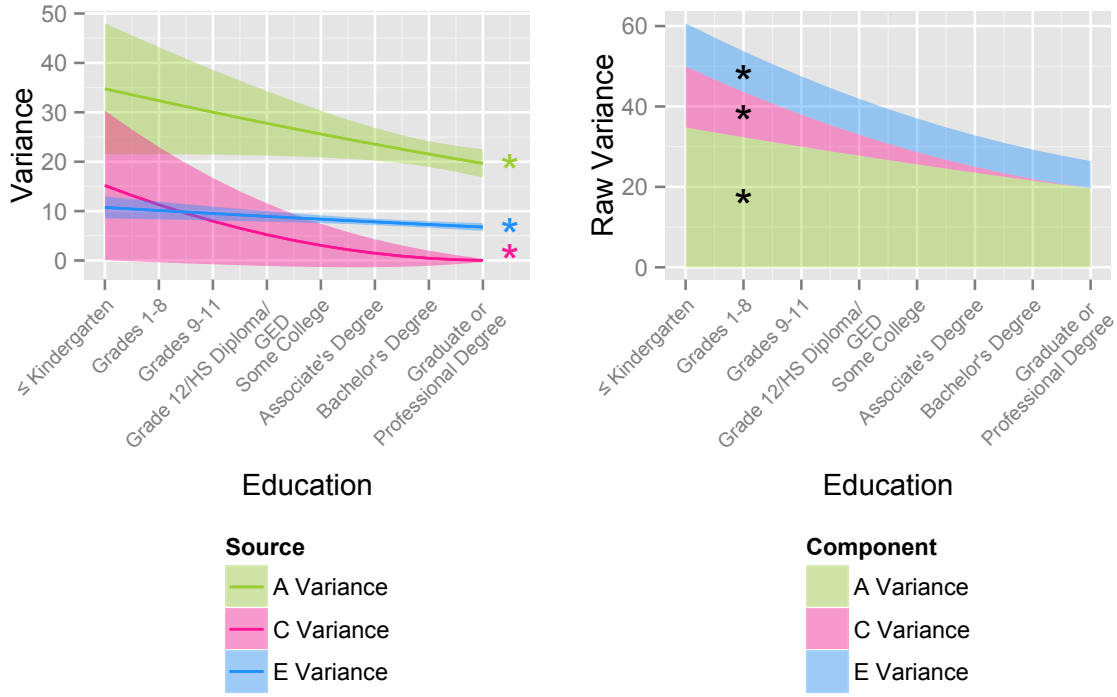


*Figure 7.50*. Gene-by-environment interaction between body mass index and educational attainment in the WSTR. The left panel shows ACE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in BMI decreases as a function of increasing educational attainment. The asterisks (*) indicate that variance in BMI depends on level of education.

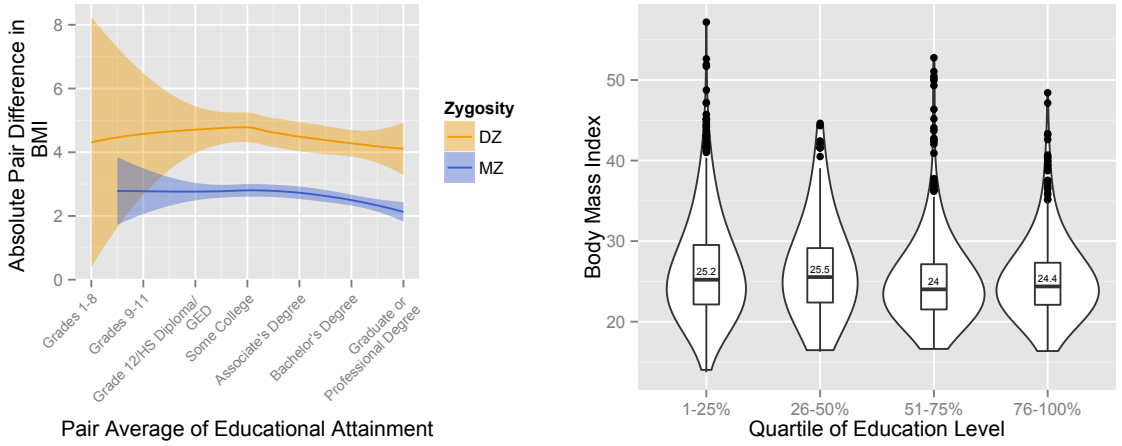


*Figure 7.51*. Illustrative analysis of the effects of educational attainment on variance in body mass index in the WSTR. The left panel shows absolute pair differences in BMI as a function of pair-average educational attainment. The distance between the MZ and DZ lines represents the additive genetic variance in BMI. The location of the MZ represents nonshared environmental variance in BMI. The right panel shows box plots overlaid with violin plots, BMI as a function of quartile of educational attainment.

variance in BMI. Evident in this plot is that the slopes of the MZ and DZ regression lines are negative while the distance between the lines appears to decrease slightly at lower with increasing educational attainment. These observations are consistent with decreasing within- and between-family variance in BMI as a function of increasing educational attainment.

This heteroscedasticity is further illustrated in the right panel of Figure 7.51, which shows box plots overlaid with violin plots (which show the probability density of the data) of BMI by quartile of educational attainment. This plot shows that the distribution of BMI becomes less platykurtic at higher levels of educational attainment, 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. Also evident is the median BMI decreases with each quartile of educational attainment, illustrating the main effect of education on BMI.

**Income.** Like with education, the best fitting model suggested that residual phenotypic variance in self-rated health decreased with increasing household income. This effect was driven by decreasing shared ($b_{1Cu}$ = –0.211, $p < 0.001$) and nonshared environmental variance ($b_{1Eu}$ = –0.093, $p < 0.001$). There was no moderation of the genetic variance as a function of income. These model results are illustrated in Figure 7.52, which shows residual ACE variance components of body mass index as a function of household income, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure clearly demonstrates how residual phenotypic and shared and nonshared environmental variance in BMI decreases

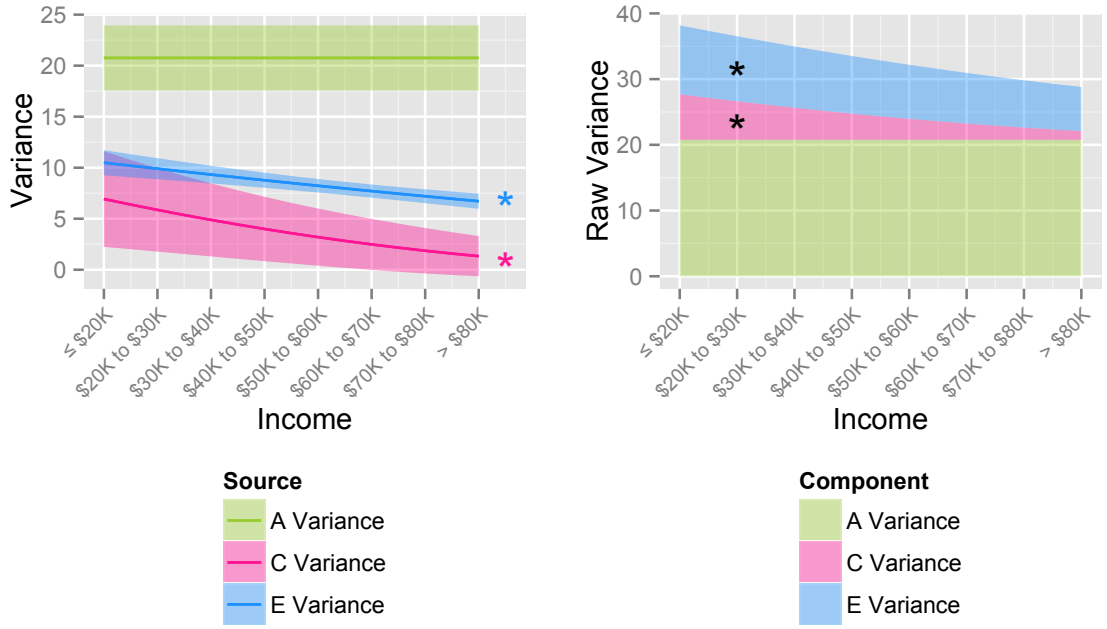


*Figure 7.52*. Gene-by-environment interaction between body mass index and household income in the WSTR. The left panel shows ACE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in BMI decreases as a function of increasing household income. The asterisks (*) indicate that variance in BMI depends on income level.

with increasing household income. Interpreted in the context of the dynamics of ACE correlations, low household income appears to potentiate genetic and environmental risk for high BMI.

These results are further illustrated in Figure 7.53. As we observed with educational attainment, total variance in BMI decreases as a function of increasing household income. Consistent with decreasing nonshared environmental variance, the slope of the MZ regression line (left panel of Figure 7.53) is negative. The violin plots (right panel of Figure 7.53) show that this constrained variance effect appears to be the result of fewer observations at the lower end of BMI when income level is high.

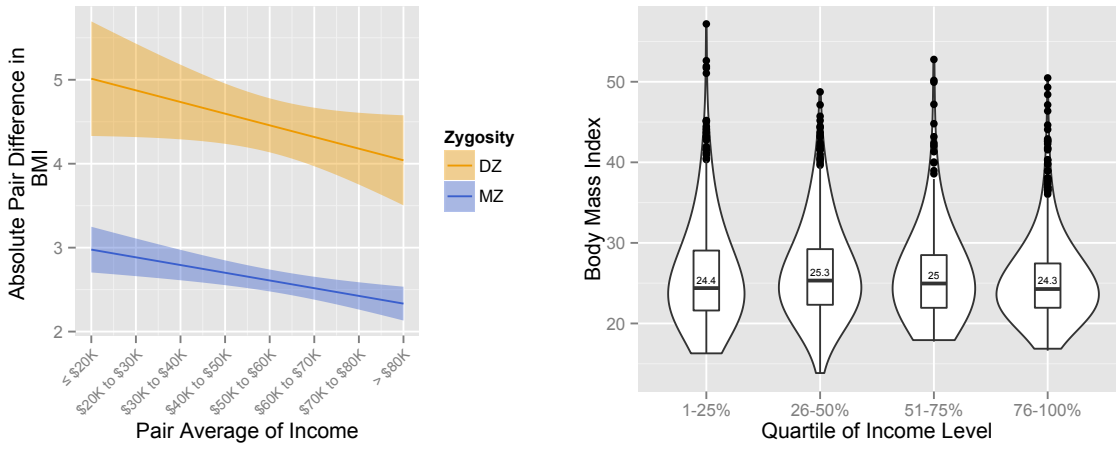


*Figure 7.53.* Illustrative analysis of the effects of household income on variance in body mass index in the WSTR. The left panel shows absolute pair differences in BMI as a function of pair-average household income. The distance between the MZ and DZ lines represents the additive genetic variance in BMI. The location of the MZ represents nonshared environmental variance in BMI. The right panel shows box plots overlaid with violin plots, BMI as a function of quartile of household income.

**Area Deprivation.** As we observed with compositional measures of SES, the best fitting model suggested that residual phenotypic variance in BMI decreased with decreasing area deprivation. This effect was driven by decreasing genetic ($b_{1Au}$ = –3.741, $p < 0.001$) and nonshared environmental variance ($b_{1Eu}$ = –2.056, $p < 0.001$). Area deprivation was unrelated to additive genetic variance in BMI. These model results are illustrated in Figure 7.54, which shows residual ACE variance components of BMI as a function of household income, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure illustrates the decrease in residual phenotypic and genetic and nonshared environmental variance in BMI as a function of decreasing area deprivation. The nonshared environmental correlation was static with respect to neighborhood-level SES (and *r*A was equal to zero), suggesting that high area deprivation potentiates genetic and environmental risk for high BMI.

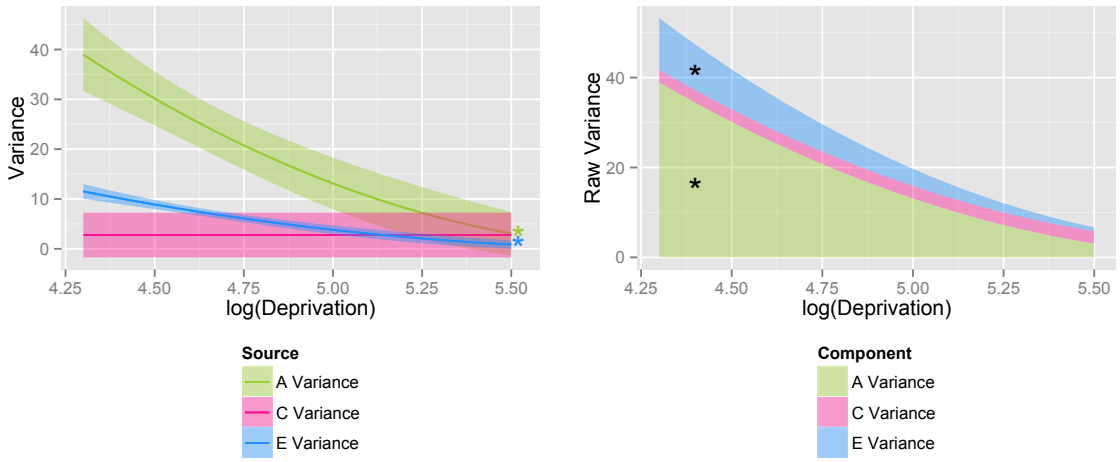


*Figure 7.54*. Gene-by-environment interaction between body mass index and area deprivation in the WSTR. The left panel shows ACE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in BMI decreases as a function of decreasing area deprivation. The asterisks (*) indicate that variance in BMI depends on level of area deprivation.

These results are further illustrated in Figure 7.55. As we observed with educational attainment and household income, total variance in BMI decreases as a function of increasing neighborhood SES. Consistent with decreasing nonshared environmental variance, the slope of the MZ regression line (left panel of Figure 7.55) is negative. The violin plots (right panel of Figure 7.55) show that this constrained variance effect appears to be the result of fewer observations at the lower end of BMI when neighborhood SES is high.

**Income Inequality.** As was observed with area deprivation, the best fitting model suggested that residual phenotypic variance in BMI decreased with increasing county-level income inequality. This effect was driven by decreasing shared environmental ($b_{1Cu}$ = –1.325, $p$ = 0.012) and nonshared environmental variance ($b_{1Eu}$ = –0.599, $p$ < 0.001). There was no moderation of the genetic variance as a function of

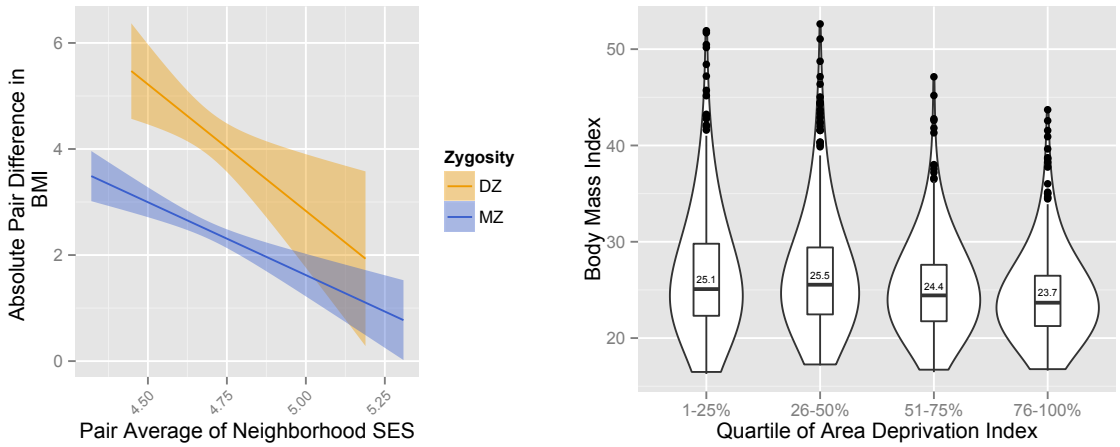


*Figure 7.55.* Illustrative analysis of the effects of neighborhood socioeconomic advantage on variance in body mass index in the WSTR. The left panel shows absolute pair differences in BMI as a function of pair-average neighborhood SES. The distance between the MZ and DZ lines represents the additive genetic variance in BMI. The location of the MZ represents nonshared environmental variance in BMI. The right panel shows box plots overlaid with violin plots, BMI as a function of quartile of neighborhood SES.

income inequality. These model results are illustrated in Figure 7.56, which shows residual ACE variance components of BMI as a function of income inequality, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure illustrates the decrease in residual phenotypic and shared and nonshared environmental variance in BMI as a function of increasing income inequality. The environmental correlation between BMI and income inequality was static with respect to income inequality, suggesting that lower income inequality potentiates genetic and environmental risk for high BMI.

These results are further illustrated in Figure 7.57. As we observed with the other socioeconomic indicators, total variance in BMI decreases as a function of increasing income inequality. Consistent with decreasing nonshared environmental variance, the slope of the MZ regression line (left panel of Figure 7.57) is negative. The violin plots

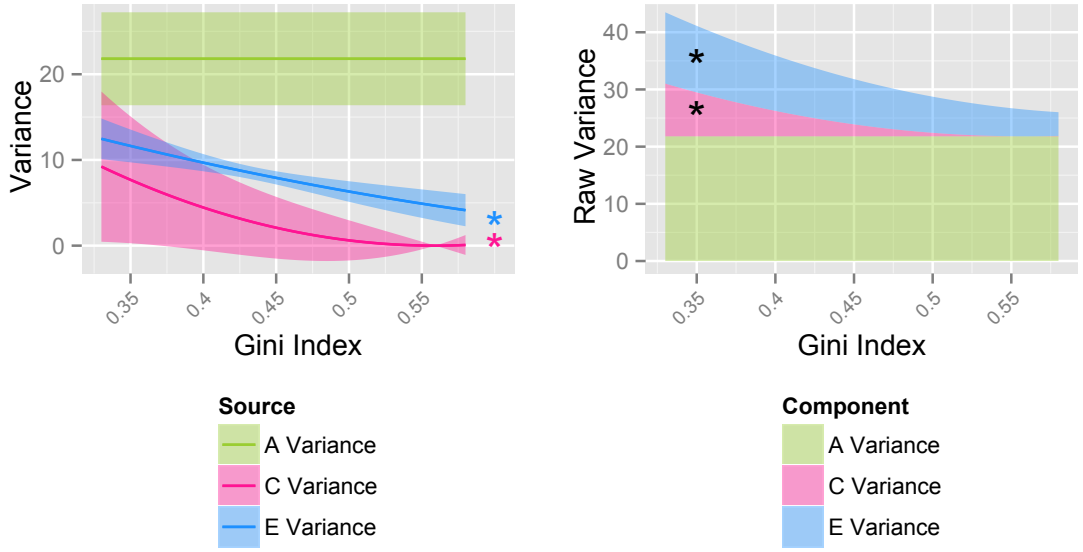


*Figure 7.56*. Gene-by-environment interaction between body mass index and income inequality in the WSTR. The left panel shows ACE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in BMI decreases as a function of increasing income inequality. The asterisks (*) indicate that variance in BMI depends on level of income inequality.

(right panel of Figure 7.57) show that this constrained variance effect appears to be the result of fewer observations at the lower end of BMI when income inequality is high.

**Brief Summary.** Both compositional and contextual measures of socioeconomic status influenced mean levels of body mass index, and operated primarily through shared environmental pathways. Shared environmental regressions tended to have the greatest magnitude, and between-family factors accounted for a much larger proportion of the total phenotypic correlation than did within-family factors. Residual variance in BMI was also influenced by compositional and contextual measures of SES. Residual phenotypic variance decreased with increasing socioeconomic status, and this decrease was driven by decreases in genetic, shared, and nonshared environmental variances. Finally, these decreases in these residual environmental variances appeared to be

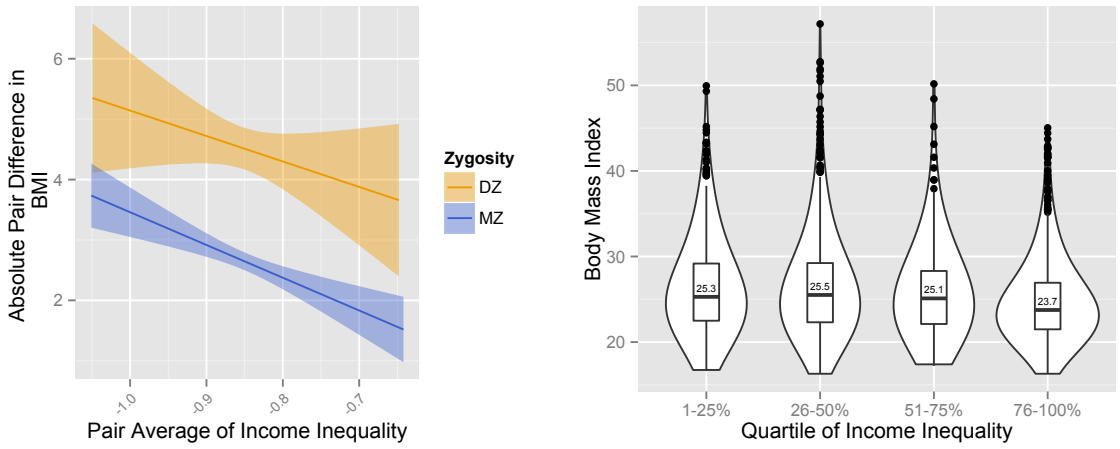


*Figure 7.57.* Illustrative analysis of the effects of income inequality on variance in body mass index in the WSTR. The left panel shows absolute pair differences in BMI as a function of pair-average county-level income inequality. The distance between the MZ and DZ lines represents the additive genetic variance in BMI. The location of the MZ represents nonshared environmental variance in BMI. The right panel shows box plots overlaid with violin plots, BMI as a function of quartile of income inequality.

potentiated by socioeconomic status, and not by social selection factors.

**Immune Functioning**

***Immune Functioning—Add Health.*** We fit the model presented in Figure 7.58 to the data separately for each socioeconomic status indicator, partialling for the linear effects of age and gender on level and variance in immune functioning. We used likelihood ratio tests to determine the best fitting model. Biometric decomposition of both the SES indicators and immune functioning are presented in Table 4.1, and parameter estimates for each of these models are presented in Table 7.6. Parental household income was log-transformed to correct for positive skew. The immune functioning factor showed no evidence of influence from shared environmental factors; therefore, we did not include C in any of the models fit to the data. In addition, the sample of individuals with parental household income data did not show evidence of

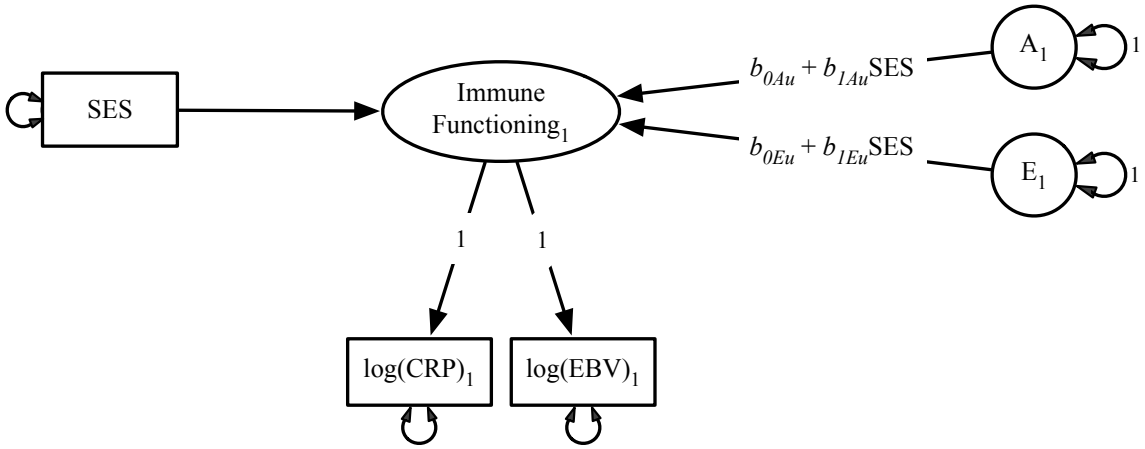


Figure 7.58. Path diagram G×SES model fit to immune functioning in the Add Health sample (only one twin shown for clarity). The residual variances for the immunological biomarkers were permitted to correlate across twins, and were estimated freely according to zygosity.

additive genetic influence on immune functioning; for this predictor, we did not include A or C (*i.e.*, estimated residual nonshared environmental variance only).

**Main Effects of SES on Immune Functioning (Dissertation Aim 1).** The main effects of SES on immune functioning are presented in Table 7.6 under the heading *Main Effect of Moderator on Immune Functioning*. We discuss the main effects results for each moderator below.

**Parental Education.** Parental education was significantly associated with better immune functioning ($b$ = –0.068, $p$ = 0.019). Adolescents with a parent who holds a college degree had scores on the latent immune functioning factor that were 0.21 standard deviations lower than their counterparts whose parent holds a high school degree.

**Family Income.** Immune functioning was not related to family household income ($b$ = 0.021, $p$ = 0.375).

Table 7.6. Parameter estimates and model fit statistics for G×SES models, immune functioning in the Adolescent Health sample.

| | Parameter | Phenotypic Model (No Moderation) | Moderation of Residual Variance | Best-Fitting Model |
|---|---|---|---|---|
| **Parental Education** | Main Effect of Parental Education on Immune Functioning | | | |
| | $b_1$ | **–.068 (.029)** | ***–.083 (.031)*** | **–.074 (.029)** |
| | Effect of Education on Residual ACE Components of Immune Functioning | | | |
| | $b_{0Au}$ | **1.731 (.828)** | 1.163 (1.781) | ***.197 (.946)*** |
| | $b_{1Au}$ | — | .121 (.127) | — |
| | $b_{0Eu}$ | ***4.235 (.793)*** | ***2.902 (.808)*** | ***3.375 (.581)*** |
| | $b_{1Eu}$ | — | ***.208 (.059)*** | ***.215 (.037)*** |
| | Model Fit | | | |
| | *-2LL* | 8108.702 | 8069.228 | 8071.034 |
| | *Δ-2LL (Δdf)* | — | 39.474 (+2) | 1.806 (–1) |
| | *p* | — | <.001† | .179 |
| **Family Household Income** | Main Effect of Family Income on Immune Functioning | | | |
| | $b_1$ | .015 (.023) | –.023 (.033) | –.023 (.033) |
| | Effect of Family Income on Residual ACE Components of Immune Functioning | | | |
| | $b_{0Eu}$ | ***5.417 (588)*** | ***4.999 (.596)*** | ***4.999 (.596)*** |
| | $b_{1Eu}$ | — | .106 (.055) | .106 (.055) |
| | Model Fit | | | |
| | *-2LL* | 7257.990 | 7250.592 | 7250.592 |
| | *Δ-2LL (Δdf)* | — | 7.398 (+1) | — |
| | *p* | — | .007 | — |
| **Neighborhood Socioeconomic Advantage** | Main Effect of Neighborhood SES on Immune Functioning | | | |
| | $b_1$ | –.081 (.045) | –.055 (.054) | –.055 (.054) |
| | Effect of Neighborhood SES on Residual ACE Components of Immune Functioning | | | |
| | $b_{0Au}$ | 1.700 (.949) | .778 (.837) | .778 (.837) |
| | $b_{1Au}$ | — | ***.325 (.074)*** | ***.325 (.074)*** |
| | $b_{0Eu}$ | ***3.484 (.802)*** | ***4.514 (.653)*** | ***4.514 (.653)*** |
| | $b_{1Eu}$ | — | ***.231 (.061)*** | ***.231 (.061)*** |
| | Model Fit | | | |
| | *-2LL* | 6730.304 | 6670.206 | 6670.206 |
| | *Δ-2LL (Δdf)* | — | 60.098 (+2) | — |
| | *p* | — | <.001† | — |

*Note:* Standard errors presented within parentheses. Estimates p < .05 **bolded**. Estimates p < .01 *italicized*. Baseline model denoted by †.

**Neighborhood Socioeconomic Advantage.** Neighborhood-level socioeconomic advantage was marginally related to better immune functioning ($b$ = –0.081, $p$ = 0.073). Adolescents living in less deprived neighborhoods (third quartile) had immune functioning scores that exceeded their peers (those living in more disadvantaged neighborhoods; first quartile) by 0.19 standard deviations.

**G×E Interaction: Effect of SES on Genetic and Environmental Etiology of**

**Immune Functioning (Dissertation Aim 2).** The moderating effects of SES on variance in immune functioning are presented in Table 7.6 under the heading *Effect of SES on Residual ACE Components of Immune Functioning.*

**Parental Education.** The best-fitting model suggested that residual variance in immune functioning decreased as a function of increasing parental educational attainment, an effect driven by decreasing nonshared environmental variance ($b_{1Eu}$ = – 0.215, $p$ < 0.001). These model results are illustrated in Figure 7.59, which shows residual AE variance components of immune functioning as a function of parental educational attainment, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure clearly demonstrates how residual phenotypic and nonshared environmental variance in immune functioning decreases with increasing parental education. It seems that high-SES environments reduce environmental risk for poorer immune functioning.

These results are further illustrated in Figure 7.60. In the left panel of Figure 7.60, we show absolute pair differences in immune functioning in MZ and DZ twins as a function of parental educational attainment. The slope of the MZ line is negative at lower levels of parental education, consistent with decreasing nonshared environmental variance in immune functioning. Notably, the MZ line closely approximates the DZ line at higher levels of parental education despite departing from it at lower levels; this observation may explain why our model did not detect changes in additive genetic variance as a function of parental educational attainment. The violin plot (right panel of Figure 7.60) shows that this constrained variance effect appears to be the result of fewer observations at the upper end of immune functioning when parental education level

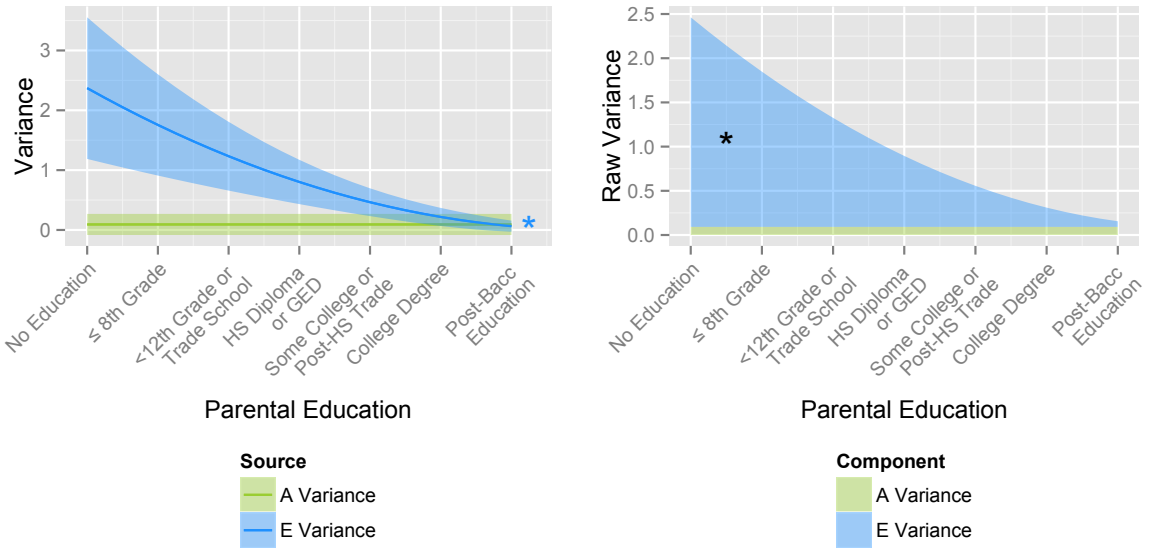


*Figure 7.59*. Gene-by-environment interaction between immune functioning and parental educational attainment in the Add Health sample. The left panel shows AE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in immune functioning decreases as a function of increasing parental education. The asterisks (*) indicate that variance in immune functioning depends on level of parental education.

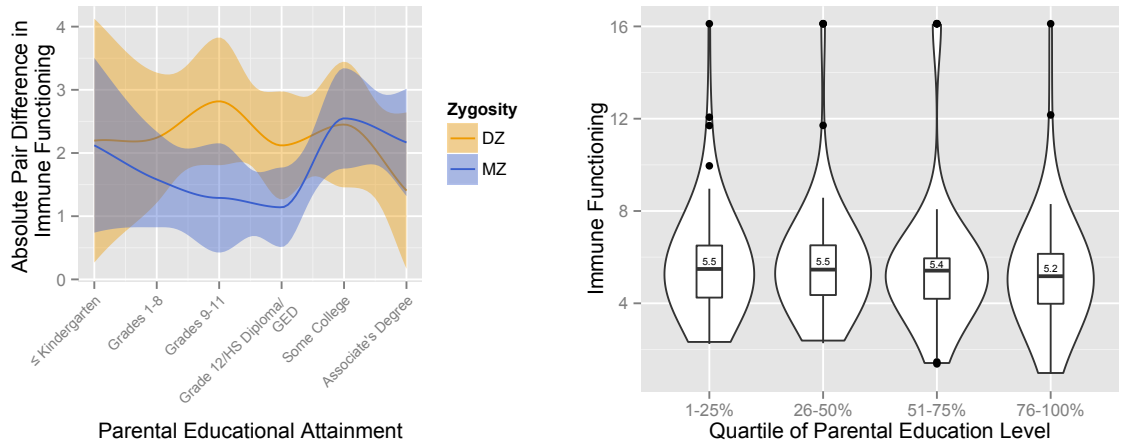


*Figure 7.60.* Illustrative analysis of the effects of parental educational attainment on variance in immune functioning in the Add Health sample. The left panel shows absolute pair differences in immune functioning as a function of parental education. The distance between the MZ and DZ lines represents the additive genetic variance in immune functioning. The location of the MZ represents nonshared environmental variance in immune functioning. The right panel shows box plots overlaid with violin plots, immune functioning as a function of quartile of parental education level.

is high (although note the increase at the highest quartile of parental education).

**Family Income.** The best-fitting model suggested that residual variance in immune functioning increased as a function of increasing family household income ($b_{1Eu}$ = 0.382, $p < 0.001$). These model results are illustrated in Figure 7.61, which shows residual phenotypic variance in immune functioning as a function of family household income, represented as both a regression line (with 95% confidence interval) and stacked variance. These results are also illustrated in Figure 7.62. Absolute pair differences in immune functioning as a function of family income level for MZ and DZ twins are presented in the left panel of Figure 7.62. The violin plots (right panel of Figure 7.62) show that this constricting variance effect appears to be the result of fewer observations in the upper tail of immune functioning when income level is high.

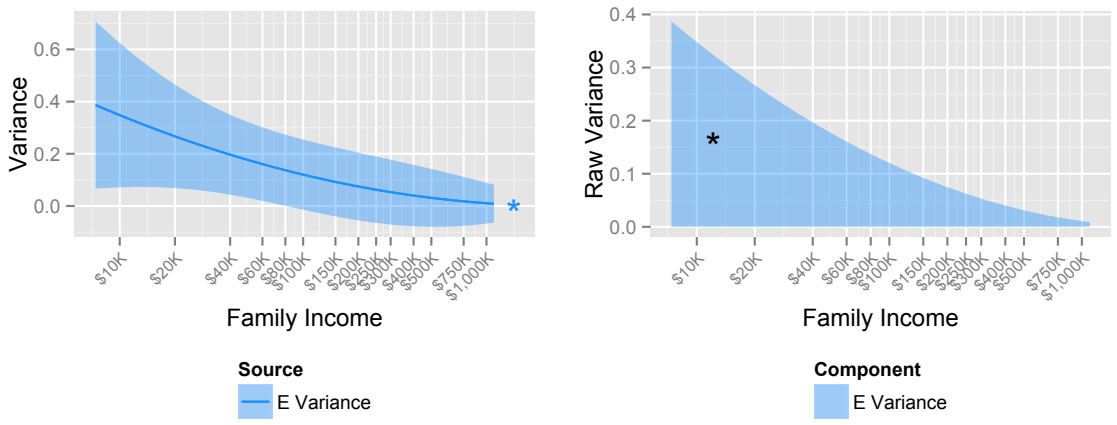


*Figure 7.61*. Gene-by-environment interaction between immune functioning and family household income in the Add Health sample. The left panel shows E variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in immune functioning decreases as a function of increasing family household income. The asterisks (*) indicate that variance in immune functioning depends on level of family household income.

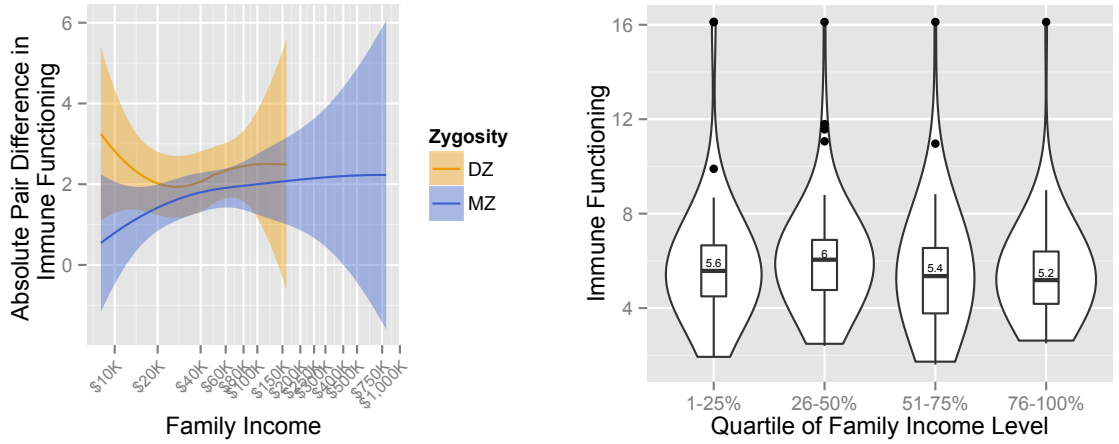


*Figure 7.62.* Illustrative analysis of the effects of family income on variance in immune functioning in the Add Health sample. The left panel shows absolute pair differences in immune functioning as a function of family income. The distance between the MZ and DZ lines represents the additive genetic variance in immune functioning. The location of the MZ represents nonshared environmental variance in immune functioning. The right panel shows box plots overlaid with violin plots, immune functioning as a function of quartile of family income level.

**Neighborhood Socioeconomic Advantage.** The best-fitting model suggested that residual variance in immune functioning is related to neighborhood-level socioeconomic advantage. This effect was driven by decreases in both additive genetic ($b_{1Au}$ = 0.325, $p < 0.001$) and nonshared environmental variance ($b_{1Eu}$ = 0.231, $p < 0.001$) as a function of increasing neighborhood-level SES. These model results are illustrated in Figure 7.63, which shows residual AE variance components of immune functioning as a function of neighborhood-level SES, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure clearly demonstrates how residual genetic and nonshared environmental variance in immune functioning decrease with increasing neighborhood socioeconomic advantage. It seems that high-SES environments protect against genetic and environmental risk for poorer immune functioning.

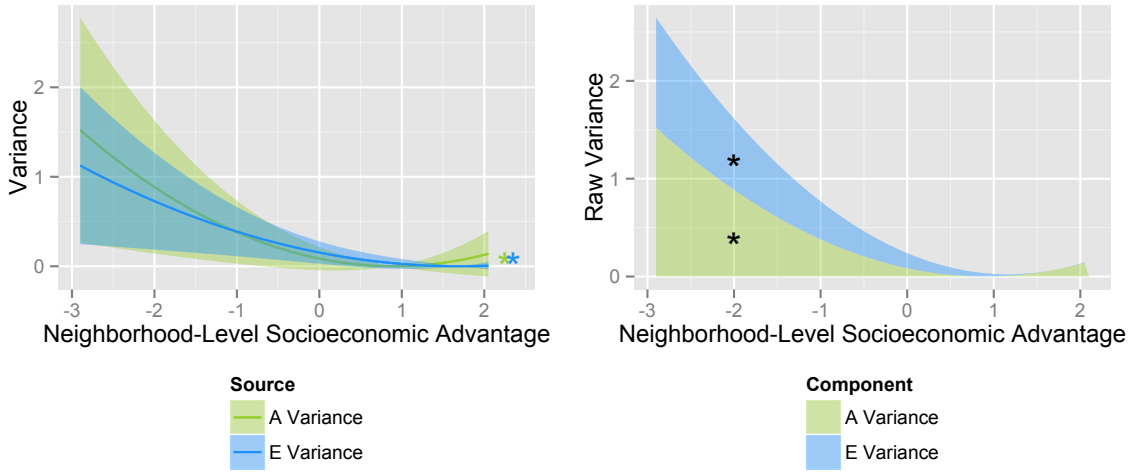


*Figure 7.63*. Gene-by-environment interaction between immune functioning and neighborhood-level socioeconomic advantage in the Add Health sample. The left panel shows AE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in immune functioning decreases as a function of increasing neighborhood-level SES. The asterisks (*) indicate that variance in immune functioning depends on level of neighborhood-level SES.

These results are further illustrated in Figure 7.64. Absolute pair differences in immune functioning as a function of neighborhood socioeconomic advantage for MZ and DZ twins are presented in the left panel of Figure 7.64. Consistent with decreasing nonshared environmental variance as a function of neighborhood SES, the MZ regression line decreases with increasing neighborhood SES. In addition, the gap between the MZ and DZ regression lines decreases as a function on increasing neighborhood SES, consistent with decreasing additive genetic variance in immune functioning with increasing SES. This effect appears to be driven by DZ twins being more similar in more advantaged neighborhoods. The violin plot (right panel of Figure 7.64) shows that this decreasing variance effect appears to be the result of fewer observations in the upper tail of immune functioning when neighborhood SES level is high.

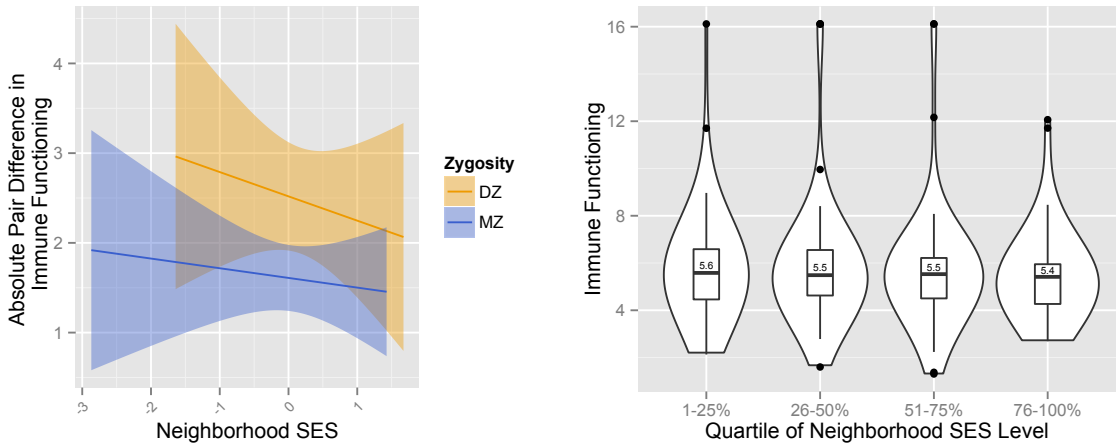


*Figure 7.64.* Illustrative analysis of the effects of neighborhood socioeconomic advantage on variance in immune functioning in the Add Health sample. The left panel shows absolute pair differences in immune functioning as a function of neighborhood SES. The distance between the MZ and DZ lines represents the additive genetic variance in immune functioning. The location of the MZ represents nonshared environmental variance in immune functioning. The right panel shows box plots overlaid with violin plots, immune functioning as a function of quartile of neighborhood SES level.

**Brief Summary.** Both compositional and contextual measures of socioeconomic status predicted immune functioning. Higher SES environments predicted greater mean levels of immune functioning. Similarly, higher SES predicted lower variance in immune functioning. Compositional SES conferred protective effects from environmental risk for poorer immune functioning; contextual measures protected against both genetic and environmental risk for poorer immune functioning.

## Chapter 8: Discussion – Physical Health

Research has long supported a link between socioeconomic status and physical health. We summarize the existing research in detail in Chapter 1, and highlight several general observations here. First, this relation is broad and spans many aspects of physical health, including self-rated health (Goodman, 1999; Eibner & Evans, 2005); various acute and chronic health conditions (Almqvist et al., 2005; Basagaña et al., 2004; Bengtsson et al., 2005; Cesaroni et al., 2003; Clark et al., 2009; Connolly et al., 2000; Cunningham & Kelsey, 1984; Diez-Roux et al., 1997; Evans et al., 2000; Haynes et al., 2006; Lee et al., 2011; Rabi et al., 2006; Stringhini et al., 2013; Sundquist et al., 2004); body mass index, adiposity, and obesity (Eibner & Evans, 2005; Stafford & Marmot, 2003); immune functioning (Alley et al., 2006; Chen et al., 2006; Dowd & Aiello, 2009; Deverts, Cohen, Kalra, & Matthews, 2012; Dowd et al., 2008); and mortality (Adler et al., 1994; Adler & Ostrove, 1999; Eibner & Evans, 2005; Goodman et al., 2003; Singh, 2003). Second, SES hastens the gradual decline in health that begins in middle adulthood (Williams et al., 2013). Third, existing behavior genetics research on the SES–physical health gradient demonstrates that this relation is far more nuanced than correlational or natural experimental studies suggest. This association is rarely found to be causal and is instead attributable to family-level confounds, especially additive genetic selection factors (Amin et al., 2015; Amin et al., 2013; Behrman et al., 2015; Behrman & Wolfe, 1989; Behrman et al., 2011; Dinescu et al., 2015; Fujiwara & Kawachi, 2009; Gerdtham et al., 2012; Johnson, 2007; Johnson & Krueger, 2005; Johnson et al., 2010; Johnson et al., 2011; Krieger et al., 2005; Lichtenstein et al., 1992; Lundborg, 2013; Madsen et al., 2010; Osler et al., 2007; Osler et al., 2009; Silventoinen et al., 2004; Svedberg et al.,

2006; Teasdale et al., 1990). Fourth, the complexities of this phenomenon are further made evident by research demonstrating SES effects on variance in physical health. Additive genetic variance in physical health tends to be greater in more disadvantaged environments (Johnson, 2007; Johnson et al., 2010; Johnson & Krueger, 2005; Johnson et al., 2011; Dinescu et al., 2015), although some studies showed this constraining effect for nonshared environmental variance as well (Johnson et al., 2011; Dinescu et al., 2015). Further, these variance changes have been interpreted as causal in nature.

This dissertation study used contemporary samples of American adolescent and adult twins reared in the same household to examine the impact of socioeconomic status on physical health more comprehensively. We examined the effect of both compositional (*e.g.*, educational attainment, household income) and contextual (*e.g.*, neighborhood socioeconomic advantage, area income inequality) measures of SES on level and variance in health, including general health (*i.e.*, self-rated health, health conditions), body mass index, and immune functioning.

**General Health**

We summarize the results for general health in Table 8.1. We provide information regarding the direction of the main effect of SES on general health (column labeled *Main Effect*) as well as the approximate percentage of the SES–health phenotypic correlation accounted for by the additive genetic, shared environmental, and nonshared environmental correlations (column labeled *Approx. %* r*P*). We also provide information regarding the direction of the influence of SES indicators on residual variance in general health (columns *Residual A*, *Residual C*, and *Residual E*), and provide a summary of the changes in variance in the context of SES–health ACE correlations and what those

Table 8.1. Summary of results for SES effects on general health.

| | SES Indicator | Main Effect | Approx. % *r*P | Residual A | Residual C | Residual E |
|---|---|---|---|---|---|---|
| Self-Rated Health (Add Health) | Parental Education | Protective | N/A | ∅ | — | ↓ |
| | Family Income | ∅ | N/A | ∅ | — | ↓ |
| | Neighborhood SES | ∅ | N/A | ∅ | — | ∅ |
| Self-Rated Health (WSTR) | Education | Protective (*r*C ↑, *r*E↓) | A = 20% → 50%<br>C = 10% → 50%<br>E = 70% → 0% | ∅ | ↓<br>lowest at high *r*C (causal) | ∅ |
| | Income | Protective (*r*C, *r*E) | A = 50% → 60%<br>C = 35% → 15%<br>E = 20% → 25% | ∅ | ↑<br>stable *r*C (causal) | ↓<br>stable *r*E (causal) |
| | Neighborhood SES | Protective (*r*C, *r*E) | —<br>C = 65% → 75%<br>E = 35% → 25% | ∅ | ↓<br>stable *r*C (causal) | ↓<br>stable *r*E (causal) |
| | Income Inequality | Protective (*r*A, *r*C) | A = 55%<br>C = 35%<br>E = 10% | ∅ | ∅ | ∅ |
| Health Conditions (WSTR) | Education | Protective (*r*A) | A = 80%<br>—<br>E = 20% | ∅ | — | ↓<br>stable *r*E (causal) |
| | Income | Protective (*r*A) | A = 80%<br>—<br>E = 20% | ↓<br>stable *r*A (causal) | — | ↓<br>stable *r*E (causal) |
| | Neighborhood SES | ∅ | —<br>—<br>— | ∅ | — | ↓<br>stable *r*E (causal) |
| | Income Inequality | ∅ | —<br>—<br>— | ∅ | — | ↓<br>stable *r*E (causal) |

changes imply about causality versus social selection processes.

As expected, socioeconomic status protected against poor self-rated health and presence of various health conditions. In our study, this was particularly true of compositional measures of SES, although contextual SES protected against poor self-rated health in adults as well. Where tests of causality were possible (*i.e.*, when SES indicators were not shared by members of a twin pair; true only for the WSTR), results were generally consistent with our research hypotheses: The effect of SES on general health was partially mediated by genetic and/or shared environmental pathways common to both phenotypes. We also observed evidence for a causal effect of SES on self-rated health, although we note that the nonshared environmental pathway generally accounted

for less than one-third of the total phenotypic correlation. In general, more advantaged individuals tend to report better overall health and endorse fewer health conditions not because of differential exposure to socioeconomic factors, but because of a genetic and/or shared environmental background common to both SES and physical health. These findings dovetail with the existing behavior genetics research suggesting a non-causal link between socioeconomic status and self-rated health (Amin et al., 2015; Behrman et al., 2015; Fujiwara & Kawachi, 2009; Gerdtham et al., 2012; Johnson et al., 2010; Lichtenstein et al., 1992; Lundborg, 2013; Krieger et al., 2005; Osler et al., 2007; Svedberg et al, 2006) and health conditions (Behrman et al., 2015; Behrman & Wolfe, 1989; Johnson & Krueger, 2005; Krieger et al., 2005; Lichtenstein et al., 1992).

As we noted with mental health, there is no "gene" for being rich that also confers physical health benefits. Instead, we argue that temperament or personality characteristics that carry a strong genetic basis (*e.g.*, neuroticism, conscientiousness) influence both phenotypes. For example, conscientious individuals may be more likely to pursue post-secondary education or seek higher paying or more skilled careers *and* be more likely to pay close attention to their health status or better adhere to physician recommendations. Somewhat unexpected, however, is that we observed that the shared environmental correlation (*r*C) between SES and self-rated health accounted for a high percentage of the total phenotypic correlation, particularly at higher SES levels. The common shared environmental pathway accounted for 10-35% of the total phenotypic correlation at the lowest SES levels, and upwards of 50-75% at the highest SES levels. This finding is somewhat unexpected given that adult phenotypes tend to demonstrate relatively small contributions from the shared environment (Beam & Turkheimer, 2013).

Of note, additive genetic and shared environmental pathways tend to be highly correlated. Indeed, the MZ:DZ pair ratio is important for precision of ACE variance and common pathway estimation (Visscher, 2004; Visscher, Gordon, & Neale, 2008), with smaller ratios yielding greater power to differentiate between these sources of covariation. The ratio of MZ:DZ twin pairs in the dissertation analyses ranged from 2.5-2.8, which could result in difficulty differentiating between shared A and C pathways. We considered whether this shared environmental association may be driven by less privileged individuals. To test this hypothesis, we fit models to a whites-only sample, but observed similar results: Shared environmental influences tended to contribute substantially, and sometimes to a greater extent than additive genetic influences to the overall phenotypic association between self-rated health and SES.

In contrast, the additive genetic correlation (*r*A) appeared largely responsible for this association when using chronic health conditions endorsed as the rubric for general health. It is interesting to see this difference between self-rated health and presence of chronic health conditions; future research will be needed to establish whether there may be differences in the mechanism by which SES impacts on self-rated health versus the presence of chronic health conditions.

Without exception, higher SES predicted less residual phenotypic variance in self-rated health and health conditions endorsed, an effect driven almost exclusively by decreases in nonshared environmental variance. Further, these changes occurred in the context of a stable nonshared environmental correlation (*r*E) with respect to socioeconomic status. That is, variance constriction as a function of SES was a causal process and was related to differential exposure to socioeconomic environments. These

results partially support our hypotheses. We predicted that higher SES environments would constrain phenotypic variance in general health (supported) and that this effect would be driven by decreases in raw additive genetic variance (*i.e.*, that low socioeconomic conditions potentiate genetic vulnerabilities for poor physical health; not supported). Past research has observed decreasing additive genetic variance in self-rated health and health conditions as a function of increasing SES (Johnson, 2007; Johnson et al., 2010; Johnson & Krueger, 2005), and we also observed this effect for one SES indicator (household income). Where our research findings depart from past research, however, is that we discovered that the mechanism driving the effect of SES on variance in general health is primarily a nonshared environmental one.

Like we observed with internalizing, socioeconomic status seemed to be working to bring in the tail of the health distribution representing worse health (lower tail for self-rated health, upper tail for health conditions endorsed). This observation in the context of E×E processes seems to support the dual distribution hypothesis (Tsang et al., 2017), which posits that the general health distribution is a combination of a normal distribution under strong genetic control and a skewed distribution that is strongly influenced by individual environmental processes. Further, because additive genetic variance in general health tended to be static with respect to SES while nonshared environmental variance decreased, heritability of general health (*i.e.*, the proportion of variance in general health accounted for by genes) increased as a function of SES[25]. That is, the relative importance of genes at high SES was substantially greater than at low levels of SES. This

[25] Note that this is a qualitative observation; no statistical tests were performed on standardized variance components in the dissertation analyses.

observation further supports the dual distribution hypothesis: Individuals at higher SES levels tend to fall on a normal distribution that is under strong genetic control because they have been less exposed to environmental processes that adversely impact on health; individuals at lower SES levels tend to fall on a skewed distribution that has substantial influence from the nonshared environment because they have experienced greater exposure to iatrogenic environmental processes.

***Implications.*** Similar to inferences we made regarding mental health (see Chapter 6), we note that because SES and general health are correlated via between-family pathways, income redistribution or higher education incentives are unlikely to cause quantifiable shifts in *mean levels* of self-rated health or chronic health conditions within the general population. Yet, our research suggests that low socioeconomic conditions exacerbate—in a causal manner—environmental risk for poor self-rated health or suffering from chronic health conditions. That is, better socioeconomic conditions may help to relieve the health burdens of some individuals in a regression-toward-the-mean fashion. General health on a grand scale remains unchanged, but *the poor health experienced by some will be less severe*.

Our findings strongly support early interventions for individuals at risk for poor physical health. We observed SES effects on variance in self-rated health and health conditions in both adolescents and adults. Policy makers might consider implementing school- or community-based interventions or health promotion programs for at-risk (*i.e.*, low-SES) children. For example, school districts (particularly those in more deprived areas) might implement programs in which school nurses perform regular physical health screenings for students. Similarly, workplace- or community-based programs may be

effective for at-risk adults. Many employers are turning to a system in which monetary incentives are offered for individuals who undergo annual physicals. Healthcare insurance providers are similarly offering subsidies based on more careful monitoring of one's health. More employers and healthcare insurance agencies might consider offering such incentives to their employees or clients. Within socioeconomically deprived neighborhoods, increasing accessibility to free or reduced-cost healthcare clinics may also be an effective intervention.

Healthcare providers and patients alike face difficult decisions when it comes to managing health conditions within the current standards of care. There is some evidence to suggest that healthcare providers prescribe or recommend treatments based on the economic resources available to an individual (Bernheim, Ross, Krumholz, & Bradley, 2008). For example, a middle-class individual who snores and chronically feels poorly rested (possible signs of sleep apnea) might be referred for a polysomnography (sleep study) by his or her physician, whereas a lower-class individual or a person without healthcare insurance might instead be encouraged to try nasal strips to treat the issue. Alternatively, low socioeconomic status may limit treatment adherence (Touchette & Shapiro, 2008). Policymakers might consider ways in which healthcare costs could be subsidized based on financial needs and insurance coverage (or lack thereof). Related, healthcare providers might offer services on a sliding scale to better serve the needs of low-SES individuals. In addition, insurance companies might consider a sliding scale model for copays or offering higher coverage rates for individuals at the lower end of income. Finally, healthcare providers should also be cognizant of environmental factors that may be adversely impacting on the health of individuals from deprived backgrounds.

**Body Mass Index**

We summarize results for the effects of socioeconomic status on body mass index in Table 8.2. Like we observed with general health, socioeconomic status protected against high BMI. Where tests of causality were possible (WSTR only), results overwhelmingly supported a non-causal explanation for this association. That is, family-level factors were largely responsible for predicting mean BMI levels. Somewhat unexpected is that the shared environmental pathway seemed particularly influential on the total phenotypic correlation between SES and BMI. Past research has largely supported a common additive genetic pathway (Dinescu et al., 2015; Johnson, 2007; Johnson & Krueger, 2005), although one report also found evidence of a common shared environmental pathway (Johnson et al., 2011). Adult studies of BMI do not typically demonstrate much evidence of shared environmental contributions to BMI (Elks et al., 2012), and shared environmental variance in our study was minimal as well ($c^2$ = 8%; see

Table 8.2. Summary of results for SES effects on body mass index.

| | SES Indicator | Main Effect | Approx. % *r*P | Residual A | Residual C | Residual E |
|---|---|---|---|---|---|---|
| BMI (Add Health) | Parental Education | Protective | N/A | ↓ | — | ∅ |
| | Family Income | ∅ | N/A | ∅ | — | ∅ |
| | Neighborhood SES | ∅ | N/A | ∅ | — | ∅ |
| BMI (WSTR) | Education | Protective (*r*C ↓) | A = 35% → 60%<br>C = 60% → 35%<br>E = 0% | ↓<br>stable *r*A (causal) | ↓<br>lowest at high *r*C (causal) | ↓<br>stable *r*E (causal) |
| | Income | Protective (*r*C, *r*E ↓) | A = 10%<br>C = 60% → 70%<br>E = 30% → 20% | ∅ | ↓<br>lowest at high *r*C (causal) | ↓<br>lowest at high *r*E (causal) |
| | Neighborhood SES | Protective (*r*C) | —<br>C = 100%<br>E = 0% | ↓<br>lowest at high *r*A (causal) | ∅ | ↓<br>lowest at high *r*E (causal) |
| | Income Inequality | Protective (*r*A, *r*C) | A = 70% → 80%<br>C = 30% → 20%<br>E = 0% | ∅ | ↓<br>lowest at high *r*C (causal) | ↓<br>lowest at high *r*E (causal) |

Chapter 4). Nevertheless, a good portion of the overlap between socioeconomic status and BMI appeared to be due to an underlying common shared environmental pathway. Like we did with self-rated health, we fit models to a whites-only sample and observed identical results. It is possible that samples demonstrating shared environmental contributions to BMI may reveal more about the manner in which SES and BMI are correlated, but future research to this effect will be needed to explore this possibility. We also note that the common additive genetic and shared environmental pathways tend to be highly correlated, and it is possible that the high MZ:DZ pair ratio we observed in the WSTR has created a condition in which we are detecting general between-family effects (rather than differentiating between A and C effects).

Consistent with our research hypotheses, socioeconomic advantage predicted reduced phenotypic variance in BMI. We predicted that this decrease would be driven primarily by decreases in additive genetic variance; this hypothesis was partially supported (particularly for variance in BMI during adolescence), although we note that we consistently observed decreases in environmental variance (particularly nonshared) across the board during adulthood. Further, these decreases were observed to be causal (*i.e.*, due to differential exposure to socioeconomic resources). Our results are consistent with past research suggesting that socioeconomic status causes decreases in additive genetic (Dinescu et al., 2015; Johnson, 2007; Johnson & Krueger, 2005) and nonshared environmental (Dinescu et al., 2015; Johnson et al., 2011) variance in BMI.

We observed that heritability of BMI seems to increase marginally with increasing SES (though it should be noted that we did not perform a statistical test on proportions of variance in BMI or any of the other phenotypes included in dissertation

analyses). This suggests that BMI is more heavily influenced by genetic factors at high SES compared with low SES. We also note that our E×E observations occurred in the context of fewer individuals in the upper tail of BMI at higher SES levels. These observations seem broadly consistent with the dual distribution hypothesis (Tsang et al., 2017) and serve as a reminder that pathological processes (such as obesity or high BMI) are individual processes that go beyond genetically programmed traits.

***Implications.*** Socioeconomic status and body mass index are correlated via between-family pathways; that is, economic resources do not demonstrate a causal effect of reducing BMI or obesity. Therefore, income redistribution or higher education incentives will not result in quantifiable shifts in *mean levels* of BMI or obesity within the general population. On the other hand, low socioeconomic conditions appear to cause increased environmental risk for high BMI and obesity, which suggests that improving individuals' socioeconomic conditions may help to reduce BMI or obesity in some individuals in a regression-toward-the-mean fashion. Body mass index on a grand scale remains unchanged, but *the obesity, and obesity-related illnesses, experienced by some will be less severe*.

Our findings suggest that early interventions for individuals at risk for high BMI may be effective. We observed almost no SES effects on variance in BMI in adolescents, but observed considerable effects in adults. Policy makers might consider implementing school- or community-based interventions or health promotion programs for at-risk (*i.e.*, low-SES) children. For example, expanding school recess time, securing funding for extracurricular activities that involve physical activity, or mandating that physical education courses remain a part of the academic curriculum throughout primary and

secondary school may help to combat obesity among childhood and adolescents. In addition, school districts might consider offering healthier food options in school cafeterias, offering healthy eating workshops for students and parents, or requiring nutrition courses as part of the academic curriculum.

Similarly, workplace- or community-based programs may demonstrate some efficacy for at-risk adults. Our results suggest that among adults, working to limit other environmental factors which exacerbate environmental risk for obesity is particularly important. Policy makers might consider developing the walkability of neighborhoods (particularly in low-SES areas), as living in more walkable neighborhoods is causally associated with lower BMI through the physical activity it promotes (Duncan, Cash, Horn, & Turkheimer, 2015). Encouraging philanthropic organizations such as the YMCA® to build low-membership fee gyms in low-SES neighborhoods may serve a dual purpose, both making workout facilities more accessible to low-SES individuals as well as drawing more economic and/or employment resources into low-SES neighborhoods. Incentivizing grocery stores or food cooperatives to pursue business ventures in low-SES neighborhoods may similarly help to reduce environmental factors that contribute to obesity. Healthcare providers should be aware that environmental factors that may be adversely impacting on the body composition of individuals from socioeconomically deprived backgrounds, and may spend additional time with patients to identify environmental factors that may interact with poor socioeconomic conditions to contribute to obesity (*e.g.*, suggest dietary modifications, encourage more physical activity or less engagement in sedentary behavior).

Obesity is correlated with many negative health outcomes, including type 2 diabetes mellitus, cardiovascular disease, and certain types of cancer (American Diabetes Association, 2008; Anderson et al., 2005; Flegal, Graubard, Williamson, & Gail, 2005; Pronk, Goodman, O'Conner, & Martinson, 1999; Wang, McPherson, Marsh, Gortmaker, & Brown, 2011). Our research findings also suggest that individuals at the low end of the socioeconomic spectrum may show greater vulnerability to developing obesity-related illnesses. We noted several implications in our discussion of general health; these implications are relevant to BMI and obesity as well.

**Immune Functioning**

We summarize results for the effects of socioeconomic status on immune functioning in Table 8.3. Measures of immune functioning were only available in the Add Health sample. Consistent with our hypotheses, parental education protected against poor immune functioning. Because parental education is a shared environmental moderator, it was not possible to make inferences regarding whether this effect is causal or instead represents a selection process. Consistent with our research hypotheses, family-level SES constrained residual phenotypic variance in immune functioning. This effect was primarily driven by decreasing nonshared environmental variance as a

Table 8.3. Summary of results for SES effects on immune functioning.

| | SES Indicator | Main Effect | Approx. % *r*P | Residual A | Residual C | Residual E |
|---|---|---|---|---|---|---|
| Immune Functioning (Add Health) | Parental Education | Protective | N/A | ∅ | — | ∅ |
| | Family Income | ∅ | N/A | — | — | ↓ |
| | Neighborhood SES | ∅ | N/A | ↓ | — | ↓ |

function of increasing SES (*i.e.*, was an E×E process). Further, it seems that SES operated by pulling in the upper tail of immune functioning; that is, fewer individuals demonstrated substantially elevated immune responses at lower levels of socioeconomic status. Overall, these results were largely consistent with the dual distribution hypothesis (Tsang et al., 2017).

***Implications.*** Our data again underscore the importance of early intervention among at-risk (*i.e.*, low-SES) youth, and suggest that relieving socioeconomic deprivation among adolescents (and their family-of-origin) may help to improve the immune functioning exhibited by some individuals by pulling them in off the fringes of poor immune functioning. The fact that socioeconomic status interacted primarily with the nonshared environment to predict variance in immune functioning suggests that interventions might be geared toward identifying environmental factors which may be contributing to poor immune functioning and are exacerbated by low socioeconomic status. Such factors may include diet or nutrition (*e.g.*, Hannigan, 1994; Kau, Ahern, Griffin, Goodman, & Gordon, 2011; Wintergerst, Maggini, & Hornig, 2007), psychological stress (Segerstrom & Miller, 2004), low levels of exercise (Pedersen & Hoffman-Goetz, 2000), poor sleep (Besedovsky, Lange, & Born, 2012), substance use (Arnson, Shoenfeld, & Amital, 2010; Cook, 1998), and exposure to harmful air pollutants such as smog (Bauer, Diaz-Sanchez, & Jaspers, 2012) or second-hand smoke (Arnson, Shoenfeld, & Amital, 2010; Bauer et al., 2012). Policy makers might consider implementing school- or community-based interventions or health promotion programs for at-risk individuals. For example, education initiatives regarding the effects of nutrition, substance use, and exercise on immune functioning might be offered at schools

or local community centers in lower-SES neighborhoods. We showed that lower-SES individuals are more at-risk for poor immune response; therefore, low-SES individuals might benefit from reduced cost or free vaccinations at local health clinics. Further, vaccination stations during flu season might also be warranted in lower-SES neighborhoods to help stop the spread of the flu within this at-risk population. Finally, physicians who encounter low-SES individuals might consider helping patients identify negative behaviors or iatrogenic environmental factors that may be adversely impacting on health or immune functioning; such interventions might coincide with childhood vaccination schedules.

**Summary**

Socioeconomic status protected against expression of poor physical health, but without exception was non-causally related to physical health indicators. Instead, these phenotypes shared a common genetic and/or shared environmental etiology. We hypothesize that this etiology is in the form of personality characteristics or temperaments with a strong genetic basis that predict behaviors which both interfere with upward movement on the socioeconomic spectrum *and* facilitate and/or maintain health pathology. In terms of real-world implications, these results imply that eliminating socioeconomic burdens in society will not influence mean physical health levels. We note, however, that our results indeed revealed much more about this complex relation.

Almost without exception, socioeconomic status predicted decreases in phenotypic variance in physical health indicators. Further, these decreases tended to be driven by decreases in nonshared environmental variance. In addition, these variance changes were the result of E×E interactions that were pulling individuals from the upper

tail of the distribution marking worse physical health or immune functioning. While income redistribution or widespread availability of educational opportunities may not affect mean levels of physical health, such social justice policies may reduce this burden for some, particularly those at greatest environmental risk for poor health.

# Chapter 9: Results – Health Behavior

The effect of socioeconomic status on health behavior was evaluated using several indicators of health behaviors, including substance use (*e.g.*, alcohol use, smoking status) and physical activity (*e.g.*, engagement in athletics or leisure exercise, time spent in moderate-to-vigorous physical activity) and sedentary behavior (*e.g.*, time spent watching television or playing video games). We discuss the influence of socioeconomic status on each of these indicators of health behaviors in turn.

## Substance Use

***Alcohol Use—Add Health.*** We fit the model presented in Figure 9.1 to the data separately for each socioeconomic status indicator, partialling for the linear effects of age and gender on level and variance in the drinking factor. We used likelihood ratio tests to determine the best fitting model. We first established a baseline model by conducting omnibus tests for the moderation by SES of the residual variance in alcohol use. We then systematically eliminated parameters from the baseline model to evaluate statistical significance of moderation parameters. Biometric decomposition of both the SES indicators and the alcohol use factor are presented in Table 4.1, and parameter estimates for each of these models are presented in Table 9.1. Parental household income and number of drinks were log-transformed to correct for positive skew. The latent alcohol use factor showed no evidence of influence from shared environmental factors; therefore, we did not include C in any of the models fit to the data.

**Main Effects of SES on Alcohol Use (Dissertation Aim 1).** The main effects of SES on alcohol use are presented in Table 9.1 under the heading *Main Effect of Moderator on Alcohol Use*. None of the socioeconomic indicators were related to mean

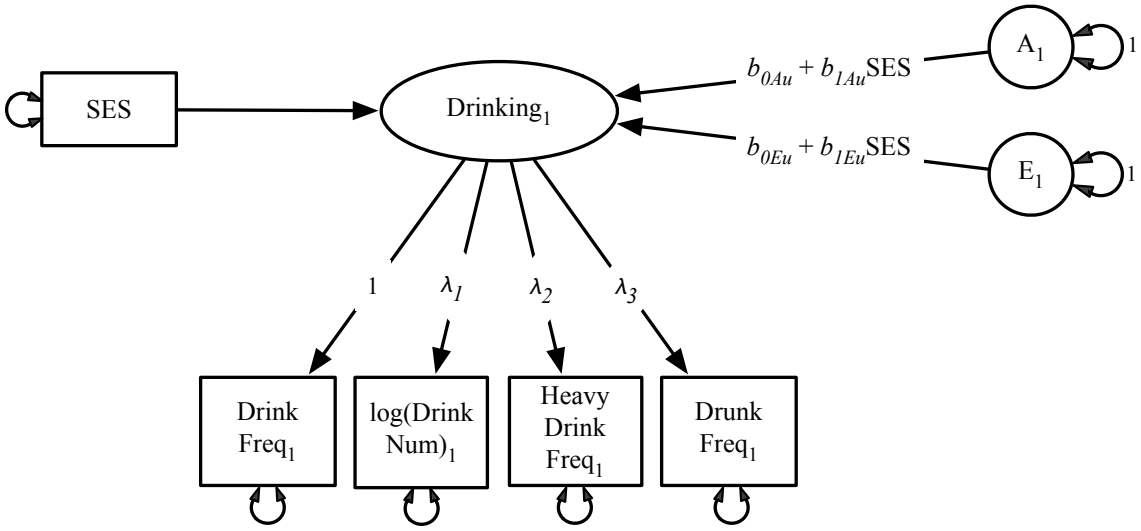


*Figure 9.1*. Path diagram G×SES model fit to alcohol use in the Add Health sample (only one twin shown for clarity). The residual variances for the alcohol use items were permitted to correlate across twins, and were estimated freely according to zygosity.

levels of alcohol use (parental education: $b = 0.026$, $p = 0.398$; family household income: $b = 0.037$, $p = 0.166$; neighborhood-level SES: $b = 0.019$, $p = 0.430$).

**G×E Interaction: Effect of SES on Genetic and Environmental Etiology of Alcohol Use (Dissertation Aim 2).** The moderating effects of SES on variance in alcohol use are presented in Table 9.1 under the heading *Effect of SES on Residual ACE Components of Alcohol Use.*

**Parental Education.** The best-fitting model suggested that residual phenotypic variance in alcohol use did not vary as a function of parental education.

**Family Income.** The best-fitting model suggested that residual variance in alcohol use increases with family income, an effect driven by increasing nonshared environmental variance ($b_{1Eu} = –0.041$, $p = 0.025$[26]). These model results are illustrated

[26] Although this coefficient is negative, nonshared environmental variance in fact was increasing as a function of family income level, holding gender and age constant.

Table 9.1. Parameter estimates and model fit statistics for G×SES models, alcohol use in the Add Health sample.

| | Parameter | Phenotypic Model (No Moderation) | Moderation of Residual Variance | Best-Fitting Model |
|---|---|---|---|---|
| **Parental Education** | Main Effect of Parental Education on Alcohol Use | | | |
| | $b_1$ | .026 (.030) | .023 (.031) | .026 (.030) |
| | Effect of Education on Residual ACE Components of Alcohol Use | | | |
| | $b_{0Au}$ | ***1.851 (.459)*** | ***1.700 (.489)*** | ***1.851 (.459)*** |
| | $b_{1Au}$ | — | .032 (.033) | — |
| | $b_{0Eu}$ | .635 (.342) | **.706 (.352)** | .635 (.342) |
| | $b_{1Eu}$ | — | –.022 (.026) | — |
| | Model Fit | | | |
| | *-2LL* | 15268.422 | 15267.180 | 15268.422 |
| | *Δ-2LL (Δdf)* | — | 1.242 (+2) | — |
| | *p* | —† | .537 | — |
| **Family Household Income** | Main Effect of Family Income on Alcohol Use | | | |
| | $b_1$ | .037 (.026) | ***.045 (.016)*** | .035 (.024) |
| | Effect of Family Income on Residual ACE Components of Alcohol Use | | | |
| | $b_{0Au}$ | ***1.737 (.529)*** | ***1.923 (.538)*** | ***1.861 (.541)*** |
| | $b_{1Au}$ | — | ***–.051 (.018)*** | — |
| | $b_{0Eu}$ | .309 (.370) | .076 (.380) | .092 (.382) |
| | $b_{1Eu}$ | — | ***–.043 (.016)*** | **–.041 (.018)** |
| | Model Fit | | | |
| | *-2LL* | 13558.120 | 13551.084 | 13553.816 |
| | *Δ-2LL (Δdf)* | — | 7.036 (+2) | 2.732 (–1) |
| | *p* | — | .030† | .098 |
| **Area Deprivation** | Main Effect of Neighborhood SES on Alcohol Use | | | |
| | $b_1$ | .019 (.024) | .016 (.056) | .019 (.024) |
| | Effect of Area Deprivation on Residual ACE Components of Alcohol Use | | | |
| | $b_{0Au}$ | ***3.426 (.537)*** | ***3.267 (.573)*** | ***3.426 (.537)*** |
| | $b_{1Au}$ | — | –.025 (.046) | — |
| | $b_{0Eu}$ | **.963 (.408)** | ***1.148 (.408)*** | **.963 (.408)** |
| | $b_{1Eu}$ | — | ***.205 (.072)*** | — |
| | Model Fit | | | |
| | *-2LL* | 9926.148 | 9924.200 | 9926.148 |
| | *Δ-2LL (Δdf)* | — | 1.948 (+2) | — |
| | *p* | —† | .378 | — |

*Note:* Standard errors presented within parentheses. Estimates p < .05 **bolded**. Estimates p < .01 *italicized*. Baseline model denoted by †.

in Figure 9.2, which shows residual AE variance components of alcohol use as a function of family household income, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure illustrates that high-SES environments potentiate genetic and environmental risk for alcohol use.

These results are also illustrated in Figure 9.3. Absolute pair differences in alcohol use as a function of family income level for MZ and DZ twins are presented in

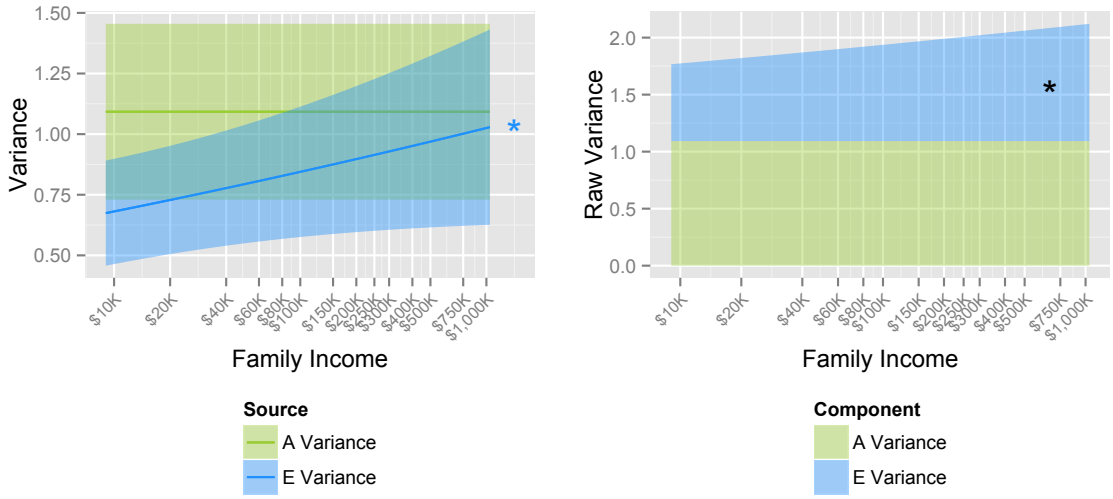


*Figure 9.2*. Gene-by-environment interaction between alcohol use and family income in the Add Health sample. The left panel shows AE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in alcohol use increases as a function of increasing family income. The asterisks (*) indicate that variance in alcohol use depends on level of family income.

the left panel of Figure 9.3. Consistent with increasing nonshared environmental variance as a function of family income, the MZ and DZ regression lines increase with increasing family income. In addition, the MZ and DZ lines closely approximate one another, consistent with no changes in additive genetic variation in this phenotype. The violin plots (right panel of Figure 9.3) show that this expanding variance effect appears to be the result of more observations in the upper tail of alcohol use when family income level is high.

**Neighborhood-Level Socioeconomic Advantage.** Like education, neighborhood SES was not a predictor of residual variance in alcohol use in this sample.

**Brief Summary.** Socioeconomic status was unrelated to mean levels of alcohol use. Higher parental income predicted increased nonshared environmental risk for alcohol use.

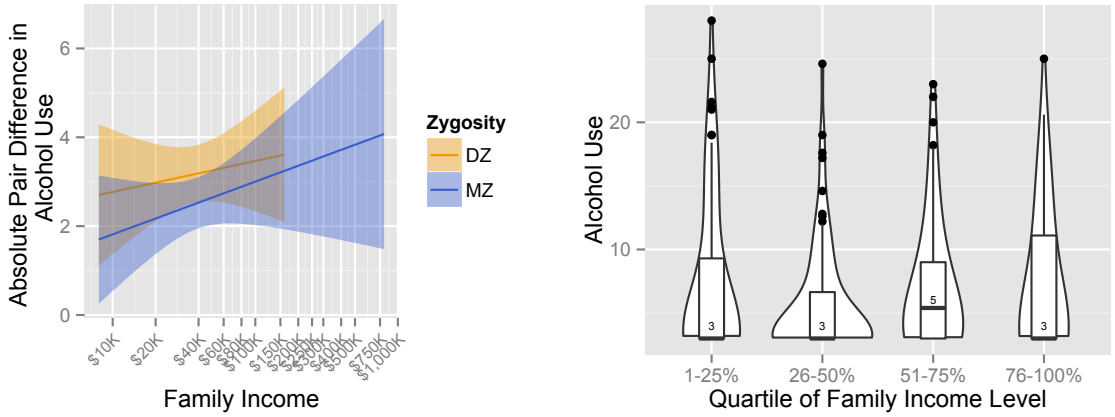


*Figure 9.3.* Illustrative analysis of the effects of family income on variance in alcohol use in the Add Health sample. The left panel shows absolute pair differences in alcohol use as a function of family income. The distance between the MZ and DZ lines represents the additive genetic variance in alcohol use. The location of the MZ represents nonshared environmental variance in alcohol use. The right panel shows box plots overlaid with violin plots, alcohol use as a function of quartile of family income level.

***Alcohol Use—WSTR.*** We fit the model presented in Figure 9.4 to the data separately for each socioeconomic status indicator, partialling for the linear effects of age and gender on level and variance in the latent alcohol use factor. We used likelihood ratio tests to determine the best fitting model. We first established a baseline model by conducting omnibus tests for the moderation by SES of both the residual variance in alcohol use and the main effects of SES on alcohol use. We then systematically eliminated parameters from the baseline model to evaluate statistical significance of moderation parameters. Biometric decomposition of both the SES indicators and alcohol use are presented in Table 4.2, and parameter estimates and model fit statistics for the baseline and best-fitting models presented in Table 9.2. The Area Deprivation Index was log-transformed to correct for positive skew, and the Gini Index was scaled by a factor of 10 to facilitate model convergence. These transformed values were used in all analyses in which the ADI or Gini Index was used as the indicator of contextual socioeconomic

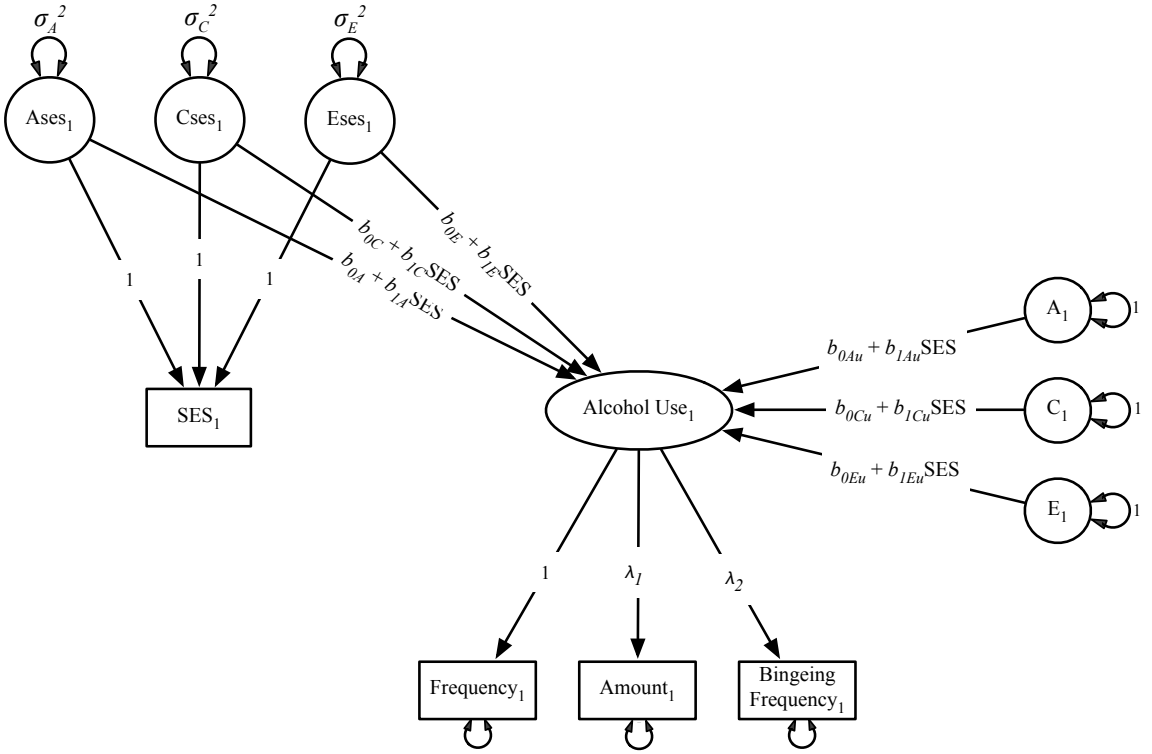


*Figure 9.4*. Path diagram G×SES model fit to latent alcohol use in the WSTR (only one twin shown for clarity). In the fully saturated model, the main effects of SES on alcohol use and the ACE variances of alcohol use vary as a function of SES. The residual variances for the alcohol use items were permitted to correlate across twins, and were estimated freely according to zygosity.

status. We did not include A for the ADI since there was no evidence of genetic influences on area deprivation in our univariate model.

**Causal Pathways vs. rGE: Main Effects of SES on Alcohol Use (Dissertation Aim 1).** The main effects of SES on body mass index are presented in Table 9.2 under the heading *Main Effect of Moderator on Alcohol Use*. We discuss the main effects results for each moderator below.

**Education.** Educational attainment was not phenotypically associated with latent alcohol use ($b = 0.007$, $p = 0.373$).

Table 9.2. Parameter estimates and model fit statistics for G×SES models, alcohol use in the WSTR.

| | Parameter | Phenotypic Model | Quasi-Causal Model | Moderation of Residual Variance | Moderation of Main Effects | Best-Fitting Model |
|---|---|---|---|---|---|---|
| **Education** | Main Effect of Education on Alcohol Use | | | | | |
| | $b_{0A}$ | –.007 (.008) | .045 (.050) | .043 (.052) | .215 (.170) | .043 (.052) |
| | $b_{1A}$ | — | — | — | –.028 (.027) | — |
| | $b_{0C}$ | –.007 (.008) | –.037 (.058) | –.055 (.059) | –.043 (.190) | –.055 (.059) |
| | $b_{1C}$ | — | — | — | –.002 (.030) | — |
| | $b_{0E}$ | –.007 (.008) | .003 (.015) | .003 (.015) | .038 (.081) | .003 (.015) |
| | $b_{1E}$ | — | — | — | –.006 (.013) | — |
| | Effect of Education on Residual ACE Components of Alcohol Use | | | | | |
| | $b_{0Au}$ | ***.549 (.081)*** | ***.547 (.081)*** | ***.834 (.160)*** | ***.824 (.162)*** | ***.834 (.160)*** |
| | $b_{1Au}$ | — | — | **–.046 (.019)** | **–.044 (.020)** | **–.046 (.019)** |
| | $b_{0Cu}$ | **.307 (.147)** | **.308 (.147)** | ***.879 (.162)*** | ***.883 (.161)*** | ***.879 (.162)*** |
| | $b_{1Cu}$ | | | ***–.105 (.015)*** | ***–.107 (.015)*** | ***–.105 (.015)*** |
| | $b_{0Eu}$ | ***.380 (.038)*** | ***.380 (.038)*** | ***.408 (.063)*** | ***.687 (.062)*** | ***.408 (.063)*** |
| | $b_{1Eu}$ | — | — | ***.051 (.008)*** | ***.051 (.008)*** | ***.051 (.008)*** |
| | Model Fit | | | | | |
| | *-2LL* | — | 58656.708 | 58448.644 | 58442.574 | 58448.644 |
| | *Δ-2LL (Δdf)* | — | — | 208.064 (+3) | 6.070 (+3) | — |
| | *p* | — | — | <.001† | .108 | — |
| **Household Income** | Main Effect of Income on Alcohol Use | | | | | |
| | $b_{0A}$ | .003 (.004) | .034 (.033) | .035 (.037) | ***.224 (.058)*** | .045 (.028) |
| | $b_{1A}$ | — | — | — | ***–.031 (.008)*** | — |
| | $b_{0C}$ | .003 (.004) | –.037 (.064) | –.054 (.074) | **–.208 (.091)** | .106 (.114) |
| | $b_{1C}$ | — | | | .022 (.013) | ***–.047 (.013)*** |
| | $b_{0E}$ | .003 (.004) | –.003 (.007) | –.001 (.007) | –.039 (.019) | –.005 (.007) |
| | $b_{1E}$ | — | — | — | .007 (.004) | — |
| | Effect of Income on Residual ACE Components of Alcohol Use | | | | | |
| | $b_{0Au}$ | ***.465 (.085)*** | ***.463 (.087)*** | ***.476 (.094)*** | ***1.102 (.135)*** | ***.520 (.096)*** |
| | $b_{1Au}$ | — | — | **–.017 (.008)** | **–.016 (.007)** | ***–.029 (.001)*** |
| | $b_{0Cu}$ | ***.362 (.136)*** | ***.359 (.138)*** | ***.431 (.123)*** | ***.519 (.185)*** | ***.448 (.119)*** |
| | $b_{1Cu}$ | | | .007 (.014) | .001 (.010) | — |
| | $b_{0Eu}$ | ***.399 (.039)*** | ***.396 (.039)*** | ***.504 (.046)*** | ***.514 (.046)*** | ***.488 (.045)*** |
| | $b_{1Eu}$ | — | — | ***–.021 (.005)*** | ***–.023 (.004)*** | ***–.019 (.005)*** |
| | Model Fit | | | | | |
| | *-2LL* | — | 63233.970 | 63164.730 | 63152.630 | 63154.780 |
| | *Δ-2LL (Δdf)* | — | — | 69.240 (+3) | 12.100 (+3) | 2.150 (–3) |
| | *p* | — | — | <.001 | .007† | .542 |
| **Area Deprivation** | Main Effect of Area Deprivation on Alcohol Use | | | | | |
| | $b_{0C}$ | .140 (.095) | ***.680 (.230)*** | ***.703 (.231)*** | .118 (.158) | ***.713 (.232)*** |
| | $b_{1C}$ | — | — | — | –.006 (.011) | — |
| | $b_{0E}$ | .140 (.095) | –.091 (.130) | –.163 (.148) | –.076 (.061) | –.168 (.150) |
| | $b_{1E}$ | — | — | — | .005 (.005) | — |
| | Effect of Area Deprivation on Residual ACE Components of Alcohol Use | | | | | |
| | $b_{0Au}$ | .757 (.556) | **.791 (.379)** | 1.363 (1.804) | .437 (.328) | ***.750 (.127)*** |
| | $b_{1Au}$ | — | — | –.140 (.401) | **.046 (.023)** | — |
| | $b_{0Cu}$ | .108 (.735) | .169 (.511) | ***5.622 (.894)*** | ***.439 (.110)*** | ***5.899 (.774)*** |
| | $b_{1Cu}$ | — | — | ***–1.277 (.171)*** | –.043 (.038) | ***–1.311 (.166)*** |
| | $b_{0Eu}$ | ***.226 (.059)*** | ***.227 (.056)*** | .827 (.517) | ***.382 (.108)*** | .827 (.517) |
| | $b_{1Eu}$ | — | — | –.137 (.110) | –.016 (.008) | — |
| | Model Fit | | | | | |
| | *-2LL* | — | 18355.382 | 18322.314 | 18320.818 | 18324.714 |
| | *Δ-2LL (Δdf)* | — | — | 33.068 (+3) | 1.496 (+2) | 2.400 (–2) |
| | *p* | — | — | <.001† | .473 | .301 |
| **Income Inequality** | Main Effect of Income Inequality on Alcohol Use | | | | | |
| | $b_{0A}$ | **.088 (.037)** | –.993 (1.636) | –.981 (1.642) | | –.993 (1.636) |
| | $b_{1A}$ | — | — | — | | — |
| | $b_{0C}$ | **.088 (.037)** | .598 (.413) | .595 (.417) | | .598 (.413) |
| | $b_{1C}$ | — | — | — | | — |
| | $b_{0E}$ | **.088 (.037)** | .059 (.054) | .059 (.055) | | .059 (.054) |
| | $b_{1E}$ | — | — | — | | — |
| | Effect of Income Inequality on Residual ACE Components of Alcohol Use | | | | | |
| | $b_{0Au}$ | ***.607 (.099)*** | ***.602 (.112)*** | .474 (.289) | | ***.602 (.112)*** |
| | $b_{1Au}$ | — | — | .028 (.063) | | — |
| | $b_{0Cu}$ | .122 (.215) | .099 (.232) | .054 (.444) | | .099 (.232) |
| | $b_{1Cu}$ | — | — | –.036 (.096) | | — |
| | $b_{0Eu}$ | ***.337 (.043)*** | ***.338 (.044)*** | .420 (.217) | | ***.338 (.044)*** |
| | $b_{1Eu}$ | — | — | –.018 (.048) | | — |
| | Model Fit | | | | | |
| | *-2LL* | — | 34208.414 | 34207.886 | | 34208.414 |
| | *Δ-2LL (Δdf)* | — | — | .528 (+3) | | — |
| | *p* | — | —† | .913 | | — |

*Note:* Standard errors presented within parentheses. Estimates $p < .05$ **bolded**. Estimates $p < .01$ *italicized*. Baseline model denoted by †.

**Income.** The phenotypic regression of latent alcohol use on household income was not statistically significant ($b$ = 0.003, $p$ = 0.410). The best-fitting model suggested, however, the presence of a negative shared environmental effect that emerges at high income levels (≥ \$60,000; $b_{0C}$ = 0.106, $p$ = 0.353; $b_{1C}$ = –0.047, $p$ = 0.029). As is evident in the left panel of Figure 9.5, the between-family contributions to this association tend to be larger than the within-family contributions, although none are statistically significant except C at high income. Additionally, between-family confounds collectively account for more of the total phenotypic correlation than within-family factors, evident in the right panel of Figure 9.5.

To demonstrate what these effects look like within and between twin pairs, we conducted an illustrative analysis of the main effect of household income on alcohol use and how it varies according to the level of household income, which we present in Figure 9.6. We identified twin pairs concordant for lower income levels (≤ \$40K) and pairs concordant for higher income levels (> \$50K). Within these groups, we then compared the mean alcohol use of the twin earning less (orange) with that of the twin earning more (navy). Overall, there is a main effect of household income on alcohol use such that greater income levels are associated with higher alcohol use on average (this is evident comparing the height of the bars in the left panel to those in the right panel). Examining the relation at the within-pair level, however, shows that at lower levels of household income (left panel) there is a large difference between DZ co-twins (but not MZ co-twins), consistent with genetic selection. At higher income levels of household income, there are no differences between MZ and DZ co-twins, consistent with shared

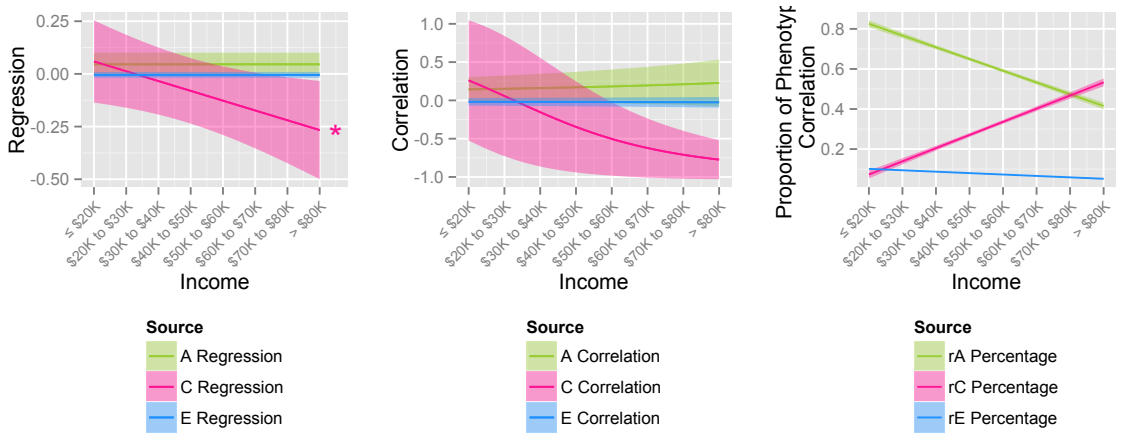


*Figure 9.5*. Main effects of household income on latent alcohol use in the WSTR. The left panel shows the regression of alcohol use on household income. The same relation is presented in the middle panel as genetic (green), shared environmental (pink), and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic, shared environmental, and nonshared environmental correlations. The asterisks (*) indicate that the magnitude of the regression of alcohol use on household income depends on income level (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). The shaded regions represent 95% confidence intervals around the regression lines.

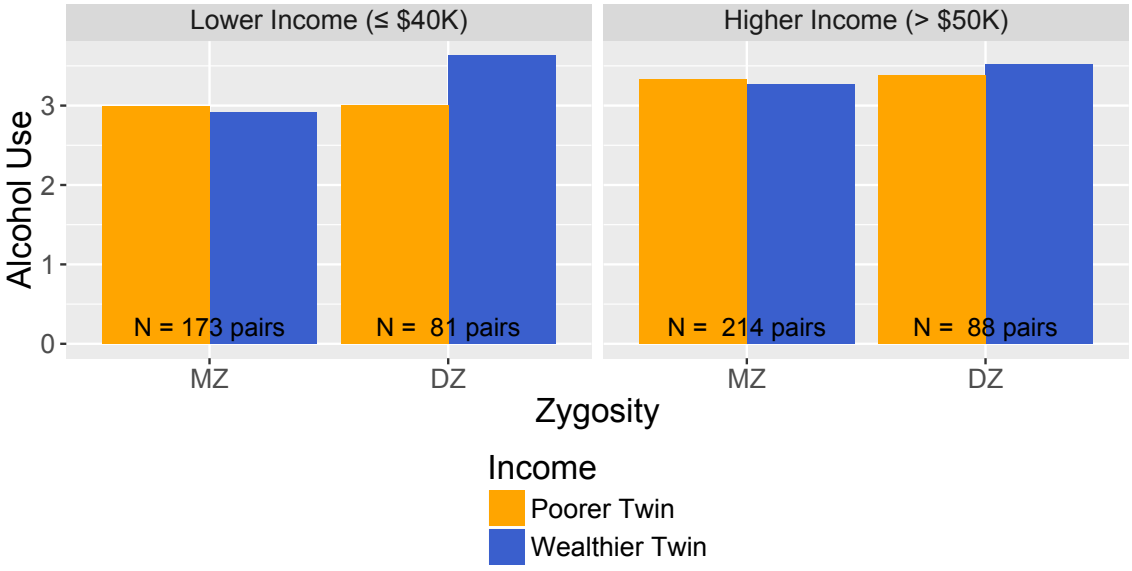


*Figure 9.6*. Illustrative analysis of the main effects of household income on alcohol use in the WSTR. This plot shows alcohol use at lower (≤ $40K; left panel) and higher (> $50K; right panel) levels of household income. Within each panel, the twin earning less income (orange) is compared with the twin earning more income (navy) in MZ and DZ twin pairs.

environmental selection. This plot demonstrates that genetic selection process is greatest at lower levels of household income, whereas shared environmental selection is greatest at higher income levels.

**Area Deprivation.** At the phenotypic level, the ADI was not statistically significantly associated with alcohol use ($b$ = 0.140, $p$ = 0.140). The best-fitting model suggested, however, that a statistically significant shared environmental pathway was present ($b_{0C}$ = 0.713, $p$ = 0.002) which was attenuated by a (nonsignificant) nonshared environmental pathway that was opposite in direction ($b_{0E}$ = –0.168, $p$ = 0.262; as a note, we did not observe genetic contributions to the ADI, and therefore no common genetic pathway was estimated). As is evident in the left panel of Figure 9.7, the between-family contribution to this association is larger than the within-family contribution. Also evident

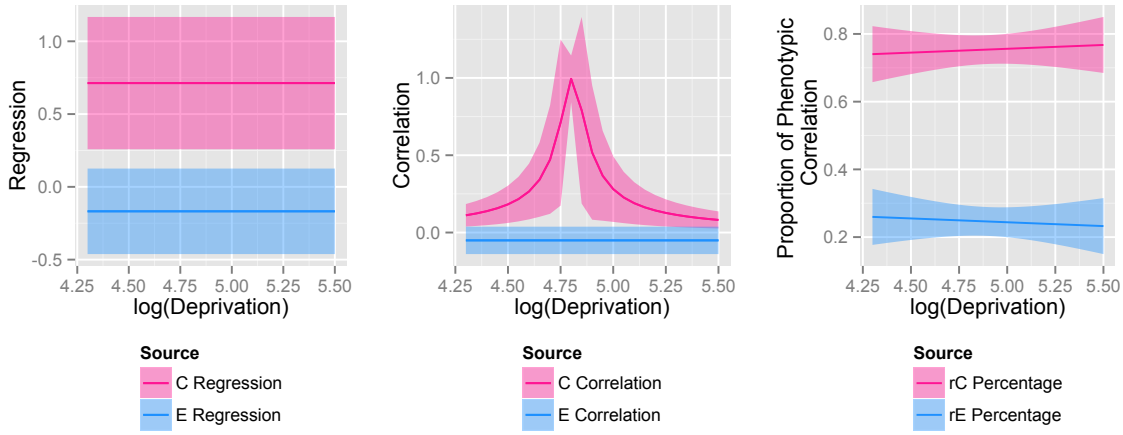


*Figure 9.7*. Main effects of area deprivation on latent alcohol use in the WSTR. The left panel shows the regression of alcohol use on the (log-transformed) ADI. The same relation is presented in the middle panel as shared environmental (pink) and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the shared and nonshared environmental correlations. The asterisks (*) indicate that the magnitude of the regression of alcohol use on area deprivation depends on level of deprivation (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). The shaded regions represent 95% confidence intervals around the regression lines.

is that the shared environmental pathway, compared with the nonshared environmental pathway, accounts for approximately three times as much of the total phenotypic correlation, as illustrated in the right panel of Figure 9.7.

Illustrative analyses further highlight this shared environmental correlation. In Figure 9.8, we show pair differences in alcohol use as a function of pair differences in the log-transformed ADI within randomly paired individuals (unrelated relation; blue line) and within MZ (red line) and DZ (green line) pairs. If the protective effect of lower area deprivation on alcohol use were causal, the slopes of these lines would closely approximate one another. Comparison of these lines suggests, however, that differences in the ADI do not predict differences in alcohol use within families, only between them. The slopes of the MZ and DZ lines are quite close, consistent with a common shared environmental pathway between area deprivation and alcohol use.

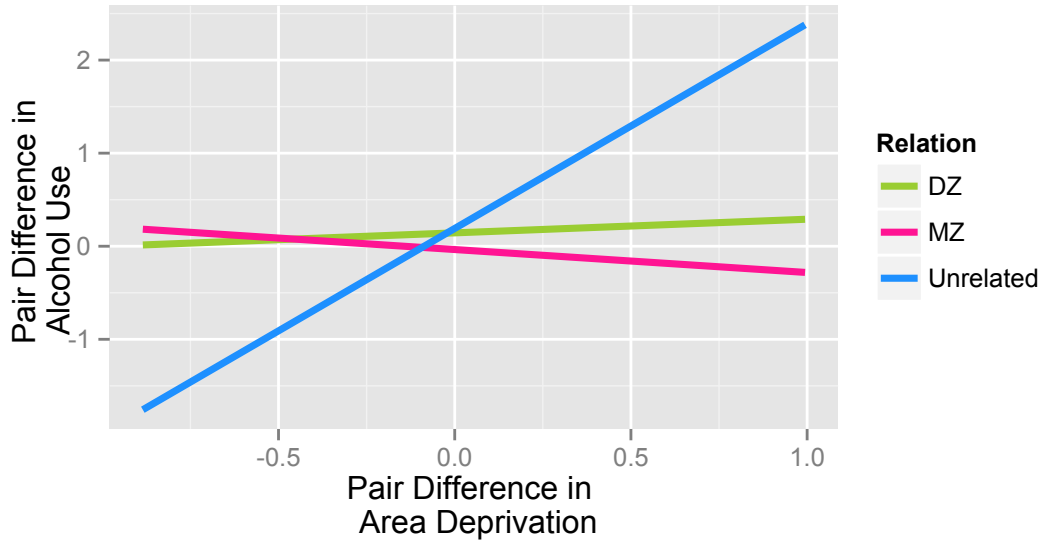


*Figure 9.8*. Illustrative analysis of the main effects of area deprivation on alcohol use in the WSTR. This plot shows pair differences in alcohol use as a function of pair differences in log-transformed ADI in the population (referred to as the ‘‘Unrelated Relation’’) and within pairs of MZ and DZ twins.

**Income Inequality.** At the phenotypic level, income inequality was statistically significantly associated with alcohol use ($b$ = 0.088, $p$ = 0.018), but the effect size was negligible. Individuals at the third quartile of the Gini Index had latent alcohol use scores that were 0.05 standard deviations higher on average than individuals at the first quartile of the Gini Index. The best fitting model suggested that alcohol use was not quasi-causally associated with income inequality ($b_{0E}$ = 0.059, $p$ = 0.276); the genetic ($b_{0A}$ = –0.991, $p$ = 0.544) and shared environmental ($b_{0C}$ = 0.598, $p$ = 0.147) pathways were also not statistically significant, which likely represents lack of power to differentiate between these sources of covariation. When estimating the total between-family effect (achieved by constraining $b_A$ and $b_C$ to be equal), however, there was significant positive between-family confounding between income inequality and alcohol use ($b_{0A}$ = $b_{0C}$ = 0.245, $p$ = 0.037; $b_{0E}$ = 0.040, $p$ = 0.427; results from this model not presented in Table 9.2). These results suggest that the relation between income inequality and alcohol use is best explained by between-family factors that are common to both phenotypes rather than by systematic differences in exposure to socioeconomic factors. As is evident in the left panel of Figure 9.9, the between-family contributions to this association are larger than the within-family contribution. In addition, between-family contributions account for approximately nine times as much of the total phenotypic correlation compared with the within-family contribution, as illustrated in the right panel of Figure 9.9.

These phenotypic and within-family effects are demonstrated in Figure 9.10, an illustrative analysis of the main effect of income inequality on alcohol use. We identified twin pairs concordant for living in counties with greater income inequality (orange), pairs

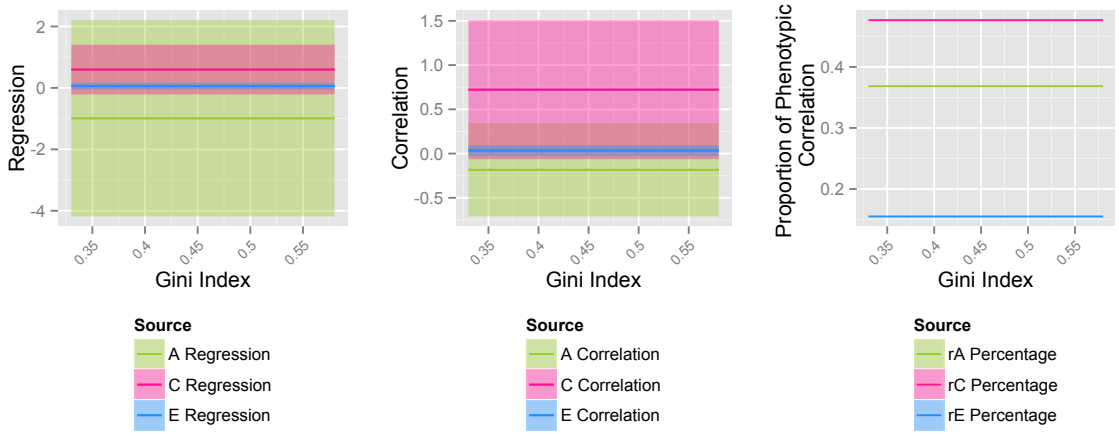


*Figure 9.9*. Main effects of income inequality on latent alcohol use in the WSTR. The left panel shows the regression of alcohol use on the Gini Index. The same relation is presented in the middle panel as genetic, shared environmental, and nonshared environmental correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic, shared environmental, and nonshared environmental correlations. The asterisks (*) indicate that the magnitude of the regression of alcohol use on income inequality depends on level of income inequality (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). The shaded regions represent 95% confidence intervals around the regression lines.

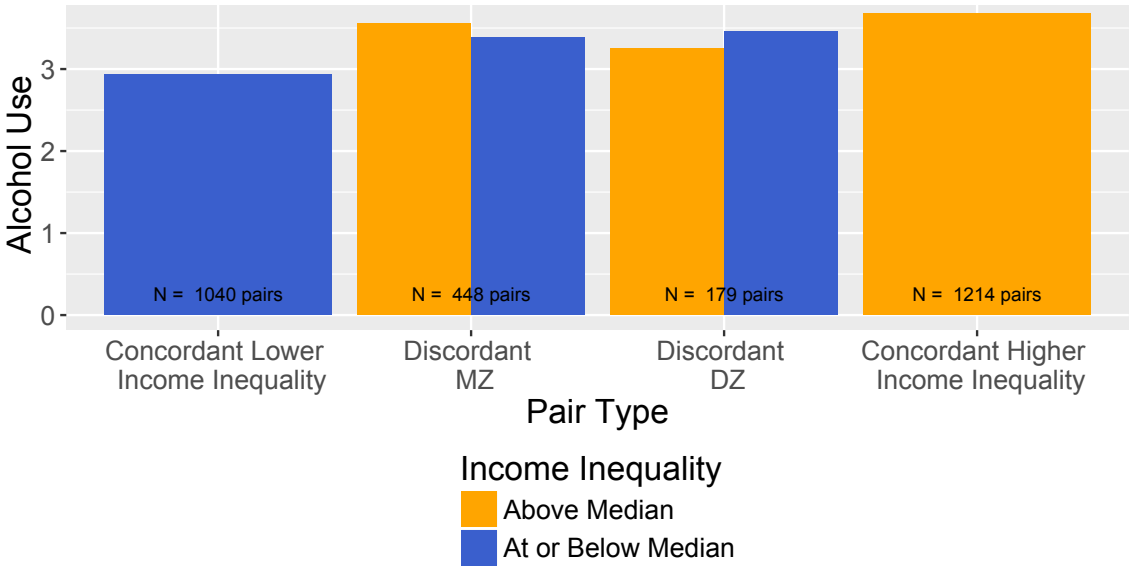


*Figure 9.10*. Illustrative analysis of the main effects of income inequality on alcohol use in the WSTR. The twin residing in a county with greater income inequality (orange) is compared with the twin residing in a county with lower income inequality (navy) in MZ and DZ twin pairs.

concordant for living in counties with lower income inequality (navy), and pairs discordant for county-level income inequality, and compared the mean alcohol use levels of individuals within each of these groups. Overall, there is a main effect of income inequality on alcohol use such that greater income inequality is associated with greater alcohol use (this is evident comparing the outer bars in this figure). It is also evident that this relationship is substantially reduced within pairs of MZ and DZ twins, consistent with between-family mediation of this association.

**G×E Interaction: Effect of SES on Genetic and Environmental Etiology of Alcohol Use (Dissertation Aim 2).** The moderating effects of SES on variance in latent alcohol use are presented in Table 9.2 under the heading *Effect of SES on Residual ACE Components of Alcohol Use.* We discuss the interactive effect of each SES indicator on variance in alcohol use below.

**Education.** The best fitting model suggested that residual phenotypic variance in latent alcohol use decreased with increasing educational attainment. This effect was driven by decreases in all ACE variance components ($b_{1Au}$ = –0.046, $p$ = 0.016; $b_{1Cu}$ = –0.105, $p < 0.001$; $b_{1Eu}$ = –0.051, $p < 0.001$). These model results are illustrated in Figure 9.11, which shows residual ACE variance components of alcohol use as a function of educational attainment, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure clearly demonstrates how residual phenotypic (and ACE) variance(s) in alcohol use decreases with increasing educational attainment. The correlation between alcohol use and education was static with respect to educational attainment, suggesting that low education potentiates genetic and

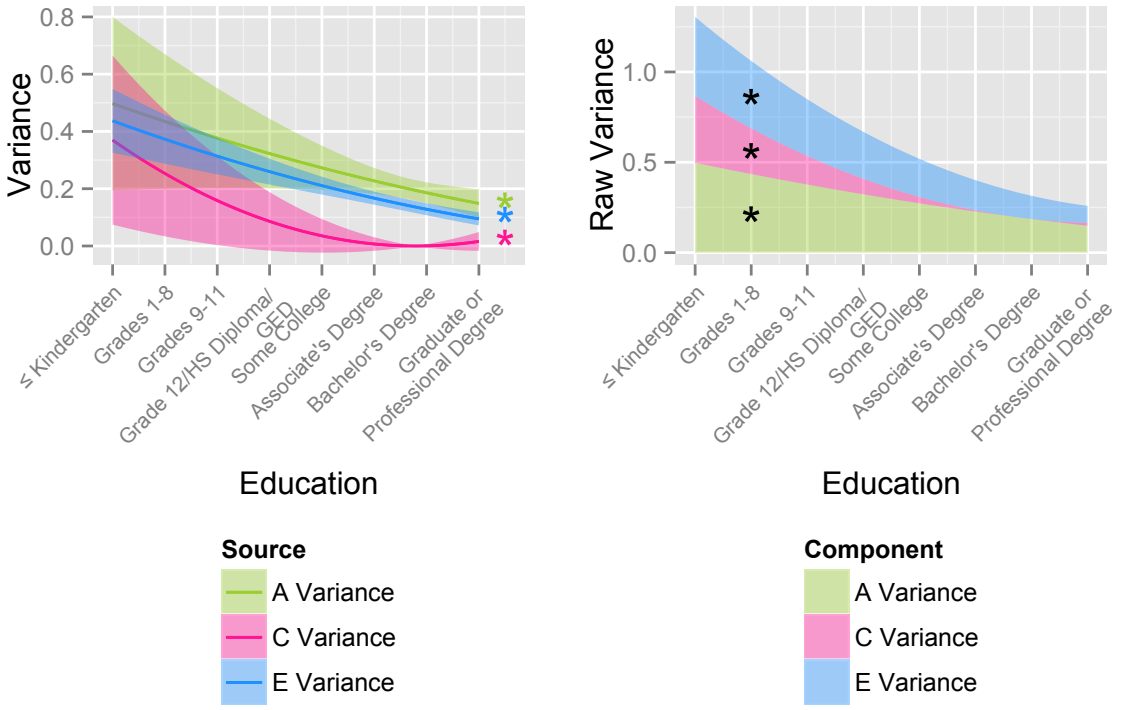


*Figure 9.11.* Gene-by-environment interaction between latent alcohol use and educational attainment in the WSTR. The left panel shows ACE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in alcohol use decreases as a function of increasing educational attainment. The asterisks (*) indicate that variance in alcohol use depends on level of education.

environmental risk for alcohol use.

These results are further illustrated in Figure 9.12. Evident in this plot is that total variance in alcohol use decreases as a function of increasing educational attainment. Consistent with decreasing nonshared environmental variance, the slope of the MZ regression line (left panel of Figure 9.12) is negative. Further, the MZ and DZ regression lines converge slightly, consistent with decreasing A variance as a function of increasing educational attainment. The violin plots (right panel of Figure 9.12) show that this constrained variance effect appears to be the result of fewer observations in the upper tail of alcohol use when educational level is high.

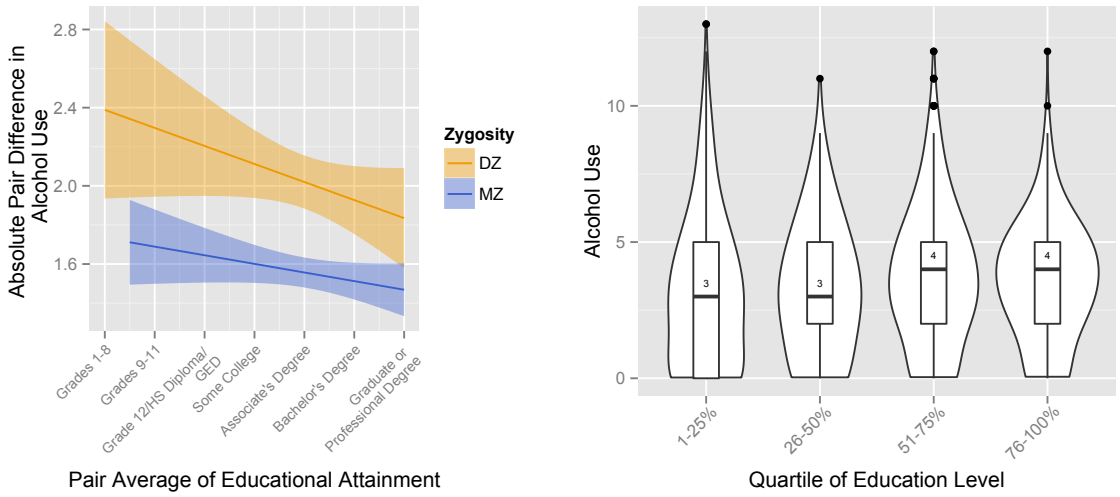


*Figure 9.12.* Illustrative analysis of the effects of educational attainment on variance in alcohol use in the WSTR. The left panel shows absolute pair differences in alcohol use as a function of educational attainment. The distance between the MZ and DZ lines represents the additive genetic variance in alcohol use. The location of the MZ represents nonshared environmental variance in alcohol use. The right panel shows box plots overlaid with violin plots, alcohol use as a function of quartile of education level.

**Income.** Like we observed with education, the best fitting model suggested that residual phenotypic variance in alcohol use decreased with increasing household income. This effect was driven by decreasing genetic ($b_{1Au}$ = –0.029, $p$ = 0.008) and nonshared environmental variance ($b_{1Eu}$ = –0.019, $p$ < 0.001). There was no moderation of shared environmental variance as a function of household income. These model results are illustrated in Figure 9.13, which shows residual ACE variance components of alcohol use as a function of household income, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure clearly demonstrates how residual phenotypic and genetic and nonshared environmental variance in alcohol use decreases with increasing household income. Interpreted in the context of the dynamics of ACE correlations, low household income appears to potentiate genetic and nonshared environmental risk for alcohol use.

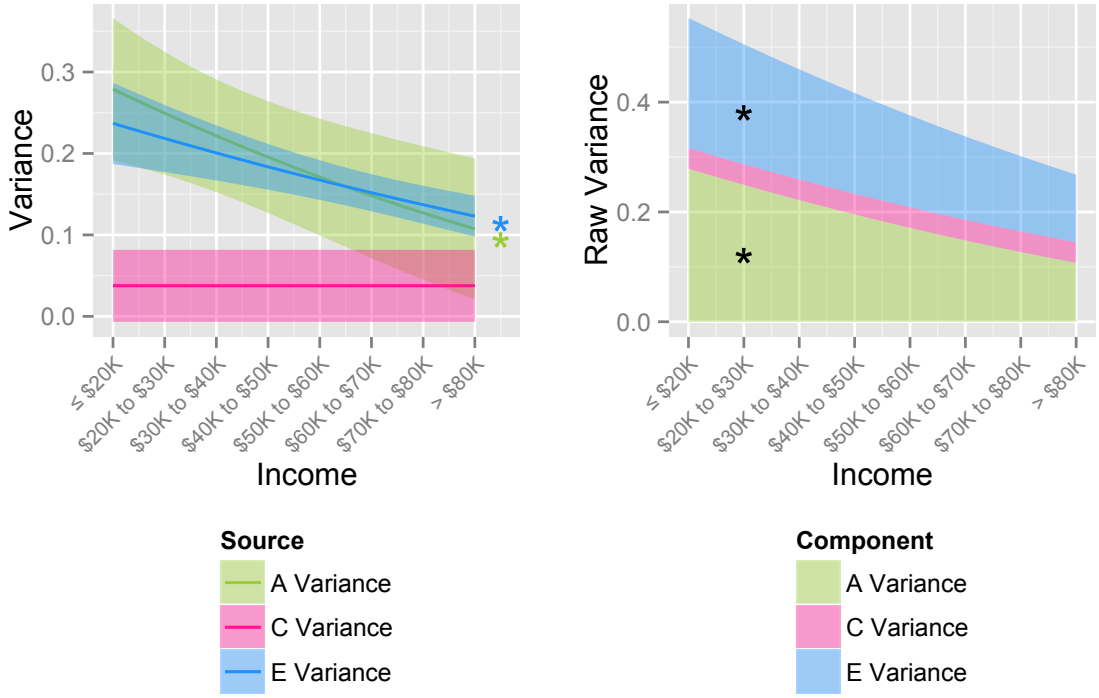


*Figure 9.13*. Gene-by-environment interaction between latent alcohol use and household income in the WSTR. The left panel shows ACE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in alcohol use decreases as a function of increasing household income. The asterisks (*) indicate that variance in alcohol use depends on income level.

These results are further illustrated in Figure 9.14. As we observed with educational attainment, total variance in alcohol use decreases as a function of increasing household income. Consistent with decreasing nonshared environmental variance, the slope of the MZ regression line (left panel of Figure 9.14) is negative. In addition, the DZ regression line approaches the MZ regression line at high levels of household income, consistent with decreasing A variance as a function of income level. The violin plots (right panel of Figure 9.14) show that this constrained variance effect appears to be the result of fewer observations in the upper tail of alcohol use when income level is high.

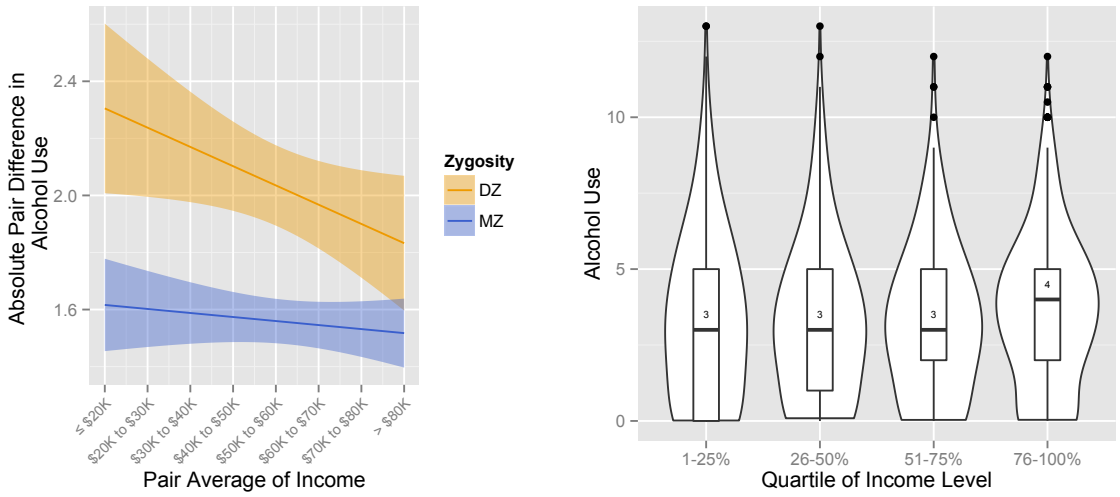


*Figure 9.14.* Illustrative analysis of the effects of household income on variance in alcohol use in the WSTR. The left panel shows absolute pair differences in alcohol use as a function of household income. The distance between the MZ and DZ lines represents the additive genetic variance in alcohol use. The location of the MZ represents nonshared environmental variance in alcohol use. The right panel shows box plots overlaid with violin plots, alcohol use as a function of quartile of income level.

**Area Deprivation.** The best fitting model suggested that residual phenotypic variance in alcohol use decreases with decreasing area deprivation. This effect was driven by decreasing shared environmental variance ($b_{1Cu}$ = –1.311, $p < 0.001$). No moderation of genetic or nonshared environmental variance was evident. These model results are illustrated in Figure 9.15, which shows residual ACE variance components of latent alcohol use as a function of area deprivation, represented as both regression lines (with 95% confidence intervals) and stacked variances. This plot illustrates the decrease in residual phenotypic and shared environmental variance in alcohol use as a function of decreasing area deprivation. The shared environmental correlation as a function of the ADI was lowest where shared environmental variance was highest, suggesting that high area deprivation potentiates family environmental risk for high alcohol use. These results are further illustrated in Figure 9.16.

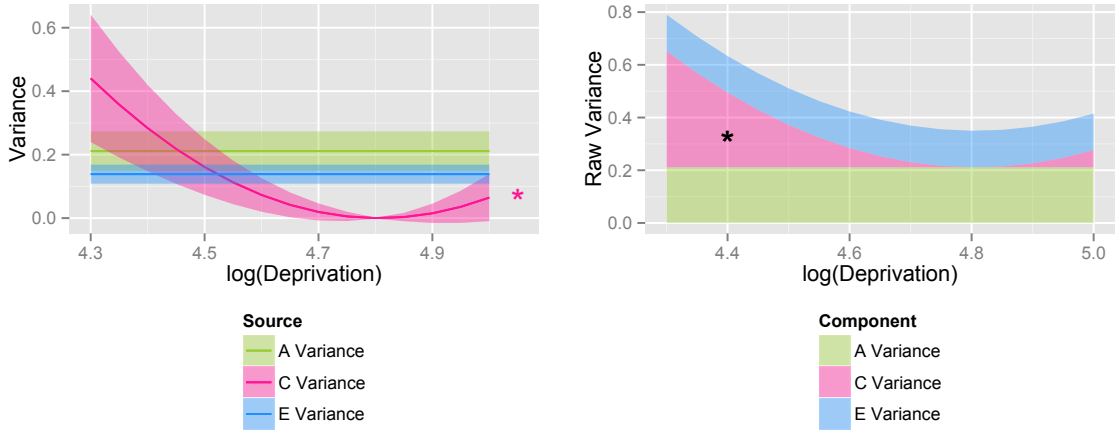


*Figure 9.15*. Gene-by-environment interaction between alcohol use and area deprivation in the WSTR. The left panel shows ACE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in alcohol use decreases as a function of decreasing area deprivation. The asterisks (*) indicate that variance in alcohol use depends on income level. *Note:* Approximately 96% of individuals in the WSTR sample had log-transformed ADI scores under 5.0; therefore, we opted to plot he model-predicted results within the range of the majority of individuals in our sample in order to keep the plots representative of the data.

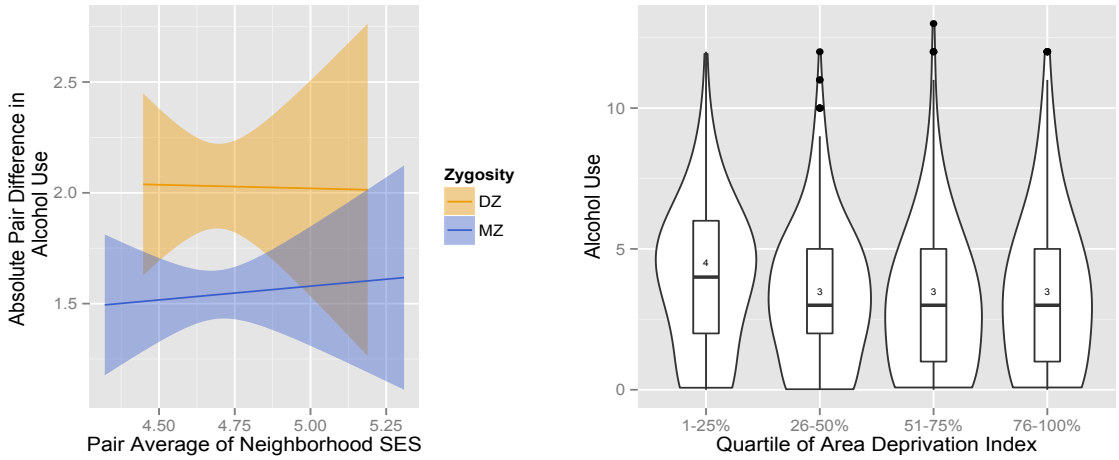


*Figure 9.16.* Illustrative analysis of the effects of neighborhood socioeconomic advantage on variance in alcohol use in the WSTR. The left panel shows absolute pair differences in alcohol use as a function of neighborhood SES. The distance between the MZ and DZ lines represents the additive genetic variance in alcohol use. The location of the MZ represents nonshared environmental variance in alcohol use. The right panel shows box plots overlaid with violin plots, alcohol use as a function of quartile of neighborhood SES level.

**Income Inequality.** The best fitting model suggested that residual phenotypic variance in alcohol use does not vary as a function of county-level income inequality.

**Brief Summary.** Socioeconomic status indicators did not tend to be correlated with alcohol use at the phenotypic level. There was evidence, however, of between-family confounds in these associations which were attenuated by competing within-family influences; the between-family influences were larger than the within-family influences, and accounted for a large majority of the total phenotypic correlation. Residual phenotypic variance in alcohol use tended to decrease with increasing SES; this decrease was driven by decreases in genetic and environmental variance as a function of compositional measures of SES, and shared environmental variance as a function of contextual measures of SES. Finally, decreases in these residual variances appeared to be potentiated by socioeconomic status, and not by social selection factors.

***Smoking—Add Health.*** Cigarette use in the Add Health sample was measured using a single item assessing the number of days smoked during the past 30 days (see Chapter 3). Respondents indicating that they smoked one or more days per month were considered to be a smoker. We fit the model presented in Figure 9.17 to the data separately for each socioeconomic indicator, partialling for the effects of age and gender on level and variance in smoking. We used likelihood ratio tests to determine the best fitting model. We first established a baseline model by conducting omnibus tests for the moderation by SES of the residual variance in smoking status. We then systematically eliminated parameters from the baseline model to evaluate statistical significance of moderation parameters. Biometric decomposition of both the SES indicators and smoking status are presented in Table 4.1, and parameter estimates for each of these

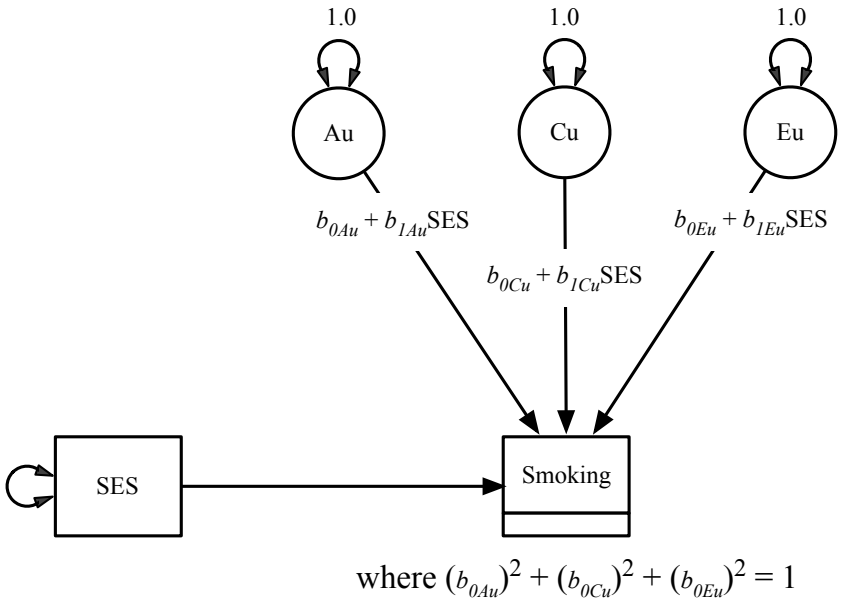


*Figure 9.17*. Path diagram G×SES model fit to smoking in the National Longitudinal Study of Adolescent Health sample (only one twin shown for clarity).

models are presented in Table 9.3.

**Causal Pathways vs. rGE: Main Effects of SES on Smoking (Dissertation Aim 1).** The main effects of SES on smoking status are presented in Table 9.3 under the heading *Main Effect of Moderator on Smoking*. None of the socioeconomic indicators showed a statistically significant association with smoking status (parental education: $b$ = 0.007, 95% confidence interval = –0.798 to 2.584; family income: $b$ = 0.020, 95% confidence interval = –1.577 to 4.309; neighborhood socioeconomic advantage: $b$ = 0.114, 95% confidence interval = –10.000 to 8.901).

**G×E Interaction: Effect of SES on Genetic and Environmental Etiology of Smoking (Dissertation Aim 2).** The moderating effects of SES on variance in smoking status are presented in Table 9.3 under the heading *Effect of SES on Residual ACE Components of Smoking.* None of the socioeconomic indicators predicted residual

Table 9.3. Parameter estimates and model fit statistics for G×SES models, ADHD diagnosis in the WSTR.

| | Parameter | Phenotypic Model | Moderation of Residual Variance | Best-Fitting Model |
|---|---|---|---|---|
| **Parental Education** | Main Effect of Parental Education on Smoking | | | |
| | $b_1$ | .007 (–.798, 2.584) | –.764 (–1.698, .350) | .007 (–.798, 2.584) |
| | Effect of Parental Education on Residual ACE Components of Smoking | | | |
| | $b_{0Au}$ | .915 (–1.000, 1.000) | .441 (–1.000, 1.000) | .915 (–1.000, 1.000) |
| | $b_{1Au}$ | — | **2.221 (.046, 4.347)** | — |
| | $b_{0Cu}$ | .169 (–1.000, 1.000) | .443 (–1.000, 1.000) | .169 (–1.000, 1.000) |
| | $b_{1Cu}$ | — | –.272 (–1.879, 2.203) | — |
| | $b_{0Eu}$ | .498 (–1.000, 1.000) | .894 (–1.000, 1.000) | .498 (–1.000, 1.000) |
| | $b_{1Eu}$ | — | –.791 (–1.652, 2.614) | — |
| | Model Fit | | | |
| | *-2LL* | 865.902 | 861.308 | 865.902 |
| | *Δ-2LL (Δdf)* | — | 4.594 (+3) | — |
| | *p* | —† | .204 | — |
| **Family Household Income** | Main Effect of Family Income on Smoking | | | |
| | $b_1$ | .020 (–1.577, 4.309) | –.024 (–6.147, .305) | .020 (–1.577, 4.309) |
| | Effect of Family Income on Residual ACE Components of Smoking | | | |
| | $b_{0Au}$ | .981 (–1.000, 1.000) | .964 (–1.000, 1.000) | .981 (–1.000, 1.000) |
| | $b_{1Au}$ | — | **.175 (.022, 10.00)** | — |
| | $b_{0Cu}$ | .358 (–1.000, 1.000) | .191 (–1.000, 1.000) | .358 (–1.000, 1.000) |
| | $b_{1Cu}$ | — | –.076 (–10.00, 10.00) | — |
| | $b_{0Eu}$ | .075 (–1.000, 1.000) | .541 (–1.000, 1.000) | .075 (–1.000, 1.000) |
| | $b_{1Eu}$ | — | –.001 (–1.826, 3.855) | — |
| | Model Fit | | | |
| | *-2LL* | 785.116 | 778.804 | 785.116 |
| | *Δ-2LL (Δdf)* | — | 6.315 (+3) | — |
| | *p* | —† | .097 | — |
| **Neighborhood Socioeconomic Advantage** | Main Effect of Neighborhood SES on Smoking | | | |
| | $b_1$ | .114 (–10.00, 8.901) | .274 (–8.097, 10.00) | .114 (–10.00, 8.901) |
| | Effect of Neighborhood SES on Residual ACE Components of Smoking | | | |
| | $b_{0Au}$ | .265 (–1.000, 1.000) | .627 (–1.000, 1.000) | .265 (–1.000, 1.000) |
| | $b_{1Au}$ | — | .335 (–10.00, 10.00) | — |
| | $b_{0Eu}$ | .964 (–1.000, 1.000) | .779 (-1.000, 1.000) | .964 (–1.000, 1.000) |
| | $b_{1Eu}$ | — | .440 (–10.00, .940) | — |
| | Model Fit | | | |
| | *-2LL* | 773.543 | 770.236 | 773.543 |
| | *Δ-2LL (Δdf)* | — | 3.307 (+3) | — |
| | *p* | —† | .191 | — |

*Note:* 95% confidence intervals presented within parentheses. Estimates $p < .05$ **bolded**. Baseline model denoted by †.

variance in smoking status.

**Brief Summary.** Neither compositional nor contextual measures of socioeconomic status were found to be protective against being a current smoker. Socioeconomic status was also not associated with residual variance in smoking.

***Smoking—WSTR.*** Cigarette use in the WSTR was measured using a single binary item assessing whether the individual currently smokes (see Chapter 3). We fit

the model presented in Figure 9.18 to the data separately for each socioeconomic indicator, partialling for the effects of age and gender on level and variance in smoking. We used likelihood ratio tests to determine the best fitting model. We first established a baseline model by conducting omnibus tests for the moderation by SES of both the residual variance in smoking and the main effects of SES on smoking. We then systematically eliminated parameters from the baseline model to evaluate statistical significance of moderation parameters. Biometric decomposition of both the SES indicators and smoking status are presented in Table 4.2, and parameter estimates for each of these models are presented in Table 9.4. The Area Deprivation Index was log-transformed to correct for positive skew, and the Gini Index was scaled by a factor of 100 to facilitate model convergence. These transformed values were used in all analyses in which the ADI or Gini Index was used as the indicator of contextual socioeconomic status. We did not include A for the ADI since there was no evidence of genetic influences on area deprivation in our univariate model.

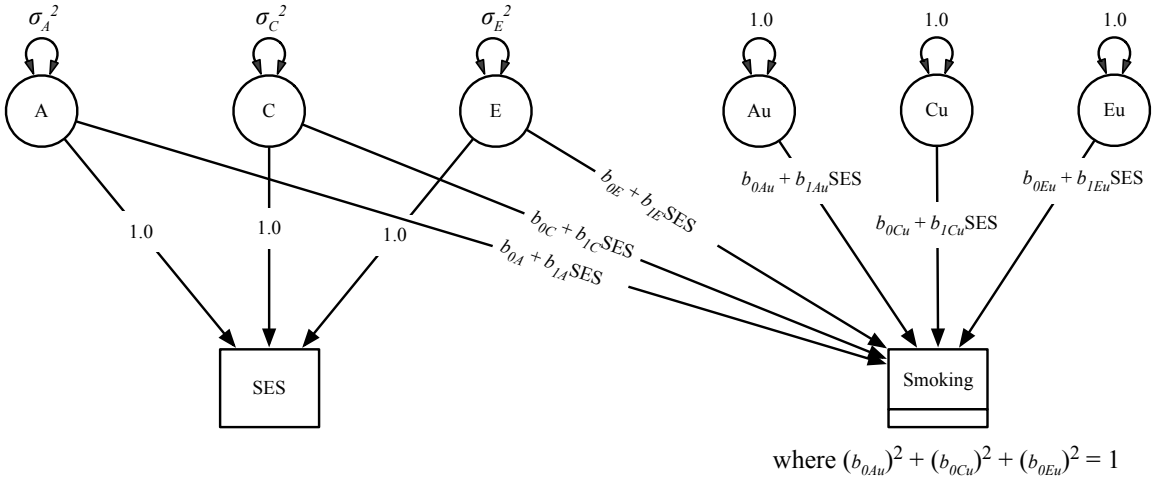


*Figure 9.18*. Path diagram G×SES model fit to smoking in the WSTR (only one twin shown for clarity).

Table 9.4. Parameter estimates and model fit statistics for G×SES models, ADHD diagnosis in the WSTR.

| | Parameter | Phenotypic Model | Quasi-Causal Model | Moderation of Residual Variance | Best-Fitting Model |
|---|---|---|---|---|---|
| **Education** | Main Effect of Education on Smoking | | | | |
| | $b_{0A}$ | **–.180 (–.300, –.083)** | **–.366 (–.786, –.129)** | **–.249 (–.707, –.088)** | **–.366 (–.786, –.129)** |
| | $b_{1A}$ | — | — | — | — |
| | $b_{0C}$ | **–.180 (–.300, –.083)** | –.154 (–.455, .118) | –.108 (–.442, .113) | –.154 (–.455, .118) |
| | $b_{1C}$ | — | — | — | — |
| | $b_{0E}$ | **–.180 (–.300, –.083)** | **–.088 (–.178, –.022)** | **–.060 (–.178, –.011)** | **–.088 (–.178, –.022)** |
| | $b_{1E}$ | — | — | — | — |
| | Effect of Education on Residual ACE Components of Smoking | | | | |
| | $b_{0Au}$ | .447 (–.995, .964) | .667 (–.995, .972) | .765 (–1.000, 1.000) | .667 (–.995, .972) |
| | $b_{1Au}$ | — | — | .042 (–.092, .006) | — |
| | $b_{0Cu}$ | .821 (–.495, .989) | .632 (–.669, .990) | .635 (–1.000, 1.000) | .632 (–.669, .990) |
| | $b_{1Cu}$ | — | — | –.009 (–.100, .095) | — |
| | $b_{0Eu}$ | **.357 (.137, .712)** | **.396 (.130, .745)** | .108 (–.192, .501) | **.396 (.130, .745)** |
| | $b_{1Eu}$ | — | — | .030 (–.002, .095) | — |
| | Model Fit | | | | |
| | *-2LL* | — | 21351.580 | 21346.010 | 21351.580 |
| | *Δ-2LL (Δdf)* | — | — | 5.572 (+3) | — |
| | *p* | — | —† | .134 | — |
| **Household Income** | Main Effect of Income on Smoking | | | | |
| | $b_{0A}$ | **–.081 (–.136, –.058)** | **–.191 (–.460, –.153)** | **–.094 (–.331, –.072)** | **–.191 (–.460, –.153)** |
| | $b_{1A}$ | — | — | — | — |
| | $b_{0C}$ | **–.081 (–.136, –.058)** | –.071 (–.582, .491) | –.020 (–.331, .352) | –.071 (–.582, .491) |
| | $b_{1C}$ | — | — | — | — |
| | $b_{0E}$ | **–.081 (–.136, –.058)** | **–.061 (–.097, –.030)** | **–.027 (–.073, –.012)** | **–.061 (–.097, –.030)** |
| | $b_{1E}$ | — | — | — | — |
| | Effect of Income on Residual ACE Components of Smoking | | | | |
| | $b_{0Au}$ | **.899 (.291, .999)** | .806 (–.995, .996) | .128 (–.866, .997) | .806 (–.995, .996) |
| | $b_{1Au}$ | — | — | –.019 (–.066, .058) | — |
| | $b_{0Cu}$ | .325 (–.999, .906) | .459 (–.998, .998) | .971 (–.999, .999) | .459 (–.998, .998) |
| | $b_{1Cu}$ | — | — | –.019 (–.065, .065) | — |
| | $b_{0Eu}$ | **.294 (.038, .709)** | **.374 (.057, .702)** | **.200 (.026, .557)** | **.374 (.057, .702)** |
| | $b_{1Eu}$ | — | — | .006 (–.010, .033) | — |
| | Model Fit | | | | |
| | *-2LL* | — | 27717.440 | 27710.070 | 27717.440 |
| | *Δ-2LL (Δdf)* | — | — | 7.368 (+3) | — |
| | *p* | — | —† | .061 | — |
| **Area Deprivation** | Main Effect of Area Deprivation on Smoking | | | | |
| | $b_{0C}$ | **–.742 (–1.346, –.013)** | **–2.121 (–3.463, –.979)** | **–.713 (–10.00, –.134)** | **–2.121 (–3.463, –.979)** |
| | $b_{1C}$ | — | — | — | — |
| | $b_{0E}$ | **–.742 (–1.346, –.013)** | –.394 (–1.168, .315) | –.136 (–7.378, .932) | –.394 (–1.168, .315) |
| | $b_{1E}$ | — | — | — | — |
| | Effect of Area Deprivation on Residual ACE Components of Smoking | | | | |
| | $b_{0Au}$ | **.652 (.250, .919)** | .704 (–.123, .998) | .705 (–1.000, 1.000) | .704 (–.123, .998) |
| | $b_{1Au}$ | — | — | –.100 (–.277, 1.691) | — |
| | $b_{0Cu}$ | .634 (–.908, .997) | .475 (–.972, .927) | .653 (-1.000, 1.000) | .475 (–.972, .927) |
| | $b_{1Cu}$ | — | — | –.172 (–2.599, 2.599) | — |
| | $b_{0Eu}$ | **.416 (.259, .691)** | **.529 (.061, .914)** | .286 (-1.000, 1.000) | **.529 (.061, .914)** |
| | $b_{1Eu}$ | — | — | –.092 (–.183, 1.231) | — |
| | Model Fit | | | | |
| | *-2LL* | — | –1136.495 | –1136.876 | –1136.495 |
| | *Δ-2LL (Δdf)* | — | — | .380 (+3) | — |
| | *p* | — | —† | .944 | — |
| **Income Inequality** | Main Effect of Income Inequality on Smoking | | | | |
| | $b_{0A}$ | –.011 (–.031, .002) | –2.874 (–10.00, 10.00) | –1.949 (–10.00, 10.00) | 2.582 (–10.00, 10.00) |
| | $b_{1A}$ | — | — | — | — |
| | $b_{0C}$ | –.011 (–.031, .002) | –.050 (–.110, .145) | –.037 (–.618, 2.777) | .045 (–.259, .141) |
| | $b_{1C}$ | — | — | — | — |
| | $b_{0E}$ | –.011 (–.031, .002) | .010 (–.005, .030) | .035 (–.011, .624) | .013 (–.018, .058) |
| | $b_{1E}$ | — | — | — | — |
| | Effect of Income Inequality on Residual ACE Components of Smoking | | | | |
| | $b_{0Au}$ | .534 (–.393, .939) | **.831 (–.978, .991)** | .899 (–1.000, 1.000) | .985 (–1.000, 1.000) |
| | $b_{1Au}$ | — | — | .049 (–.009, .611) | — |
| | $b_{0Cu}$ | .723 (–.951, .951) | .300 (–.939, .939) | .429 (–1.000, 1.000) | .119 (–1.000, 1.000) |
| | $b_{1Cu}$ | — | — | .006 (–.413, .413) | — |
| | $b_{0Eu}$ | **.439 (.200, .901)** | **.474 (.136, .895)** | .093 (–1.000, 1.000) | .131 (–.141, 1.000) |
| | $b_{1Eu}$ | — | — | **–.029 (–.334, –.006)** | — |
| | Model Fit | | | | |
| | *-2LL* | — | 24960.490 | 24.956.030 | 23542.840 |
| | *Δ-2LL (Δdf)* | — | — | 4.462 (+3) | — |
| | *p* | — | —† | .216 | — |

*Note:* 95% confidence intervals presented within parentheses. Estimates $p < .05$ **bolded**. Baseline model denoted by †.

**Causal Pathways vs. rGE: Main Effects of SES on Smoking (Dissertation Aim 1).** The main effects of SES on smoking status are presented in Table 9.4 under the heading *Main Effect of Moderator on Smoking*. We discuss the main effects results for each moderator below.

**Education.** Education demonstrated a phenotypic association with smoking ($b$ = –0.180, 95% confidence interval = –0.300 to –0.083). Controlling for age and gender, individuals with a graduate or professional degree were 51% less likely to smoke than individuals with a high school degree. The best-fitting model suggested that these associations are partially (but not entirely) mediated by additive genetic factors ($b_{0A}$ = –0.366, 95% confidence interval = –0.786 to –0.129). The quasi-causal association was also statistically significant ($b_{0E}$ = –0.088, 95% confidence interval = –0.178 to –0.022). The shared environmental regression was not statistically distinguishable from zero ($b_{0C}$ = –0.154, 95% confidence interval = –0.455 to 0.118). These results suggest that the protective effect of educational attainment on smoking behavior can be attributed to systematic differences in exposure to socioeconomic factors as well as genetic factors that are common to both phenotypes.

As illustrated in the left panel of Figure 9.19, which shows the magnitude of the ACE regressions of internalizing on educational attainment, the between-family regressions are larger than the within-family regression. These regression lines are re-represented as ACE correlations in the middle panel of Figure 9.19. We also present the proportions of the total phenotypic correlation accounted for by the genetic and nonshared environmental correlations in the right panel of Figure 9.19. Evident in this

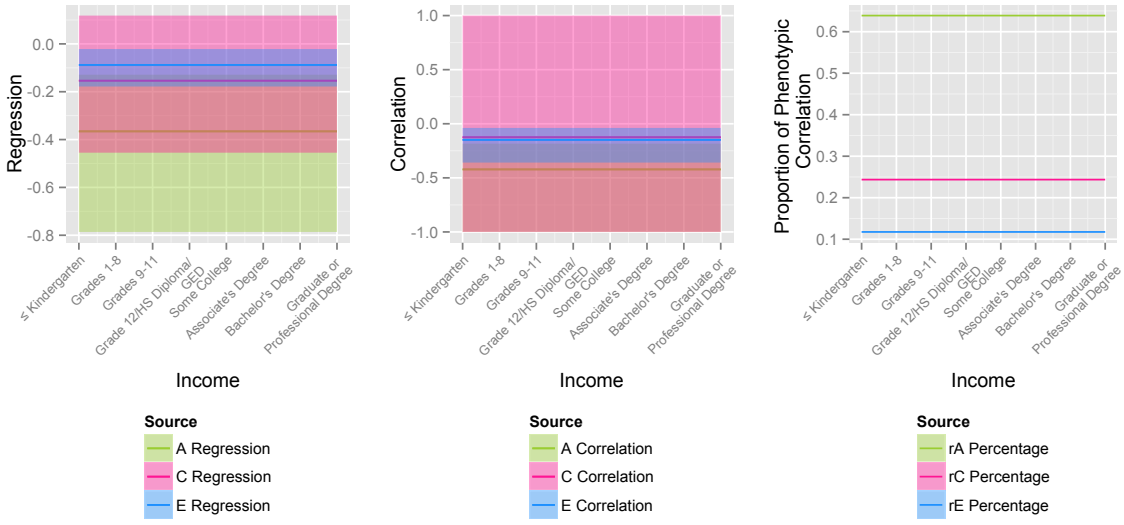


*Figure 9.19*. Main effects of educational attainment on smoking status in the WSTR. The left panel shows the regression of smoking on educational attainment. The same relation is presented in the middle panel as genetic (green), shared environmental (pink), and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic, shared environmental, and nonshared environmental correlations. Asterisks (*) indicate that the magnitude of the regression of smoking status on educational attainment depends on level of education (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). Shaded regions represent 95% confidence intervals around regression lines.

figure is that the nonshared environmental correlation accounts for just 10% of the overall phenotypic correlation, whereas the genetic and shared environmental correlations account for approximately 25% and 65%, respectively.

These phenotypic and within-family effects are demonstrated in Figure 9.20, an illustrative analysis of the main effect of educational attainment on smoking status. We identified twin pairs concordant for lower education (up to Associate's degree; orange), pairs concordant for higher education (Bachelor's degree or higher; navy), and pairs discordant for educational status, and compared the proportion of individuals who currently smoke within each of these groups. Overall, there is a main effect of

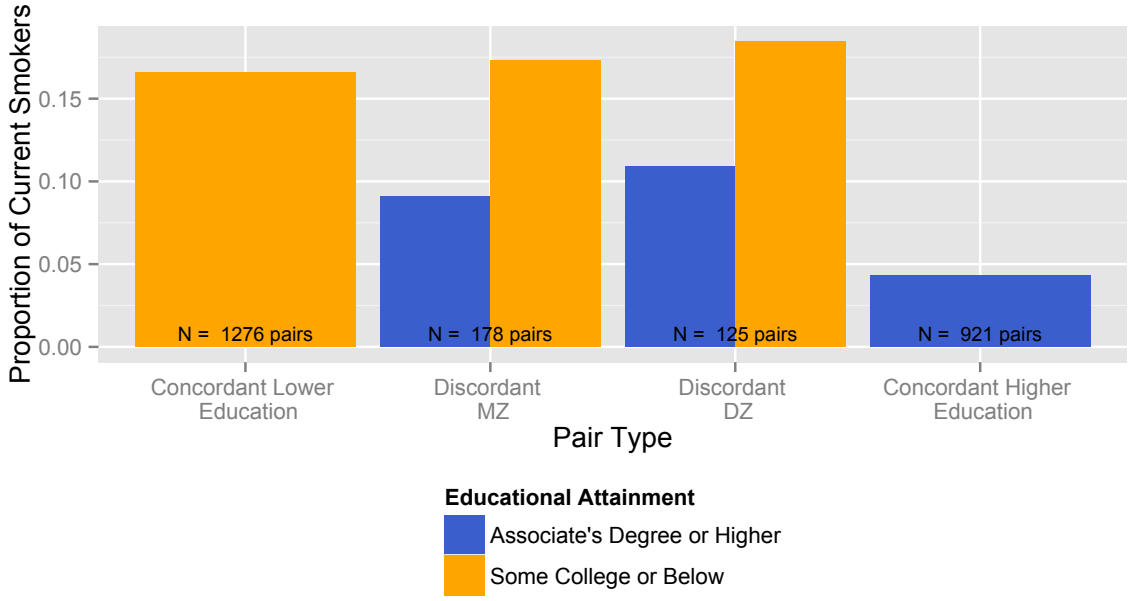


*Figure 9.20*. Illustrative analysis of the main effects of educational attainment on smoking status in the WSTR. The less educated twin (orange) is compared with the more educated twin (navy) in MZ and DZ twin pairs.

educational attainment on smoking status such that higher education is associated with lower likelihood of being a current smoker (this is evident comparing the outer bars in this figure). It is also evident that this relationship exists within pairs of MZ and DZ twins, but consistent with partial between-family mediation, the within-family difference is not as large as that observed at the phenotypic level.

**Income.** The phenotypic regression of the general internalizing factor on income showed a significant negative relationship ($b$ = –0.081, 95% confidence interval = –0.136 to –0.058). That is, controlling for age and gender, individuals at the third quartile of earned income were 33% less likely than their counterparts at the first quartile of earned income to be a current smoker. The best-fitting model showed a statistically significant quasi-causal effect ($b_{0E}$ = –0.061, 95% confidence interval = –0.097 to –0.030). The

common genetic background to income and smoking status was also statistically significant ($b_{0A}$ = –0.191, 95% confidence interval = –0.460 to –0.153), suggesting that genes contributing to earned income may also be the same genes influencing cigarette use. The common shared environmental pathway was not statistically significant ($b_{0C}$ = –0.071, 95% confidence interval = –0.582 to 0.491). These results suggest that, like with income, the relation between earned income and smoking status is partially (but not entirely) explained by underlying genetic factors that are common to both phenotypes. There is also evidence that systematic differences in exposure to socioeconomic factors contribute to an individual's smoking status. Results from the best-fitting model are illustrated in Figure 9.21. Like educational attainment, household income tended to be

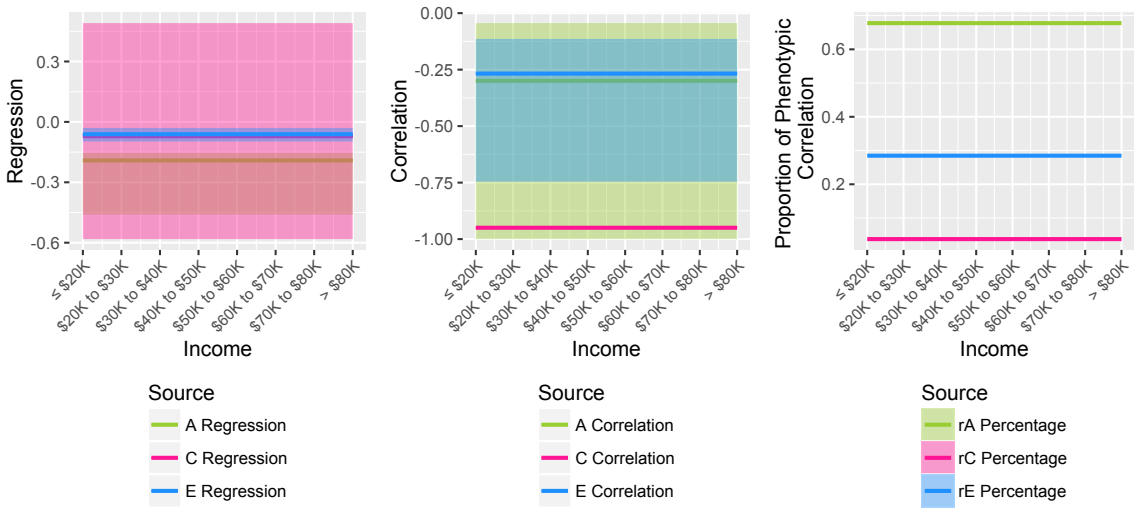


*Figure 9.21*. Main effects of household income on smoking status in the WSTR. The left panel shows the regression of internalizing on smoking. The same relation is presented in the middle panel as genetic (green), shared environmental (pink), and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic, shared environmental, and nonshared environmental correlations. Asterisks (*) indicate that the magnitude of the regression of smoking status on household income depends on level of income (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). Shaded regions represent 95% confidence intervals around regression lines.

correlated with smoking status primarily via an additive genetic environmental pathway common to both phenotypes. This pathway accounted for approximately 70% of the phenotypic correlation, and the nonshared environmental pathway accounted for approximately 30%.

These phenotypic and within-family effects are illustrated in Figure 9.22. Consistent with a causal effect of household income on smoking status, the overall effect of household income on smoking exists within pairs of MZ and DZ twins, although is somewhat diminished. The difference in smoking status within DZ twins discordant for household income exceeds that of discordant MZ twins, consistent with partial genetic mediation of this association.

**Area Deprivation.** At the phenotypic level, the ADI was statistically significantly associated with the general internalizing factor ($b$ = –0.742, 95% confidence

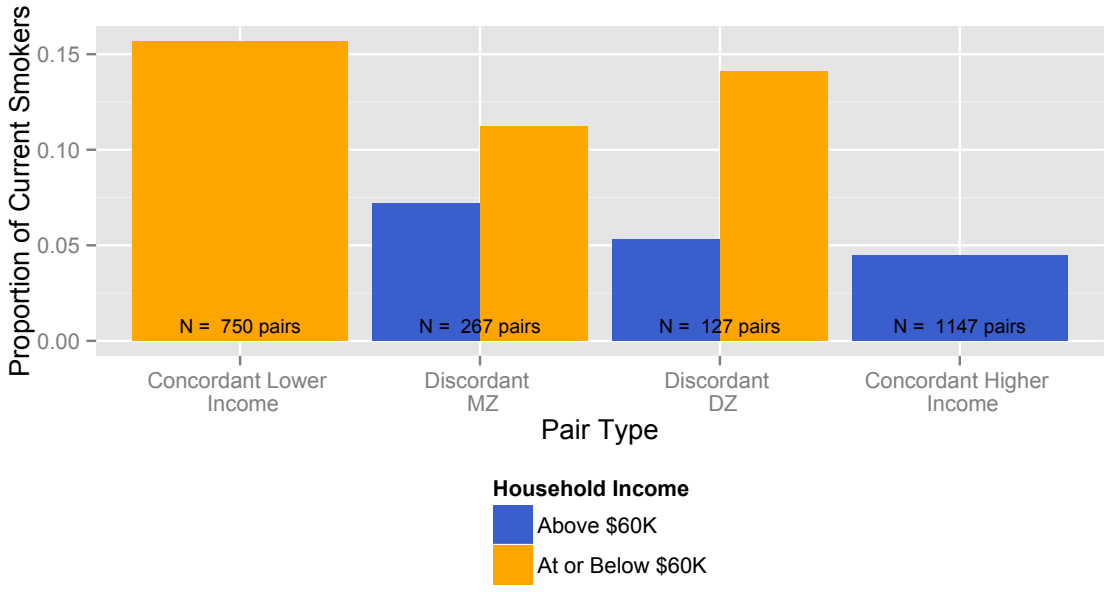


*Figure 9.22*. Illustrative analysis of the main effects of household income on smoking status in the WSTR. The twin earning less income (orange) is compared with the twin earning more income (navy) in MZ and DZ twin pairs.

interval = –1.346 to –0.013). Individuals at the third quartile of the ADI were 14% less likely to be a current smoker than individuals at the first quartile of the ADI. General internalizing was not quasi-causally associated with the ADI ($b_{0E}$ = –0.394, 95% confidence interval = –1.168 to 0.315) and instead was correlated via a common shared environmental pathway ($b_{0C}$ = –2.121, 95% confidence interval = –3.463 to –0.979). These results are largely consistent between-family confounding of the SES-smoking association observed using individual-level SES measures. Model results are illustrated in Figure 9.23, where it is evident that the magnitude of the between-family correlation exceeds that of the within-family correlation. Likewise, the between-family pathway from neighborhood-level SES to the smoking status factor accounted for nearly three

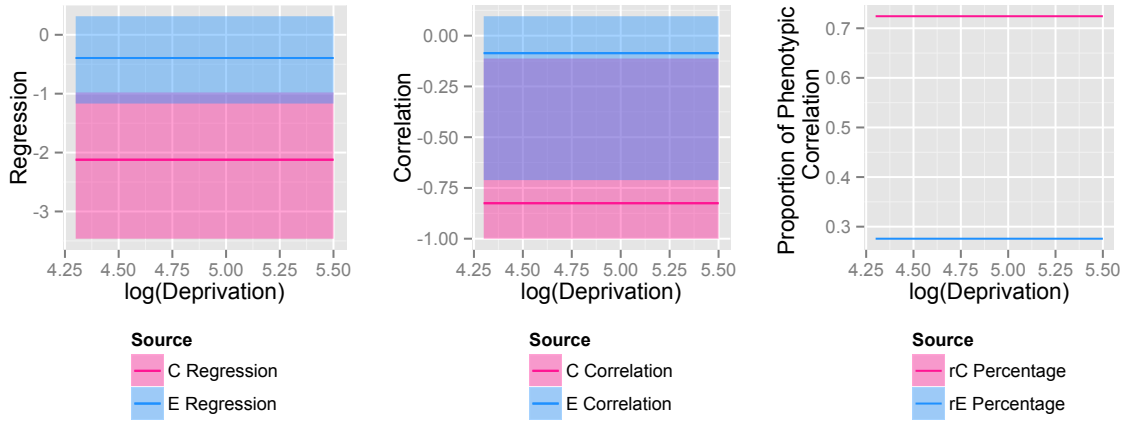


*Figure 9.23*. Main effects of area deprivation on smoking status in the WSTR. The left panel shows the regression of smoking on neighborhood-level SES. The same relation is presented in the middle panel as shared environmental (pink) and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the shared and nonshared environmental correlations. Asterisks (*) indicate that the magnitude of the regression of smoking status on neighborhood-level SES depends on level of SES (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). Shaded regions represent 95% confidence intervals around regression lines.

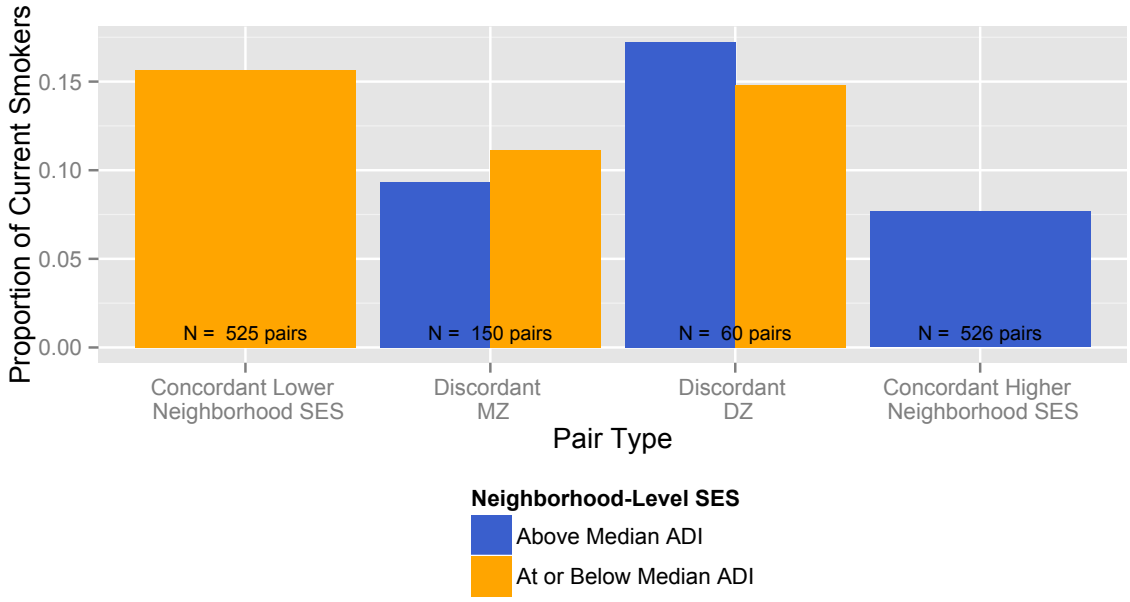


*Figure 9.24*. Illustrative analysis of the main effects of neighborhood-level socioeconomic status on smoking status in the WSTR. The twin residing in the less socioeconomically advantaged neighborhood (orange) is compared with the twin living in the more affluent neighborhood (navy) in MZ and DZ twin pairs.

times as much of the total phenotypic correlation than did the within-family pathway. Illustrative analyses (see Figure 9.24) also demonstrate the lack of effect of neighborhood-level SES within twin pairs.

**Income Inequality.** Income inequality showed no evidence of influence on smoking status ($b$ = –0.011, 95% confidence interval = –0.031 to 0.002).

**G×E Interaction: Effect of SES on Genetic and Environmental Etiology of Smoking (Dissertation Aim 2).** The moderating effects of SES on variance in smoking status are presented in Table 9.4 under the heading *Effect of SES on Residual ACE Components of Smoking*. None of the indicators of socioeconomic status were related to residual variance in smoking status.

**Brief Summary.** Both compositional and contextual measures of socioeconomic

status were found to be protective against being a current smoker. Between-family (*i.e.*, non-causal) factors tended to explain the majority of this association, although compositional SES tended to also show evidence of causal influences on smoking status. Residual variance in smoking status was influenced by neither compositional nor contextual measures of SES.

**Physical Activity and Sedentary Behavior**

***Physical Activity—Add Health.*** Physical activity in the Add Health sample was measured using three items tapping the frequency of participation in various physical activities (see Chapter 3). We fit the model presented in Figure 9.25 to the data separately for each socioeconomic status indicator, partialling for the linear effects of age and gender on level and variance in physical activity. We used likelihood ratio tests to determine the best fitting model. Biometric decomposition of both the SES indicators and the physical activity factor are presented in Table 4.1, and parameter estimates for

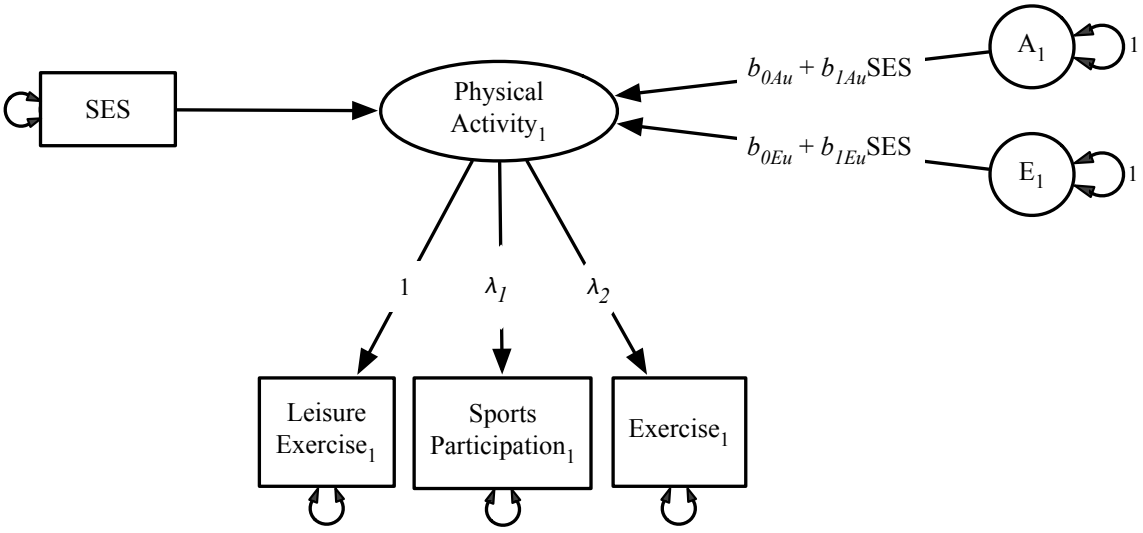


*Figure 9.25*. Path diagram G×SES model fit to physical activity in the Add Health sample (only one twin shown for clarity). The residual variances for the physical activity items were permitted to correlate across twins, and were estimated freely according to zygosity.

Table 9.5. Parameter estimates and model fit statistics for G×SES models, physical activity in the Add Health sample.

| | Parameter | Phenotypic Model (No Moderation) | Moderation of Residual Variance | Best-Fitting Model |
|---|---|---|---|---|
| **Parental Education** | Main Effect of Parental Education on Physical Activity | | | |
| | $b_1$ | –.005 (.022) | –.006 (.022) | –.005 (.022) |
| | Effect of Education on Residual ACE Components of Physical Activity | | | |
| | $b_{0Au}$ | ***2.526 (.439)*** | ***2.452 (.467)*** | ***2.526 (.439)*** |
| | $b_{1Au}$ | — | .018 (.028) | — |
| | $b_{0Eu}$ | ***2.128 (.371)*** | ***2.294 (.367)*** | ***2.128 (.371)*** |
| | $b_{1Eu}$ | — | **–.040 (.019)** | — |
| | Model Fit | | | |
| | *-2LL* | 10897.282 | 10892.854 | 10897.282 |
| | *Δ-2LL (Δdf)* | — | 4.428 (+2) | — |
| | *p* | —† | .109 | — |
| **Family Household Income** | Main Effect of Family Income on Physical Activity | | | |
| | $b_1$ | .027 (.027) | .014 (.011) | .027 (.027) |
| | Effect of Family Income on Residual ACE Components of Physical Activity | | | |
| | $b_{0Au}$ | ***2.749 (.455)*** | .208 (.260) | ***2.749 (.455)*** |
| | $b_{1Au}$ | — | .023 (.015) | — |
| | $b_{0Eu}$ | ***1.832 (.398)*** | .146 (.196) | ***1.832 (.398)*** |
| | $b_{1Eu}$ | — | –.003 (.009) | — |
| | Model Fit | | | |
| | *-2LL* | 9672.940 | 9668.302 | 9672.940 |
| | *Δ-2LL (Δdf)* | — | 4.638 (+2) | — |
| | *p* | —† | .098 | — |
| **Neighborhood Socioeconomic Advantage** | Main Effect of Neighborhood SES on Physical Activity | | | |
| | $b_1$ | –.027 (.035) | –.028 (.035) | –.030 (.034) |
| | Effect of Neighborhood SES on Residual ACE Components of Physical Activity | | | |
| | $b_{0Au}$ | ***3.050 (.395)*** | ***3.129 (.415)*** | ***3.078 (.376)*** |
| | $b_{1Au}$ | — | –.018 (.042) | — |
| | $b_{0Eu}$ | .591 (.407) | .624 (.396) | .600 (.392) |
| | $b_{1Eu}$ | — | **–.104 (.044)** | **–.106 (.045)** |
| | Model Fit | | | |
| | *-2LL* | 9116.276 | 9109.322 | 9109.514 |
| | *Δ-2LL (Δdf)* | — | 6.954 (+2) | .192 (–1) |
| | *p* | — | .031† | .661 |

*Note:* Standard errors presented within parentheses. Estimates p < .05 **bolded**. Estimates p < .01 *italicized*. Baseline model denoted by †.

each of these models are presented in Table 9.5. Parental household income was log-transformed to correct for positive skew. The latent physical activity factor showed no evidence of influence from shared environmental factors; therefore, we did not include C in any of the models fit to the data.

**Main Effects of SES on Physical Activity (Dissertation Aim 1).** The main effects of SES on physical activity are presented in Table 9.5 under the heading *Main Effect of Moderator on Physical Activity*. Socioeconomic status was not related to mean

levels of physical activity (parental education: $b$ = –0.005, $p$ = 0.827; parental household income: $b$ = 0.027, $p$ = 0.323; neighborhood-level SES: $b$ = –0.027, $p$ = 0.448).

**G×E Interaction: Effect of SES on Genetic and Environmental Etiology of Physical Activity.** The moderating effects of SES on variance in physical activity are presented in Table 9.5 under the heading *Effect of SES on Residual ACE Components of Physical Activity.*

**Parental Education.** The best-fitting model suggested that residual variance in physical activity does not vary as a function of parental educational attainment.

**Family Income.** The best-fitting model suggested that residual variance in physical activity was not related to family household income.

**Neighborhood Socioeconomic Advantage.** The best-fitting model suggested that residual variance in physical activity decreased as a function of increasing neighborhood-level SES, an effect driven by decreasing nonshared environmental variance ($b_{1Eu}$ = –0.106, $p$ = 0.018). These model results are illustrated in Figure 9.26, which shows residual AE variance components of physical activity as a function of neighborhood-level SES, represented as both regression lines and stacked variances. This figure demonstrates how residual phenotypic and nonshared environmental variance in physical activity decrease with increasing neighborhood SES. It seems that low-SES environments potentiate environmental risk for engaging in low physical activity levels.

These results are further illustrated in Figure 9.27, which shows total variance in physical activity decreasing as a function of increasing neighborhood SES. Consistent with decreasing nonshared environmental variance, the slope of the MZ regression line (left panel of Figure 9.27) is negative. The violin plots (right panel of Figure 9.27) show

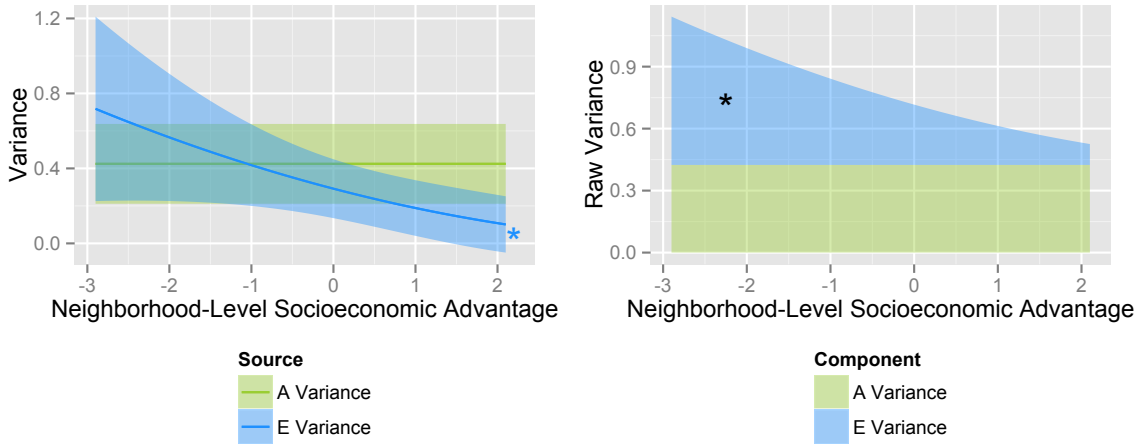


*Figure 9.26.* Gene-by-environment interaction between physical activity and neighborhood-level socioeconomic advantage in the Add Health sample. The left panel shows AE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in physical activity increases as a function of increasing neighborhood-level SES. The asterisks (*) indicate that variance in physical activity depends on level of neighborhood-level SES.

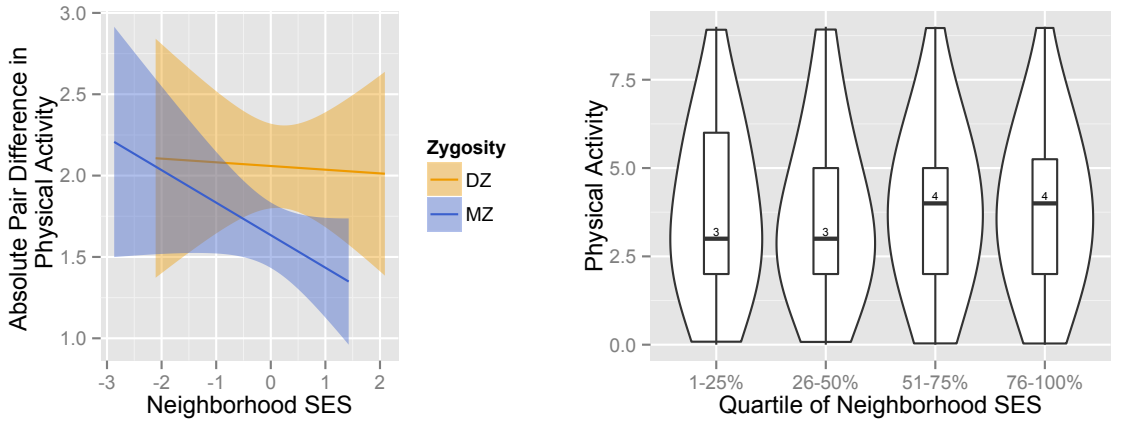


*Figure 9.27.* Illustrative analysis of the effects of neighborhood socioeconomic advantage on variance in physical activity in the Add Health sample. The left panel shows absolute pair differences in physical activity as a function of neighborhood SES. The distance between the MZ and DZ lines represents the additive genetic variance in physical activity. The location of the MZ represents nonshared environmental variance in physical activity. The right panel shows box plots overlaid with violin plots, physical activity as a function of quartile of neighborhood SES level.

that this constrained variance effect appears to be the result of fewer observations in the lower tail of physical activity when neighborhood-level SES is high.

**Brief Summary.** Socioeconomic status was not statistically significantly related to mean levels of physical activity. Higher neighborhood SES predicted decreased nonshared environmental variance in physical activity, however.

***Sedentary Behavior—Add Health.*** Sedentary behavior in the Add Health sample was measured using three items tapping the amount of time spent participating in various activities requiring no physical activity (*e.g.*, television watching; see Chapter 3). We fit the model presented in Figure 9.28 to the data separately for each socioeconomic status indicator, partialling for the linear effects of age and gender on level and variance in sedentary behavior. We used likelihood ratio tests to determine the best fitting model. Biometric decomposition of both the SES indicators and the physical activity factor are presented in Table 4.1, and parameter estimates for each of these models are presented in

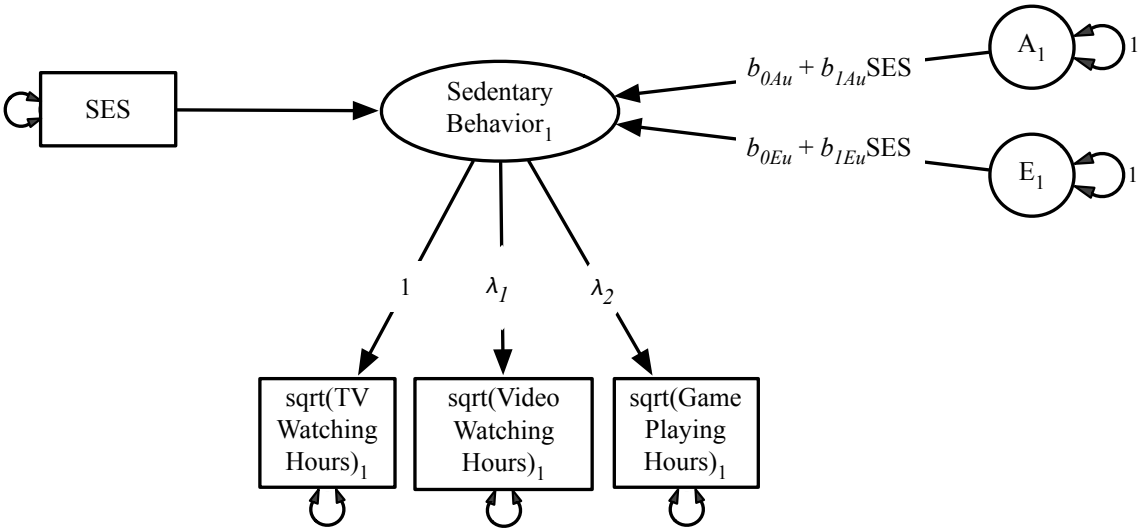


*Figure 9.28*. Path diagram G×SES model fit to sedentary behavior in the Add Health sample (only one twin shown for clarity). The residual variances for the sedentary behavior items were permitted to correlate across twins, and were estimated freely according to zygosity.

*Table 9.6*. Parameter estimates and model fit statistics for G×SES models, sedentary behavior in the Add Health sample.

| | Parameter | Phenotypic Model (No Moderation) | Moderation of Residual Variance | Best-Fitting Model |
|---|---|---|---|---|
| **Parental Education** | Main Effect of Parental Education on Sedentary Behavior | | | |
| | $b_1$ | .009 (.012) | .008 (.012) | .009 (.012) |
| | Effect of Education on Residual ACE Components of Sedentary Behavior | | | |
| | $b_{0Au}$ | ***.870 (.223)*** | ***.732 (.216)*** | ***.870 (.223)*** |
| | $b_{1Au}$ | — | .017 (.012) | — |
| | $b_{0Eu}$ | .368 (.202) | **.527 (.208)** | .368 (.202) |
| | $b_{1Eu}$ | — | –.022 (.013) | — |
| | Model Fit | | | |
| | *-2LL* | 12271.636 | 12267.960 | 12271.636 |
| | *Δ-2LL (Δdf)* | — | 3.676 (+2) | — |
| | *p* | —† | .159 | — |
| **Family Household Income** | Main Effect of Family Income on Sedentary Behavior | | | |
| | $b_1$ | .005 (.014) | .000 (.018) | .005 (.014) |
| | Effect of Family Income on Residual ACE Components of Sedentary Behavior | | | |
| | $b_{0Au}$ | ***1.055 (.301)*** | ***.995 (.298)*** | ***1.055 (.301)*** |
| | $b_{1Au}$ | — | –.005 (.027) | — |
| | $b_{0Eu}$ | *.951 (.305)* | ***.929 (.279)*** | *.951 (.305)* |
| | $b_{1Eu}$ | — | –.030 (.031) | — |
| | Model Fit | | | |
| | *-2LL* | 10886.692 | 10884.352 | 10886.692 |
| | *Δ-2LL (Δdf)* | — | 2.340 (+2) | — |
| | *p* | —† | .310 | — |
| **Neighborhood Socioeconomic Advantage** | Main Effect of Neighborhood SES on Sedentary Behavior | | | |
| | $b_1$ | –.070 (.060) | –.028 (.028) | –.028 (.027) |
| | Effect of Neighborhood SES on Residual ACE Components of Sedentary Behavior | | | |
| | $b_{0Au}$ | 1.278 (.674) | ***.649 (.240)*** | ***.641 (.235)*** |
| | $b_{1Au}$ | — | –.051 (.027) | **–.051 (.025)** |
| | $b_{0Eu}$ | .170 (.484) | ***.784 (.250)*** | ***.809 (.256)*** |
| | $b_{1Eu}$ | — | –.043 (.025) | — |
| | Model Fit | | | |
| | *-2LL* | 10300.718 | 10290.524 | 10293.794 |
| | *Δ-2LL (Δdf)* | — | 10.896 (+2) | 3.270(–1) |
| | *p* | — | .004† | .071 |

*Note:* Standard errors presented within parentheses. Estimates p < .05 **bolded**. Estimates p < .01 *italicized*. Baseline model denoted by †.

Table 9.6. Parental household income and number of hours spent in each sedentary behavior were log-transformed to correct for positive skew. The latent sedentary behavior factor showed no evidence of influence from shared environmental factors; therefore, we did not include C in any of the models fit to the data.

**Main Effects of SES on Sedentary Behavior (Dissertation Aim 1).** The main effects of SES on sedentary behavior are presented in Table 9.6 under the heading *Main*

*Effect of Moderator on Sedentary Behavior*. None of the socioeconomic indicators showed associations with this phenotype (parental education: $b$ = 0.007, $p$ = 0.572; family household income: $b$ = 0.005, $p$ = 0.732; neighborhood-level SES: $b$ = –0.070, $p$ = 0.249).

**G×E Interaction: Effect of SES on Genetic and Environmental Etiology of Sedentary Behavior (Dissertation Aim 2).** The moderating effects of SES on variance in sedentary behavior are presented in Table 9.6 under the heading *Effect of SES on Residual ACE Components of Sedentary Behavior.*

**Parental Education.** The best-fitting model suggested that residual variance in sedentary behavior does not vary as a function of parental educational attainment.

**Family Income.** The best-fitting model suggested that residual variance in sedentary behavior was not related to family household income.

**Area Deprivation.** The best-fitting model suggested that residual variance in sedentary behavior decreased as a function of increasing neighborhood-level SES. This effect was driven by decreasing additive genetic variance ($b_{1Au}$ = –0.051, $p$ = 0.041). These model results are illustrated in Figure 9.29, which shows residual AE variance components of sedentary behavior as a function of neighborhood-level SES, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure clearly demonstrates how residual phenotypic and additive genetic variance in sedentary behavior decreases with increasing neighborhood socioeconomic advantage. It seems that high-SES environments mitigate genetic risk for engaging in sedentary behavior.

These results are further illustrated in Figure 9.30. Similar to our results for

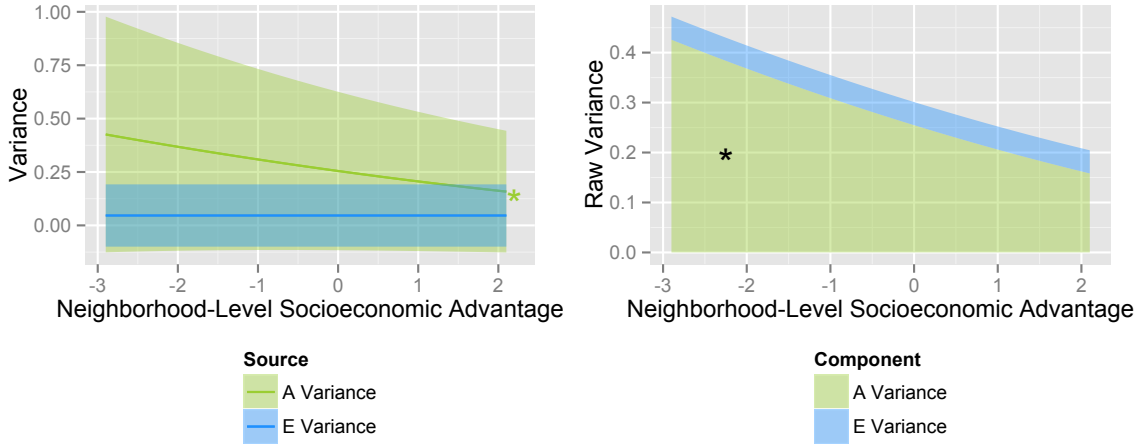


*Figure 9.29*. Gene-by-environment interaction between sedentary behavior and neighborhood-level socioeconomic advantage in the Add Health sample. The left panel shows AE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in sedentary behavior increases as a function of increasing neighborhood-level SES. The asterisks (*) indicate that variance in sedentary behavior depends on level of neighborhood-level SES.

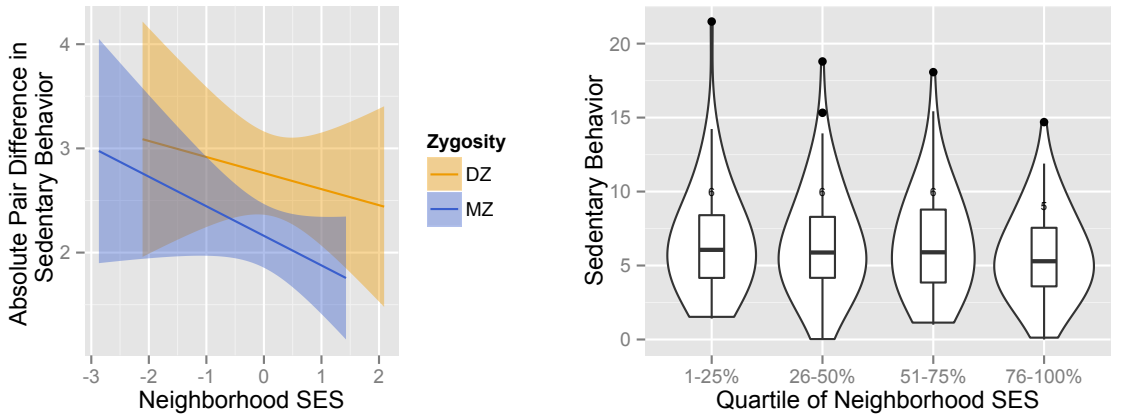


*Figure 9.30*. Illustrative analysis of the effects of neighborhood socioeconomic advantage on variance in sedentary behavior in the Add Health sample. The left panel shows absolute pair differences in sedentary behavior as a function of neighborhood SES. The distance between the MZ and DZ lines represents the additive genetic variance in sedentary behavior. The location of the MZ represents nonshared environmental variance in sedentary behavior. The right panel shows box plots overlaid with violin plots, sedentary behavior as a function of quartile of neighborhood SES level.

physical activity, we observed that total variance in sedentary behavior decreased as a function of increasing neighborhood SES. Consistent with decreasing additive genetic variance, the slope of the MZ regression line diverges from the DZ regression line as neighborhood SES increases (left panel of Figure 9.30). The violin plots (right panel of Figure 9.30) show that this constrained variance effect appears to be the result of fewer observations in the upper tail of sedentary behavior when neighborhood SES is high.

**Brief Summary.** Socioeconomic status was not statistically significantly related to mean levels of sedentary behavior. Neighborhood-level socioeconomic advantage was associated with decreased residual additive genetic variance in sedentary behavior.

***Physical Activity—WSTR.*** Physical activity in the WSTR was operationalized as hours per week of moderate-to-vigorous physical activity (MVPA). We fit the model presented in Figure 9.31 to the data separately for each socioeconomic status indicator, partialling for the linear effects of age and gender on level and variance in moderate-to-vigorous physical activity. We used likelihood ratio tests to determine the best fitting model. We first established a baseline model by conducting omnibus tests for the

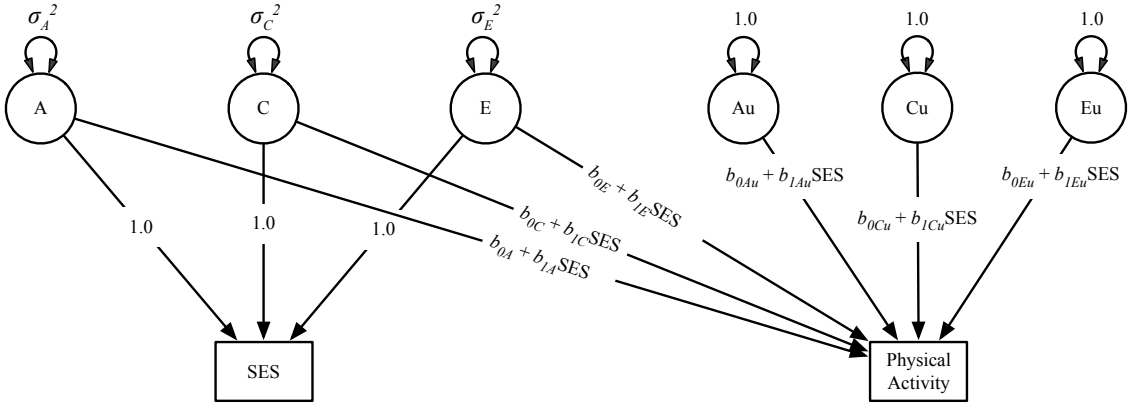


*Figure 9.31*. Path diagram G×SES model fit to physical activity in the WSTR (only one twin shown for clarity). In the fully saturated model, the main effects of SES on physical activity and the ACE variances of physical activity vary as a function of SES.

Table 9.7. Parameter estimates and model fit statistics for G×SES models, moderate-to-vigorous physical activity in the WSTR.

| | Parameter | Phenotypic Model | Quasi-Causal Model | Moderation of Residual Variance | Moderation of Main Effects | Best-Fitting Model |
|---|---|---|---|---|---|---|
| **Education** | Main Effect of Education on MVPA | | | | | |
| | $b_{0A}$ | ***.134 (.019)*** | ***.242 (.048)*** | ***.241 (.048)*** | .310 (.199) | ***.242 (.048)*** |
| | $b_{1A}$ | — | — | — | –.012 (.032) | — |
| | $b_{0E}$ | ***.134 (.019)*** | .046 (.035) | .045 (.035) | .066 (.184) | .046 (.035) |
| | $b_{1E}$ | — | — | — | –.003 (.030) | — |
| | Effect of Education on Residual ACE Components of MVPA | | | | | |
| | $b_{0Au}$ | ***.913 (.110)*** | ***.918 (.112)*** | ***1.055 (.177)*** | ***1.044 (.180)*** | ***.927 (.112)*** |
| | $b_{1Au}$ | — | — | –.023 (.024) | –.021 (.025) | — |
| | $b_{0Eu}$ | ***1.133 (.068)*** | ***1.122 (.068)*** | ***1.236 (.106)*** | ***1.274 (.106)*** | ***1.288 (.091)*** |
| | $b_{1Eu}$ | — | — | –.020 (.015) | –.020 (.015) | ***–.030 (.011)*** |
| | Model Fit | | | | | |
| | *-2LL* | — | 39548.856 | 39540.166 | 39539.910 | 39541.026 |
| | *Δ-2LL (Δdf)* | — | — | 8.690 (+2) | .256 (+2) | .860 (–1) |
| | *p* | — | — | .013† | .880 | .354 |
| **Household Income** | Main Effect of Income on MVPA | | | | | |
| | $b_{0A}$ | ***.043 (.009)*** | ***.147 (.035)*** | ***.147 (.035)*** | | ***.147 (.035)*** |
| | $b_{1A}$ | — | — | — | | — |
| | $b_{0E}$ | ***.043 (.009)*** | –.012 (.015) | –.012 (.015) | | –.012 (.015) |
| | $b_{1E}$ | — | — | — | | — |
| | Effect of Income on Residual ACE Components of MVPA | | | | | |
| | $b_{0Au}$ | ***.929 (.112)*** | ***.925 (.115)*** | ***.906 (.127)*** | | ***.925 (.115)*** |
| | $b_{1Au}$ | — | — | –.007 (.013) | | — |
| | $b_{0Eu}$ | ***1.128 (.069)*** | ***1.118 (.069)*** | ***1.177 (.075)*** | | ***1.118 (.069)*** |
| | $b_{1Eu}$ | — | — | **–.017 (.008)** | | — |
| | Model Fit | | | | | |
| | *-2LL* | — | 44938.844 | 44932.962 | | 44938.844 |
| | *Δ-2LL (Δdf)* | — | — | 5.882 (+2) | | — |
| | *p* | — | —† | .053 | | — |
| **Area Deprivation** | Main Effect of Area Deprivation on MVPA | | | | | |
| | $b_{0E}$ | **.645 (.262)** | | **.647 (.252)** | 1.925 (4.701) | **.637 (.251)** |
| | $b_{1E}$ | — | | — | –.262 (.963) | — |
| | Effect of Area Deprivation on Residual ACE Components of MVPA | | | | | |
| | $b_{0Au}$ | ***.956 (.143)*** | | 2.202 (1.386) | ***2.159 (1.392)*** | ***.969 (.143)*** |
| | $b_{1Au}$ | — | | ***–.263 (.295)*** | –.255 (.296) | — |
| | $b_{0Eu}$ | ***1.123 (.089)*** | | ***2.260 (.861)*** | ***2.300 (.874)*** | ***2.808 (.618)*** |
| | $b_{1Eu}$ | — | | ***–.243 (.184)*** | –.251 (.186) | ***–.361 (.131)*** |
| | Model Fit | | | | | |
| | *-2LL* | 8138.828 | | 8130.898 | 8130.824 | 8131.704 |
| | *Δ-2LL (Δdf)* | — | | 7.930 (+2) | .074 (+2) | .806 (–1) |
| | *p* | — | | .019† | .964 | .369 |
| **Income Inequality** | Main Effect of Income Inequality on MVPA | | | | | |
| | $b_{0A}$ | **.190 (.092)** | 1.382 (1.366) | 1.396 (1.379) | | 1.382 (1.366) |
| | $b_{1A}$ | — | — | — | | — |
| | $b_{0E}$ | **.190 (.092)** | .077 (.122) | .074 (.122) | | .077 (.122) |
| | $b_{1E}$ | — | — | — | | — |
| | Effect of Income Inequality on Residual ACE Components of MVPA | | | | | |
| | $b_{0Au}$ | ***.914 (.121)*** | ***.905 (.123)*** | .280 (.561) | | ***.905 (.123)*** |
| | $b_{1Au}$ | — | — | .142 (.124) | | — |
| | $b_{0Eu}$ | ***1.127 (.075)*** | ***1.122 (.075)*** | ***1.397 (.359)*** | | ***1.122 (.075)*** |
| | $b_{1Eu}$ | — | — | –.063 (.081) | | — |
| | Model Fit | | | | | |
| | *-2LL* | — | 18794.934 | 18793.622 | | 18794.934 |
| | *Δ-2LL (Δdf)* | — | — | 1.312 (+2) | | — |
| | *p* | — | —† | .519 | | — |

*Note:* Standard errors presented within parentheses. Estimates $p < .05$ **bolded**. Estimates $p < .01$ *italicized*. Baseline model denoted by †.

moderation by SES of both the residual variance in MVPA and the main effects of SES on MVPA. We then systematically eliminated parameters from the baseline model to evaluate statistical significance of moderation parameters. Biometric decomposition of both the SES indicators and MVPA are presented in Table 4.2, and parameter estimates

and model fit statistics for the baseline and best-fitting models presented in Table 9.7. The Area Deprivation Index was log-transformed to correct for positive skew. The Gini Index was scaled by a factor of 10, and MVPA was converted from minutes to hours, to facilitate model convergence. These transformed values were used in all analyses. We did not include A for the ADI since there was no evidence of genetic influences on area deprivation in our univariate model. Likewise, we did not include C for MVPA since there was no evidence of shared environmental influences on physical activity in our univariate model.

**Causal Pathways vs. rGE: Main Effects of SES on Physical Activity (Dissertation Aim 1).** The main effects of SES on body mass index are presented in Table 9.7 under the heading *Main Effect of Moderator on MVPA*. We discuss the main effects results for each moderator below.

**Education.** Education demonstrated a phenotypic association with MVPA ($b$ = 0.134, $p$ < 0.001). Controlling for age and gender, individuals with a graduate or professional degree engaged in moderate-to-vigorous physical activity for approximately 32 minutes (an equivalent of 0.37 standard deviations) longer than individuals with just a high school degree. The best-fitting model suggested that this association is mediated by a common genetic pathway ($b_{0A}$ = 0.242, $p$ < 0.001); the quasi-causal pathway was not statistically significant ($b_{0E}$ = 0.046, $p$ = 0.193). As illustrated in the left panel of Figure 9.32, which shows the magnitude of the genetic and nonshared environmental regressions of MVPA on educational attainment, the genetic pathway is substantially larger than the nonshared environmental pathway. That is, common genetic factors are the most important contributors to the SES–physical activity association; systematic differences in

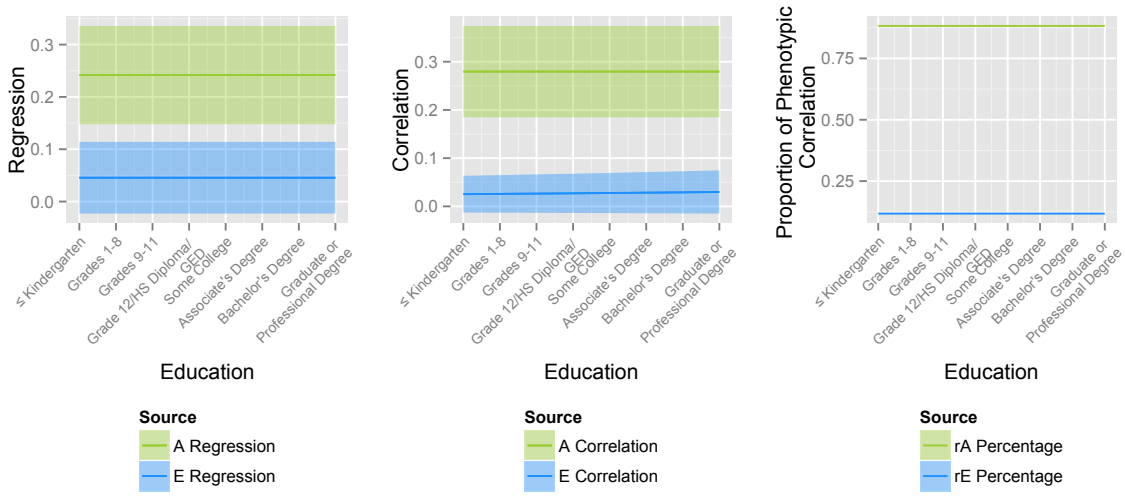


*Figure 9.32*. Main effects of educational attainment on moderate-to-vigorous physical activity in the WSTR. The left panel shows the regression of MVPA on educational attainment. The same relation is presented in the middle panel as genetic (green) and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic and nonshared environmental correlations. The asterisks (*) indicate that the magnitude of the regression of MVPA on educational attainment depends on level of education (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). The shaded regions represent 95% confidence intervals around the regression lines.

educational attainment do not appear to influence MVPA. These regression lines are re-represented as genetic and nonshared environmental correlations (middle panel of Figure 9.32). We also present the proportions of the total phenotypic correlation accounted for by the genetic and nonshared environmental correlations (right panel of Figure 9.32); evident in this figure is that the genetic correlation accounts for approximately seven times as much of the total phenotypic correlation than is accounted for by *r*E.

To demonstrate what these effects look like within and between twins, we conducted an illustrative analysis of the main effect of educational attainment on MVPA, which we present in Figure 9.33. We identified twin pairs concordant for lower

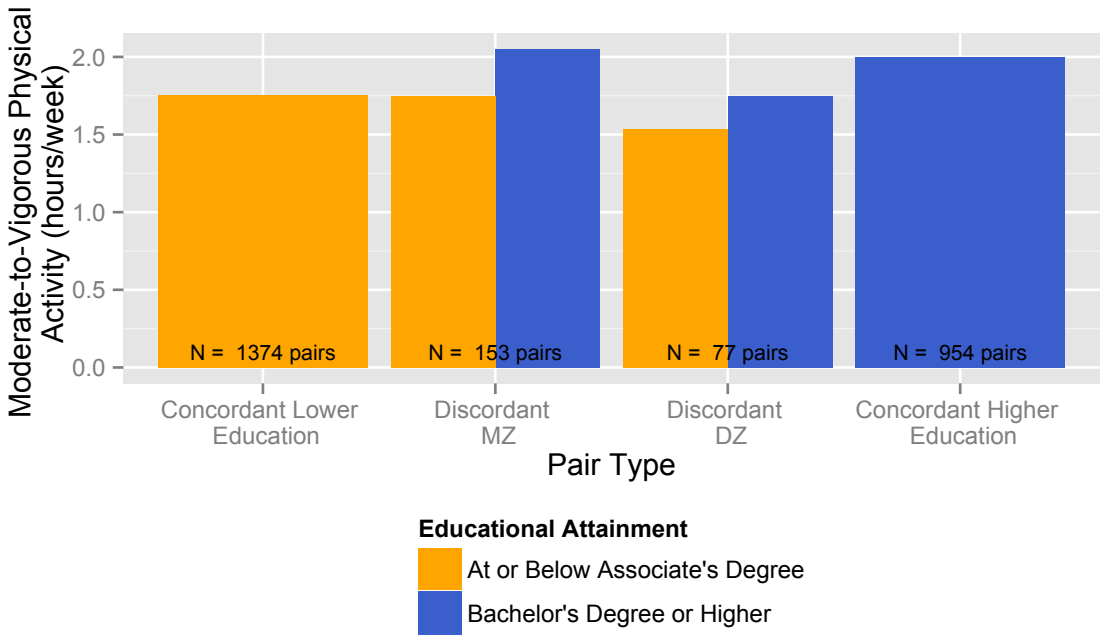


Figure 9.33. Illustrative analysis of the main effects of educational attainment on moderate-to-vigorous physical activity in the WSTR. The twin with less education (orange) is compared with the twin with more education (navy) in MZ and DZ twin pairs.

education (up to high school diploma; orange), pairs concordant for higher education (any post-high school education; navy), and pairs discordant for educational status, and compared the mean MVPA of each of these groups. Overall, there is a main effect of educational attainment on MVPA such that higher education is associated with greater physical activity levels (this is evident comparing the outer bars in this figure). Examining the relation at the within-pair level, however, shows that it is substantially attenuated within pairs of MZ twins (and less so within pairs of DZ twins), consistent with genetic mediation of this association.

**Income.** The phenotypic regression of self-rated health on household income showed a significant positive relationship ($b = 0.043$, $p < 0.001$). Controlling for age and gender, individuals at the third quartile of earned income engaged in moderate-to-

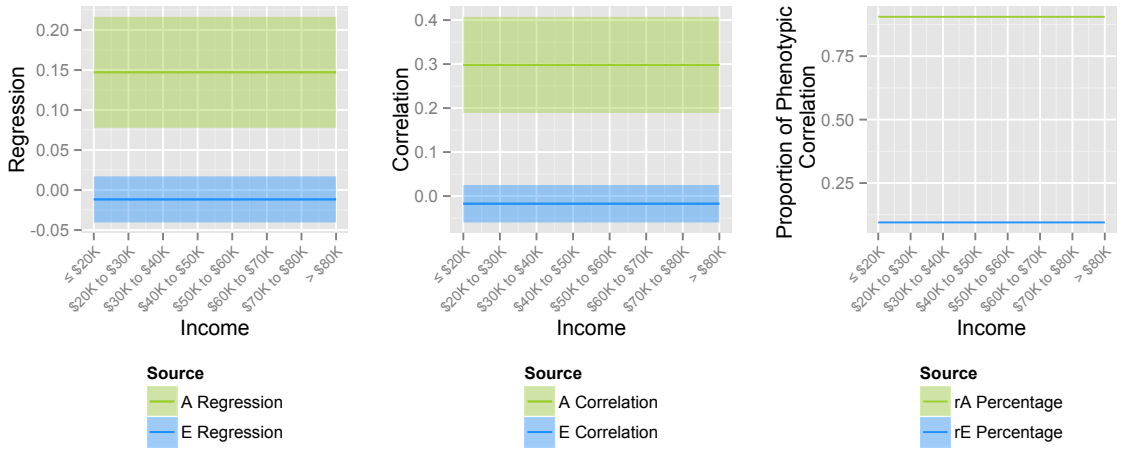


*Figure 9.34*. Main effects of household income on moderate-to-vigorous physical activity in the WSTR. The left panel shows the regression of MVPA on household income. The same relation is presented in the middle panel as genetic (green) and nonshared environmental (blue) correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic and nonshared environmental correlations. The asterisks (*) indicate that the magnitude of the regression of MVPA on household income depends on income level (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). The shaded regions represent 95% confidence intervals around the regression lines.

vigorous physical activity for approximately 13 minutes (equivalent to 0.15 standard deviations) longer than their counterparts at the first quartile of earned income. The best-fitting model suggested that this association is mediated by a common genetic background ($b_{0A}$ = 0.147, $p < 0.001$). The quasi-causal pathway was not statistically significant ($b_{0E}$ = –0.012, $p$ = 0.424). As is evident in the left panel of Figure 9.34, the genetic contributions to this association tended to be larger than the within-family contributions. Additionally, between-family confounds collectively account for nearly all of the total phenotypic correlation, evident in the right panel of Figure 9.34.

Illustrative analyses further highlight this shared environmental correlation. In Figure 9.35, we show mean MVPA durations for several pair types. We identified twin

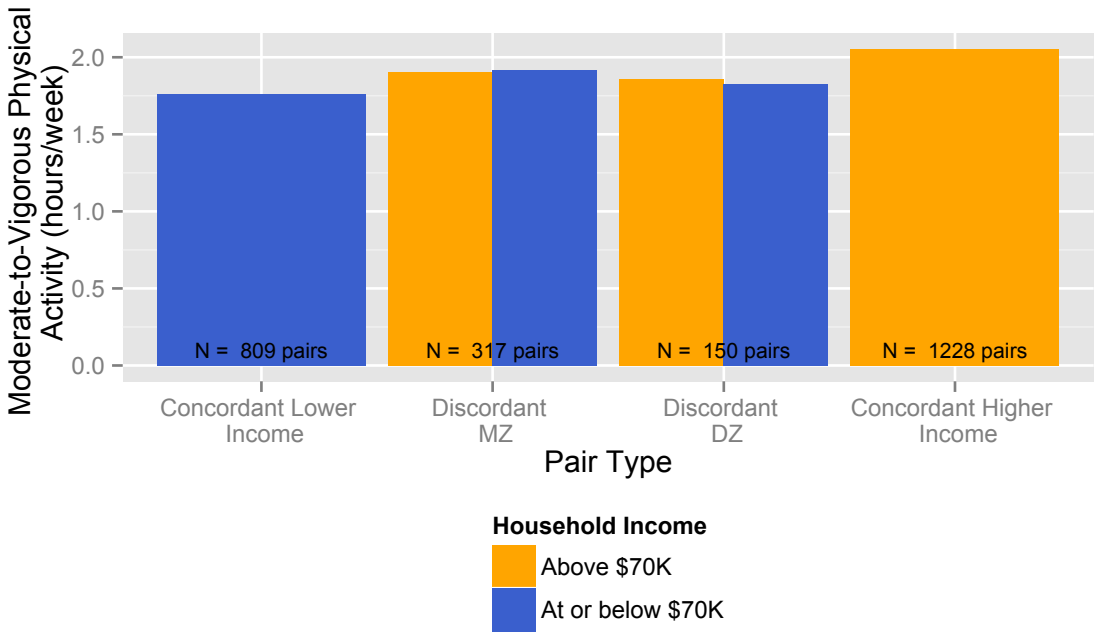


*Figure 9.35*. Illustrative analysis of the main effects of household income on moderate-to-vigorous physical activity in the WSTR. The twin earning less income (orange) is compared with the twin with earning more (navy) in MZ and DZ twin pairs.

pairs concordant for lower income (up to $70,000; orange), pairs concordant for higher income (over $70,000; navy), and pairs discordant for income level, and compared the mean MVPA of each of these groups. Overall, there is a main effect of household income on MVPA such that higher income is associated with greater physical activity levels (this is evident comparing the outer bars in this figure). Examining the relation at the within-pair level, however, shows that it is substantially attenuated within pairs of MZ twins (and slightly less so within pairs of DZ twins), consistent with genetic mediation of this association.

**Area Deprivation.** At the phenotypic level, the ADI was statistically significantly associated with greater physical activity ($b = 0.645$, $p = 0.014$), although the effect size was fairly small. Individuals at the third quartile of the ADI exercised for

approximately seven minutes (equivalent to 0.08 standard deviations) longer per week lower than individuals at the first quartile of the ADI. Because the ADI showed no influences from genetics and MVPA showed no influences from the shared environment, this effect was considered to be quasi-causal.

**Income Inequality.** At the phenotypic level, income inequality was statistically significantly associated with moderate-to-vigorous physical activity ($b$ = 0.190, $p$ = 0.040), but the effect size was negligible. Individuals at the third quartile of the Gini Index engaged in moderate-to-vigorous physical activity for approximately five minutes (equivalent to 0.06 standard deviations) longer on average than individuals at the first quartile of the Gini Index. The best fitting model suggested that MVPA was not quasi-causally associated with income inequality ($b_{0E}$ = 0.070, $p$ = 0.529); the genetic ($b_{0A}$ = 1.382, $p$ = 0.312) pathway was also not statistically significant, which likely represents lack of power to differentiate between these sources of covariation. These results suggest, however, that the relation between income inequality and MVPA is best explained by genetic factors that are common to both phenotypes rather than by systematic differences in exposure to socioeconomic factors. As is evident in the left panel of Figure 9.36, the between-family contribution to this association is larger than the within-family contribution. Also evident is that the between-family contribution accounts for nearly all of the total phenotypic correlation, as illustrated in the right panel of Figure 9.36.

Illustrative analyses mirror those observed for the other socioeconomic indicators. In Figure 9.37, we show pair differences in body mass index as a function of pair differences in the Gini Index within randomly paired individuals (unrelated relation; blue

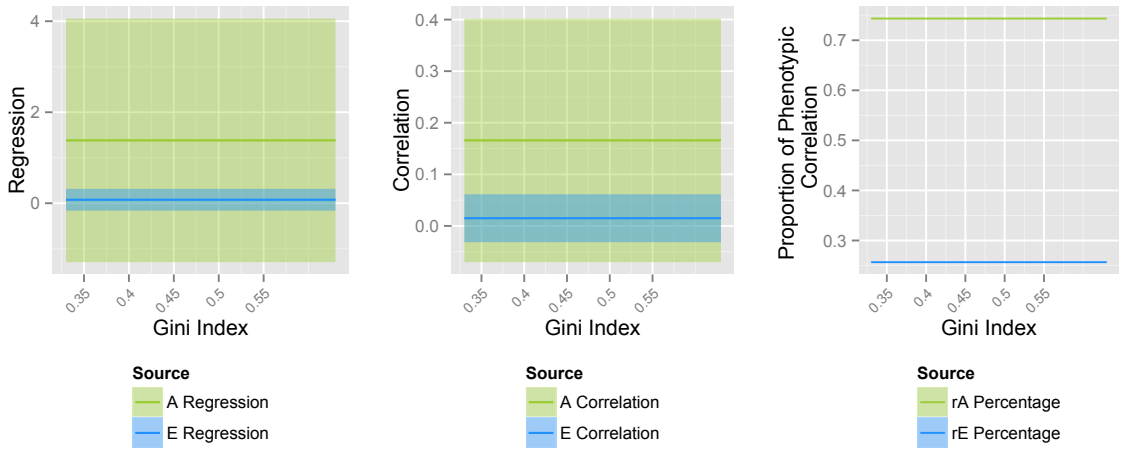


*Figure 9.36*. Main effects of income inequality on moderate-to-vigorous physical activity in the WSTR. The left panel shows the regression of MVPA on income inequality (scaled by a factor of 10). The same relation is presented in the middle panel as genetic, shared environmental, and nonshared environmental correlations. The right panel presents the proportion of the phenotypic correlation that is accounted for by the genetic, shared environmental, and nonshared environmental correlations. The asterisks (*) indicate that the magnitude of the regression of MVPA on income inequality depends on level of income inequality (these are not provided for correlations or percentages, as these are transformations of the parameters estimated in the model and therefore no statistical test was performed). The shaded regions represent 95% confidence intervals around the regression lines.

line) and within MZ (red line) and DZ (green line) pairs. If the protective effect of higher income inequality on BMI were causal, the slopes of these lines would closely approximate one another. Comparison of these lines suggests, however, that differences in income inequality do not predict differences in BMI within families, only between them. The slopes of the MZ and DZ lines are quite close, consistent with a common shared environmental pathway between income inequality and BMI.

**G×E Interaction: Effect of SES on Genetic and Environmental Etiology of Physical Activity.** The moderating effects of SES on variance in MVPA are presented in Table 9.7 under the heading *Effect of SES on Residual ACE Components of MVPA.* We discuss the interactive effect of each SES indicator on variance in MVPA below.

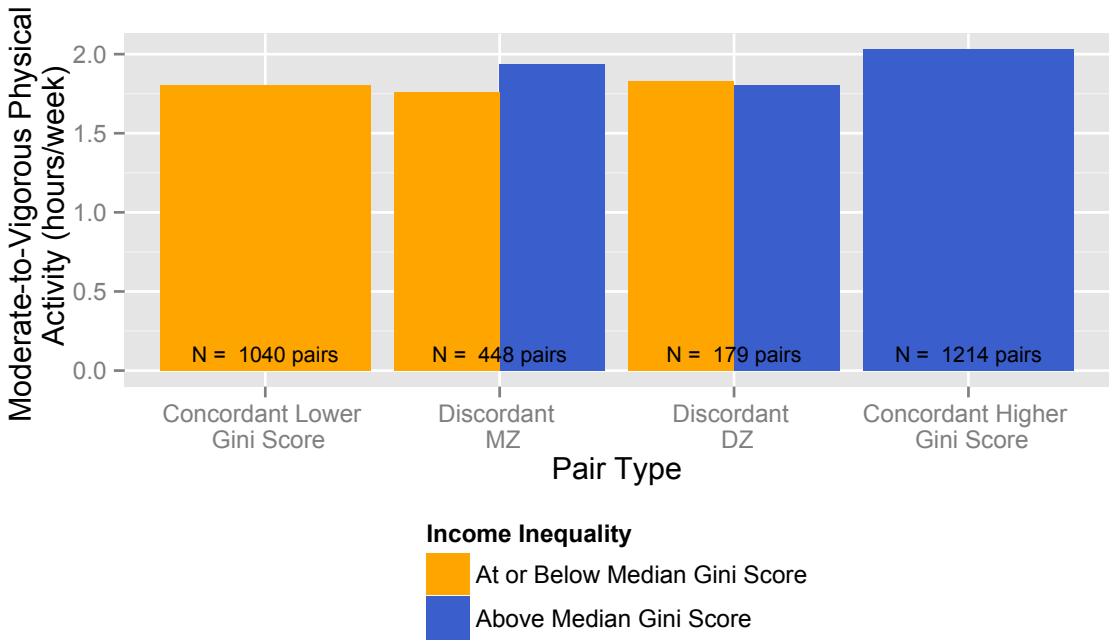


*Figure 9.37*. Illustrative analysis of the main effects of income inequality on moderate-to-vigorous physical activity in the WSTR. The twin living in a county with less income inequality (orange) is compared with the twin living in a county with greater income inequality (navy) in MZ and DZ twin pairs.

**Education.** The best fitting model suggested that residual phenotypic variance in MVPA decreased with increasing educational attainment. This effect was driven by decreases in nonshared environmental variance only ($b_{1Eu}$ = –0.030, $p$ = 0.005); there was no evidence of changes in additive genetic variance as a function of educational attainment. These model results are illustrated in Figure 9.38, which shows residual ACE variance components of MVPA as a function of educational attainment, represented as both regression lines (with 95% confidence intervals) and stacked variances. This figure clearly demonstrates how residual phenotypic and nonshared environmental variance in MVPA decreases with increasing educational attainment. The nonshared environmental correlation was static with respect to educational attainment, suggesting that low education potentiates nonshared environmental risk for low physical activity levels.

These results are also illustrated in Figure 9.39. Absolute pair differences in MVPA as a function of educational attainment for MZ and DZ twins are presented in the left panel of Figure 9.39, and violin plots showing the density of observations within each quartile of educational attainment are presented in the right panel of Figure 9.39. Consistent with our observations in the Add Health sample, variance in MVPA decreases with increasing educational attainment, an effect driven by fewer observations in the lower tail of MVPA when education level is high.

**Income.** The best fitting model suggested that residual phenotypic variance in MVPA did not vary with household income.

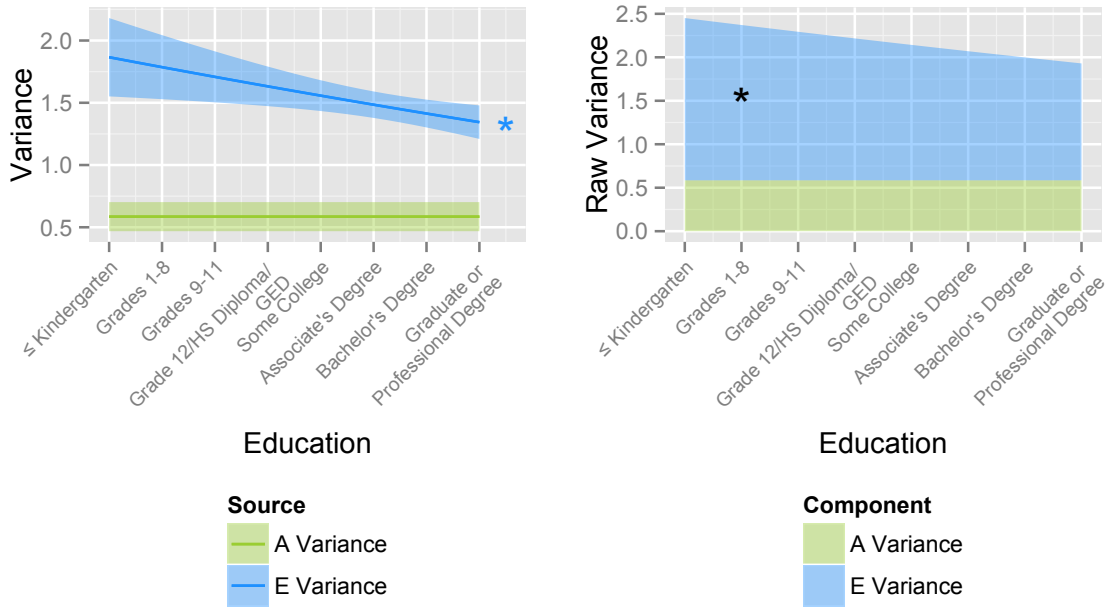


*Figure 9.38*. Gene-by-environment interaction between moderate-to-vigorous physical activity and educational attainment in the WSTR. The left panel shows ACE variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in MVPA decreases as a function of increasing educational attainment. The asterisks (*) indicate that variance in MVPA depends on level of education.

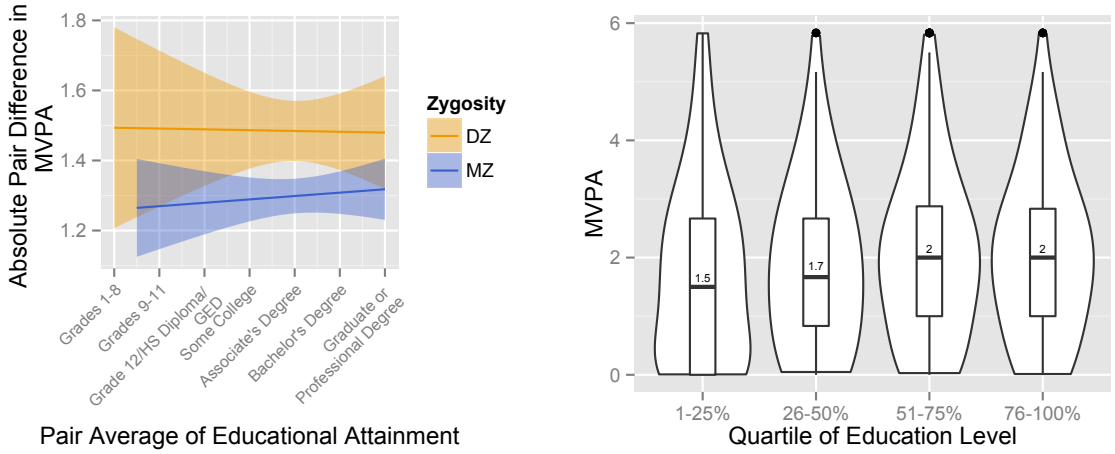


*Figure 9.39.* Illustrative analysis of the effects of educational attainment on variance in physical activity in the WSTR. The left panel shows absolute pair differences in MVPA as a function of educational attainment. The distance between the MZ and DZ lines represents the additive genetic variance in MVPA. The location of the MZ represents nonshared environmental variance in MVPA. The right panel shows box plots overlaid with violin plots, MVPA as a function of quartile of education level.

**Area Deprivation.** As was the case with educational attainment, the best fitting model suggested that residual phenotypic variance in MVPA decreased with decreasing area deprivation, an effect driven by decreasing nonshared environmental variance ($b_{1Eu}$ = –0.361, $p$ = 0.006). There was no moderation of the genetic variance as a function of area deprivation. These model results are illustrated in Figure 9.40, which shows residual A and E variance components of MVPA as a function of household income, represented as both regression lines (with 95% confidence intervals) and stacked variances. This illustrates the decrease in residual phenotypic and nonshared environmental variance in MVPA as a function of decreasing area deprivation. The nonshared environmental correlation was static with respect to household income, suggesting that high area deprivation potentiates environmental risk for low physical activity.

These results are also illustrated in Figure 9.41. Absolute pair differences in

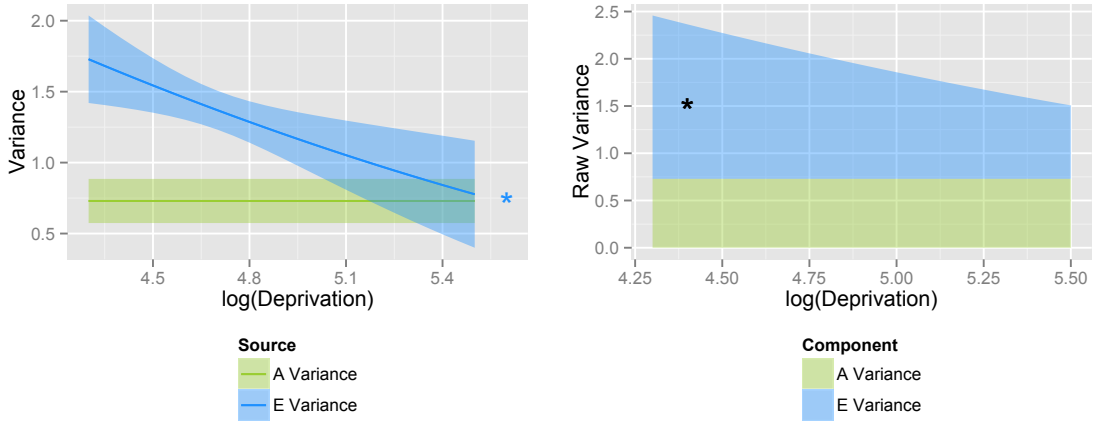


Figure 9.40. Gene-by-environment interaction between moderate-to-vigorous physical activity and area deprivation in the WSTR. The left panel shows A and E variance components as regression lines (shaded regions represent the 95% confidence intervals around these regressions). In the right panel, stacked variances illustrate how the total variance in MVPA decreases as a function of decreasing area deprivation. The asterisks (*) indicate that variance in MVPA depends on level of area deprivation.

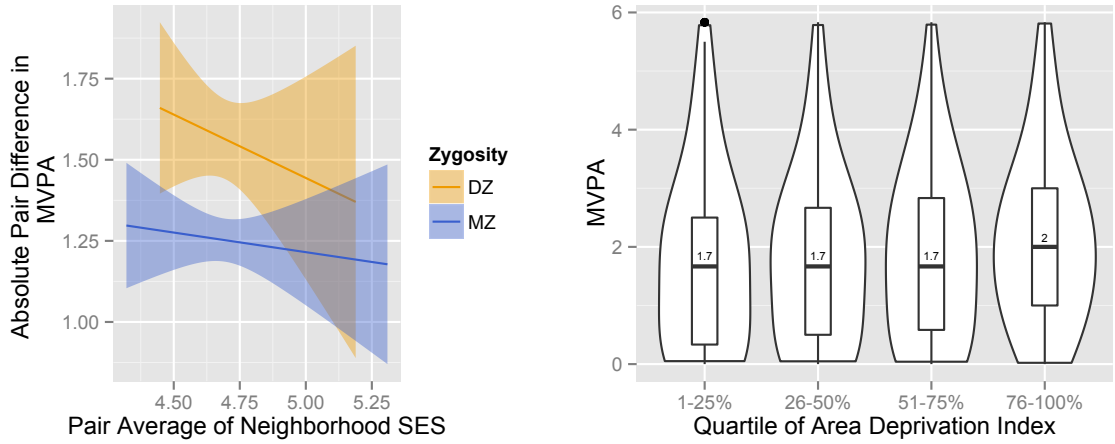


*Figure 9.41.* Illustrative analysis of the effects of neighborhood area deprivation on variance in physical activity in the WSTR. The left panel shows absolute pair differences in MVPA as a function of neighborhood SES. The distance between the MZ and DZ lines represents the additive genetic variance in MVPA. The location of the MZ represents nonshared environmental variance in MVPA. The right panel shows box plots overlaid with violin plots, MVPA as a function of quartile of neighborhood SES level.

MVPA as a function of neighborhood SES for MZ and DZ twins are presented in the left panel of Figure 9.41. Consistent with decreasing nonshared environmental variance in MVPA as a function of increasing neighborhood SES, the MZ regression line decreases slightly with increasing neighborhood SES. The violin plots (right panel of Figure 9.41) show that this constricting variance effect appears to be the result of fewer observations in the lower tail of MVPA when neighborhood SES level is high.

**Income Inequality.** The best-fitting model suggested that residual phenotypic variance in MVPA does not depend on county-level income inequality.

**Brief Summary.** Both compositional and contextual measures of socioeconomic status influenced mean levels of moderate-to-vigorous physical activity, and operated primarily through genetic pathways. Between-family factors accounted for a much larger proportion of the total phenotypic correlation than did within-family factors. Residual variance in MVPA also showed some influence from socioeconomic status. Educational attainment and neighborhood socioeconomic advantage reduced nonshared environmental variance in MPVA; genetic variance was unaffected by SES. These decreases in residual nonshared environmental variance appeared to be potentiated by socioeconomic status, and not by social selection factors.

## Chapter 10: Discussion – Health Behavior

Research consistently demonstrates a link between socioeconomic status and health behaviors such as substance use and physical activity. We summarize the existing research in detail in Chapter 1, and highlight several general observations here. First, higher SES is often associated with increased substance use, but low SES is more predictive of problematic substance use (Casswell et al., 2003; Galea et al., 2007; Hanson & Chen, 2007; Legleye et al., 2012; van Oers et al., 1999). The only exception to this observation seems to be smoking, which is robustly associated with low SES (Eibner & Evans, 2005; Goodman & Huang, 2002; Hiscock et al., 2012; Patrick et al., 2012). Similarly, lower physical activity levels are common among the less socioeconomically advantaged (Brodersen et al., 2007; Eibner & Evans, 2005; Janssen et al., 2006; Yen & Kaplan, 1998). Second, this association may strengthen with age (Fone et al., 2013; Karriker-Jaffe et al., 2013). Third, existing behavior genetics research on the SES–health behavior gradient demonstrates that this relation is far more nuanced than correlational or natural experimental studies suggest. This association is rarely found to be causal and is instead attributable to family-level confounds, especially additive genetic selection factors (Amin et al., 2015; Amin et al., 2013; Behrman et al., 2015; Krieger et al., 2005; Lundborg, 2013; Osler et al., 2007). Fourth, the complexities of this phenomenon are further made evident by research demonstrating SES effects on variance in health behaviors. For example, additive genetic variance in alcohol consumption was found to be greater among less educated, poorer individuals (Hamdi et al., 2015).

This dissertation study used contemporary samples of American adolescent and adult twins reared in the same household to examine the impact of socioeconomic status

on health behaviors more comprehensively. We examined the effect of both compositional (*e.g.*, educational attainment, household income) and contextual (*e.g.*, neighborhood socioeconomic advantage, area income inequality) measures of SES on level and variance in health behaviors, including substance use (*i.e.*, alcohol use, smoking status), physical activity, and sedentary behavior.

**Substance Use**

We summarize results for substance use in Table 10.1. We provide information regarding the direction of the main effect of SES on substance use (column labeled *Main Effect*) as well as the approximate percentage of the SES–health behavior phenotypic correlation accounted for by the additive genetic, shared environmental, and nonshared environmental correlations (column labeled *Approx. %* r*P*). We also provide information regarding the direction of the influence of SES indicators on residual variance in substance use (columns *Residual A*, *Residual C*, and *Residual E*). Also included in these variance columns is a summary of the changes in variance in the context of SES–health behavior ACE correlations and what those changes imply about causality versus social selection processes.

Our observations for the main effect of socioeconomic status on substance use were mixed. Consistent with prior research suggesting that the SES–substance use link strengthens with age (Fone et al., 2013; Karriker-Jaffe et al., 2013), we were not able to detect an effect of SES on substance use in the adolescent sample. In adults, we expected that SES would protect against engaging in substance use. What we observed was that higher compositional SES protected against alcohol use, but higher contextual SES had an iatrogenic effect. As noted above, higher SES is often associated with increased

Table 10.1. Summary of results for SES effects on substance use.

| | SES Indicator | Main Effect | Approx. % *r*P | Residual A | Residual C | Residual E |
|---|---|---|---|---|---|---|
| Alcohol Use (Add Health) | Parental Education | ∅ | N/A | ∅ | — | ∅ |
| | Family Income | ∅ | N/A | ∅ | — | ↑ |
| | Neighborhood SES | ∅ | N/A | ∅ | — | ∅ |
| Alcohol Use (WSTR) | Education | ∅ | —<br>—<br>— | ↓<br>stable *r*A<br>(causal) | ↓<br>stable *r*C<br>(causal) | ↓<br>stable *r*E<br>(causal) |
| | Income | Protective (*r*C ↑) | A = 80% → 40%<br>C = 10% → 60%<br>E = 10% → 0% | ↓<br>stable *r*A<br>(causal) | ∅ | ↓<br>stable *r*E<br>(causal) |
| | Neighborhood SES | Iatrogenic (*r*C) | —<br>C = 75%<br>E = 25% | ∅ | ↓<br>stable *r*C<br>(causal) | ∅ |
| | Income Inequality | Protective (*r*A, *r*C) | A = 35%<br>C = 55%<br>E = 10% | ∅ | ∅ | ∅ |
| Smoking (Add Health) | Parental Education | ∅ | N/A | ∅ | ∅ | ∅ |
| | Family Income | ∅ | N/A | ∅ | ∅ | ∅ |
| | Neighborhood SES | ∅ | N/A | ∅ | — | ∅ |
| Smoking (WSTR) | Education | Protective (*r*A, *r*E) | A = 65%<br>C = 25%<br>E = 10% | ∅ | ∅ | ∅ |
| | Income | Protective (*r*A, *r*E) | A = 70%<br>C = 0%<br>E = 30% | ∅ | ∅ | ∅ |
| | Neighborhood SES | Protective (*r*C) | —<br>C = 75%<br>E = 25% | ∅ | ∅ | ∅ |
| | Income Inequality | ∅ | —<br>—<br>— | ∅ | ∅ | ∅ |

substance use, but low SES is more predictive of problematic use (Casswell et al., 2003; Galea et al., 2007; Hanson & Chen, 2007; Legleye et al., 2012; van Oers et al., 1999). We note that our usage of a latent alcohol use factor did not allow us to discriminate between serious and recreational alcohol use patterns, which may have contributed to these discrepant results. The pattern for smoking status was consistent with prior research, however; higher SES reliably predicted lower likelihood of being a current smoker (Eibner & Evans, 2005; Goodman & Huang, 2002; Hiscock et al., 2012; Patrick et al., 2012). Where tests of causality were possible (*i.e.*, where SES indicators were not shared by members of a twin pair; true only for the WSTR), results were generally

consistent with our research hypotheses: The effect of SES on substance use was nearly entirely mediated by genetic and/or shared environmental pathways common to both phenotypes; nonshared environmental pathways accounted for no more than 30% of the total phenotypic correlation, and typically fell more on the order of 10%. These findings dovetail with existing behavior genetics research suggesting a non-causal link between socioeconomic status and substance use (Amin et al., 2015; Amin et al., 2013; Behrman et al., 2015; Lundborg, 2013; Osler et al., 2007).

As we noted with both mental and physical health, there is no "gene" for being rich that also makes one less likely to engage in substance use. Instead, we argue that temperament or personality characteristics that have a strong genetic basis (*e.g.*, neuroticism, conscientiousness) influence both phenotypes. For example, conscientious individuals may be more likely to pursue post-secondary education or seek higher paying or more skilled careers *and* be more likely to abstain more heavily from substance use. It is noteworthy, however, that iatrogenic contextual SES effects on alcohol use were dominated by a shared environmental background common to both phenotypes. This suggests that there may be family-of-origin characteristics that contribute to both living in a more advantaged neighborhood and engaging in greater alcohol use. Such factors might include family affluence, associated both with adolescent substance use and neighborhood choice.

Higher SES tended to predict less residual phenotypic variance in alcohol use, but was unrelated to variance in smoking status. These decreases in variance were typically environmental in nature, although there was also some evidence that, converging with prior research (Hamdi et al., 2015) and consistent with our research hypotheses, additive

genetic variance in alcohol use decreased as a function of increasing SES. Variance changes in alcohol use as a function of SES occurred in the context of stable ACE correlations between the two phenotypes, suggesting that SES operated in a causal fashion to produce changes in genetic or environmental variance. We were somewhat surprised to observe that higher family-level SES predicted increased nonshared environmental variance in adolescents. As we noted above, this may be the result of more frequent recreational alcohol use among more advantaged adolescents relative to their less advantaged peers. We would hypothesize that nonshared environmental variance would decrease with increasing SES if problematic use only were examined.

Like we observed with mental and physical health, socioeconomic status seemed to be working to bring in the upper tail of the alcohol use distribution. This observation in the context of E×E processes seems to support the dual distribution hypothesis (Tsang et al., 2017), which posits that the alcohol use distribution is a combination of a normal distribution under strong genetic control and a skewed distribution that is strongly influenced by individual environmental processes. Further, because additive genetic variance in alcohol use tended to be static (or decreased at a slower rate) with respect to SES while nonshared environmental variance decreased, heritability of alcohol use (*i.e.*, the proportion of variance in alcohol use accounted for by genes) increased as a function of SES. That is, the relative importance of genes at high SES was substantially greater than at low levels of SES. This observation further supports the dual distribution hypothesis: Individuals at higher SES levels tend to fall on a normal distribution that is under strong(er) genetic control because they have been less exposed to environmental processes that adversely impact alcohol use; individuals at lower SES levels tend to fall

on a skewed distribution that has substantial influence from the nonshared environment because they have experienced greater exposure to iatrogenic environmental processes.

***Implications.*** Like we observed for mental and physical health (see Chapters 6 and 8, respectively), we note that because SES and substance use tend to be correlated via between-family pathways, income redistribution or higher education incentives are unlikely to cause quantifiable shifts in *mean levels* of substance use within the general population. Yet, our research suggests that low socioeconomic conditions exacerbate—in a causal manner—environmental risk for greater alcohol use. That is, better socioeconomic conditions may help to reduce iatrogenic health behaviors of some individuals in a regression-toward-the-mean fashion. Substance use on a grand scale remains unchanged, but *the elevated substance use (primarily alcohol) experienced by some will be less severe*.

Our findings strongly support early interventions for individuals at risk for substance use. We observed SES effects on variance in alcohol use in adults but not adolescents, suggesting that the iatrogenic effects of low-SES accumulate over time. Policy makers might consider implementing school- or community-based interventions or healthy behavior promotion programs for at-risk (*i.e.*, low-SES) children. Movements such as the "truth" campaign, which creates anti-tobacco ads and commercials targeting youths, have been effective in reducing smoking rates among middle- (19.4% decrease) and high schoolers (8% decrease; Zucker et al., 2000). Similar campaigns for drinking behavior among youths seem long overdue. As school districts continue to witness budget cuts, classes such as health education should remain a priority. Further, harm-reduction protocols like the Brief Alcohol Screening Intervention for College Students

(BASICS; Dimeff, Baer, Kivlahan, & Marlatt, 1999), which has been shown to be effective in reducing alcohol use among college students in randomized controlled trials (Fachini, Aliane, Martinez, & Furtado, 2012), might be adapted for younger students and implemented in primary and secondary schools. Similar workplace- or community-based programs may be effective for at-risk adults. Many employers and healthcare insurance providers are implementing wellness programs that use financial incentives to set specific health targets (*e.g.*, tobacco surcharges on healthcare insurance premiums). We note, however, that such policies may in fact serve to further burden the socioeconomically disadvantaged, individuals who are already at greater risk for substance use. Employers or insurance agencies might instead consider offering financial incentives to *reduce or eliminate* substance use among individuals who identify as smokers or heavy drinkers. Employers (particularly of working-class jobs) might also consider using protocols like the BASICS program or other harm-reduction programs to help employees reach particular wellness goals. Physicians and mental health providers who encounter individuals with substance use issues might consider identifying family factors (*e.g.*, parental alcohol consumption or smoking, family attitudes regarding substance use) that may be contributing to a patient's own use.

**Physical Activity and Sedentary Behavior**

We summarize results for the effects of socioeconomic status on physical activity and sedentary behavior in Table 10.2. We did not observe any effects of SES on level of physical activity or sedentary behavior in adolescents. In adults, our results were consistent with prior research that socioeconomic status protected against low levels of physical activity Brodersen et al., 2007; Eibner & Evans, 2005; Janssen et al., 2006; Yen

Table 10.2. Summary of results for SES effects on physical activity and sedentary behavior.

| | SES Indicator | Main Effect | Approx. % *r*P | Residual A | Residual C | Residual E |
|---|---|---|---|---|---|---|
| Physical Activity (Add Health) | Parental Education | ∅ | N/A | ∅ | — | ∅ |
| | Family Income | ∅ | N/A | ∅ | — | ∅ |
| | Neighborhood SES | ∅ | N/A | ∅ | — | ↓ |
| Physical Activity (WSTR) | Education | Protective (*r*A) | A = 80%<br>—<br>E = 20% | ∅ | — | ↓<br>stable *r*E<br>(causal) |
| | Income | Protective (*r*A) | A = 80%<br>—<br>E = 20% | ∅ | — | ∅ |
| | Neighborhood SES | Protective (*r*E) | —<br>—<br>E = 100% | ∅ | — | ↓<br>stable *r*E<br>(causal) |
| | Income Inequality | Protective (*r*A) | A = 75%<br>—<br>E = 25% | ∅ | — | ∅ |
| Sedentary Behavior (Add Health) | Parental Education | ∅ | N/A | ∅ | — | ∅ |
| | Family Income | ∅ | N/A | ∅ | — | ∅ |
| | Neighborhood SES | ∅ | N/A | ↓ | — | ∅ |

& Kaplan, 1998). We hypothesized that this association would be mediated by between-family factors, and results overwhelmingly supported a non-causal explanation. Genetic factors were largely responsible for predicting mean physical activity levels, explaining upwards of 80% of the total phenotypic correlation between SES and physical activity. These observations were largely consistent with previous genetically informed research (Amin et al., 2013; Amin et al., 2015; Krieger, 2005; Lundbord, 2013; Osler et al., 2007). As we have argued elsewhere, the association between SES and physical activity is not induced by "genes for" SES causing greater exercise, but instead is a function of temperament or personality traits contributing to the expression of both phenotypes.

No research on SES effects on variance in physical activity or sedentary behavior currently exists, making this dissertation the first report of its kind. Because it was a positive health behavior, we hypothesized that SES would predict increased phenotypic variance in physical activity, and that this increase would be driven by increases in

additive genetic variance. This hypothesis is consistent with the stress–diathesis model (Bronfenbrenner & Cici, 1994), which suggests that environmental potentiation of positive or functional traits will occur in more advantaged environments. What we observed instead was that socioeconomic advantage predicted decreased phenotypic variance in physical activity and sedentary behavior. In both adolescents and adults, we observed decreased nonshared environmental variance in physical activity as a function of increasing neighborhood socioeconomic advantage. This effect appeared to be the result of fewer observations in the lower (*i.e.*, less physical activity) tail of physical activity at higher levels of neighborhood SES. In adults, we also observed this same effect for educational attainment, although we note that our results were most consistent for effects of contextual SES on physical activity levels.

We predicted that socioeconomic status would operate differently on variance in sedentary behavior due to its nature as a negative health behavior. We expected that additive genetic variance in sedentary behavior would decrease as a function of increasing SES, and this is indeed what we observed. Neighborhood socioeconomic advantage (but not other indicators of family-level SES) acted to decrease additive genetic variance in sedentary behavior. Like the majority of phenotypes investigated in this dissertation, neighborhood SES seemed to impact on sedentary behavior by pulling in the upper tail of sedentary behavior at high levels of SES. That is, fewer individuals residing in more wealthy neighborhoods were very inactive relative to their peers living in more disadvantaged neighborhoods.

***Implications.*** As we predicted and consistent with our observations for the majority of phenotypes studied in this dissertation, SES and physical activity were

correlated via additive genetic pathways common to both phenotypes. Because of this, we note that income redistribution or higher education incentives are unlikely to cause quantifiable shifts in *mean levels* of physical activity within the general population. A possible exception to this observation is neighborhood SES, which was observed to be causally related to physical activity. Our research also suggests, however, that low socioeconomic conditions exacerbate—in a causal manner—environmental risk for both low physical activity and high sedentary behavior. That is, better socioeconomic conditions may help to reduce iatrogenic health behaviors of some individuals in a regression-toward-the-mean fashion. Physical activity on a grand scale remains unchanged (although note the causal effects observed for neighborhood SES), but *the low levels of physical activity (or the extreme sedentary behavior) exhibited by some may increase (or decrease)*.

Like most of our dissertation findings, our results for the SES–physical activity/sedentary behavior gradient strongly support early interventions for individuals at risk for a sedentary lifestyle. The effects of neighborhood SES in particular seem to be operating on variance in physical activity and sedentary behavior at least as early as adolescence.

Policy makers might consider implementing school- or community-based interventions or healthy behavior promotion programs for at-risk (*i.e.*, low-SES) children. Many school districts are facing budget cuts, and physical education, recess, or extracurricular school sports are among the first activities to go (Bryant, 2011; Lang & Lillie, 2007). As budget cuts continue, policy makers might consider making attempts to preserve the physical education portion of school curricula. Ironically, some school

districts opt to spend money on materials that may increase sedentary behavior. For example, from 2015–2018, Charlottesville City schools (a Virginia school district with roughly 4,200 students) will spend $1.2 million to purchase laptops for 5th through 12th grade students (Shea, 2015) despite evidence to suggest that laptop use both interferes with in-class learning (Fried, 2008; Mueller & Oppenheimer, 2014; Sana, Weston, & Cepeda, 2008) and contributes to a sedentary lifestyle (Sandercock, Alibrahim, & Bellamy, 2016). School districts might elect to prioritize activities (such as physical education) that correlate with both better health and higher academic achievement (CDC, 2010). We note that contextual SES (neighborhood socioeconomic advantage) was the only SES indicator correlated with variance in physical activity and sedentary behavior among adolescents. This finding also underscores the importance of channeling funds into more deprived neighborhoods. Policy makers might focus on improving infrastructure (*e.g.*, adding/repairing sidewalks or bicycle lanes), reducing crime, building exercise facilities (or aiding existing facilities to reduce membership costs), and reducing or eliminating other barriers to at-risk (*i.e.*, low-SES) children and adolescents engaging in exercise within their area of residence.

Similar workplace- or community-based programs may be effective for at-risk adults. Many employers have implemented wellness programs that use financial incentives to set specific health or health behavior targets (*e.g.*, weight loss incentives, reduced fee gym memberships for individuals who work out a set number of days each week, supporting an hour of work time each day for physical activity). As we note above, policy makers might especially consider directing funds toward improving aspects of neighborhoods that promote physical activity, particularly in low-SES areas. For

example, accessible green space is associated with higher levels of physical activity (Janssen & Rosu, 2015; Toftager et al., 2011). Although low-SES individuals tend to have better access to activity-promoting facilities relative to their high-SES counterparts, research suggests they are less likely to utilize them (Giles-Corti & Donovan, 2002). Policy makers might also consider identifying actual or perceived barriers to utilizing physical activity resources or facilities among lower-SES individuals. Finally, physicians who encounter low-SES individuals might consider asking about physical activity or sedentary behaviors, and engage in collaborative problem-solving to identify how these patients can increase their activity levels.

**Health Behaviors as Mediators of the SES–Health Relation**

We briefly note that socioeconomic disparities in health behaviors may be one mediating factor in the SES–health gradient. Risky health behaviors such as poor diet, low physical activity levels, smoking, and alcohol consumption partially mediate the association between socioeconomic status and mortality (Lantz et al., 1998; Nandi, Glymour, & Subramanian, 2014; Pampel et al., 2010; Stringhini, Sabia, & Shipley, 2010). Similarly, smoking and exercise mediate the association between SES and immune response (Kershaw, Mezuk, Abdou, Rafferty, & Jackson, 2010). Physical activity mediates the association between SES and self-rated health (Khalalia, 2017; Senn, Walsh, & Carey, 2014), as does smoking, illicit drug use, and poor diet (Senn et al., 2014). As we discuss above, health behaviors and SES tended to be correlated via genetic or shared environmental pathways. The findings from these research studies, in the context of our study results, seems to lend support to our hypothesis that SES and health are mediated by personality characteristics or temperaments that contribute to both phenotypes.

**Summary**

Almost without exception, socioeconomic status is not causally related to health behaviors. Instead, these phenotypes share a common genetic (and, less commonly, shared environmental) etiology. We hypothesize that this etiology is in the form of personality characteristics or temperaments which have a strong genetic basis predicting behaviors that both interfere with upward movement on the socioeconomic spectrum *and* facilitate and/or maintain negative health behaviors (or interfere with engaging in positive health behaviors). In terms of real-world implications, these results imply that eliminating socioeconomic burdens in society will not influence mean levels of substance use or physical activity (with the exception, perhaps, of neighborhood SES and physical activity). On the other hand, our results also suggested that this association may be more nuanced than what is immediately apparent at the mean level.

We observed evidence that the effects of SES on health behaviors are the result of an interactive, reciprocally causal process which likely occurs over time and begins during childhood or adolescence. Almost without exception, socioeconomic status predicted decreased nonshared environmental variance in health behaviors. In addition, these variance changes were observed to be causal and the result of E×E interactions that were pulling individuals from the tail of the distribution marking greater engagement in poor or risky health behaviors. While income redistribution or widespread availability of educational opportunities may not affect mean levels of health behaviors, it may facilitate healthier lifestyle choices for some, particularly those at greatest socioeconomic and environmental risk for poor health.

# Chapter 11: General Discussion

We conclude this dissertation with a chapter briefly summarizing and integrating our research findings. We also outline study strengths and limitations, and discuss potential directions for future research. Finally, we end with some concluding remarks regarding what money can (and can't) buy in 21st century America.

## Brief Summary of Research Findings

Research supporting a link between socioeconomic conditions and health has long existed. The first such population report on socioeconomic predictors of mortality emerged in the late 17th century (Antonovsky, 1967), and thousands of reports documenting the SES–health gradient have followed (Evans, Wolfe, & Adler, 2012). Consistent with prior research, we observed that socioeconomic advantage protects against mental illness, poor physical health, and negative health behaviors. Also in line with previous research, we observed that this protective effect is not causal in nature but instead appears to be due to an underlying genetic, and to a lesser extent shared environmental, background common to both SES and health outcomes and behaviors.

Based on these findings, we suggested that there are no "genes for" being wealthy or attending college that also cause better health. Instead, we posited that this SES–health relation is mediated by temperamental or personality factors (which tend to have a strong genetic basis, *e.g.*, conscientiousness, neuroticism) that predict both phenotypes. We also noted that this SES–health between-family relation (particularly in the case of physical health) may be further mediated by health behaviors: Temperament may predict health behaviors, which in turn predict subsequent health. In that respect, the association between socioeconomic status and health is more about lifestyle choices that have

implications for health and well-being.

We also observed that variance in mental health, physical health, and health behaviors decreased as a function of increasing SES. Our results suggest that this decrease is both a causal process (*i.e.*, is due to differences in exposure to socioeconomic conditions) and is predominantly driven by decreases in nonshared environmental variance[27]. Further, we observed that socioeconomic status seemed to be operating by pulling in individuals from the tails of the health distribution representing pathology (*i.e.*, the very sick appeared to benefit from more advantaged socioeconomic conditions)[28]. As we note below (see discussion regarding the dual distribution hypothesis), this pattern of results has important implications for understanding how pathology develops, as well as how pathological traits are represented in statistical analyses.

Overall, results from this dissertation suggest that improving the socioeconomic conditions of the poor or underprivileged will not influence mean levels of health because these phenotypes are correlated via common genetic (and, to a lesser extent, family environmental) pathways. On the other hand, this dissertation offers good evidence that reducing the socioeconomic burden of disadvantaged individuals will reduce the health burden of some, particularly those at greatest environmental risk for poor health or negative health behaviors.

**Strengths, Limitations, & Future Directions**

***Longitudinal Designs and Causality.*** The primary limitation of this dissertation

[27] Note that heritability in health tended to increase as a function of increasing SES due to additive genetic variance remaining stable in the context of decreasing environmental variance.

[28] We demonstrated this in illustrative analyses, not statistically; we were only able to make inferences regarding how SES influences individuals in the tail ends of health.

research also highlights a much needed area of future research. Our analyses were cross-sectional in nature (as have been all genetically informed studies of the SES–health gradient), which presents two limitations. First, although we are able to control for many confounds (in adults) by using the co-twin control design, the possibility always exists that the direction of effects are reversed or, more likely, reciprocal. Where it seemed necessary, we tested for the direction of causality (c.f. Duffy & Martin, 1994; Heath et al., 1993). Nevertheless, a longitudinal study design facilitates more careful examination of causal processes. Second, we engaged in considerable speculation regarding why we observed the results that we did in the context of existing theories of genetic and environmental influences on (and interactions in) the expression of human behavior. Longitudinal studies will help to better elucidate the mechanisms by which, for example, SES leads to reduced phenotypic and nonshared environmental variance in internalizing psychopathology. Below we discuss several potential study designs that may achieve these ends.

We note that shared SES indicators make it impossible to determine causality, regardless of whether a longitudinal design is employed. Tests of causality will not be possible in children or adolescents when family-level indicators are used. Unfortunately, these also happen to be the SES indicators that may be most appropriate for inferring causality in the SES–health gradient. Lifespan research suggests that the effects of low SES on health accumulate over time (Fone et al., 2013; Karriker-Jaffe et al., 2013; Ross & Wu, 1996; Miech & Shanahan, 2000; Prus, 2007; Singh-Manoux, Ferrie, Chandola, Marmot, 2004; van de Mheen, Stronks, & Mackenbach, 1998; Williams et al., 2013; Yen & Kaplan, 1998) and have their debut in childhood (Singh-Manoux et al., 2004; van de

Mheen et al., 1998). Future research may explore opportunities for conducting natural experiments on the SES–health gradient, comparing, for example, SES effects on health in children and adolescents in capitalist versus socialist nations; countries with high income inequality versus those with low inequality; or societies with inexpensive or no-cost post-secondary educational opportunities versus those with costly ones. Alternatively, researchers may choose to use samples with twins or siblings separated at birth where one child is reared by the biological parents and the other is adopted into a non-biological family (*e.g.*, the Swedish Twin Registry; see Kendler, Turkheimer, Ohlsson, Sundquist, & Sundquist, 2015), or children-of-twin designs where the subjects of interest (cousins) are the children of identical or fraternal twins (*e.g.*, the Australian Twin Registry; see D'Onofrio et al., 2005). Longitudinal research also has the benefit in that it allows for the investigation of the impact that moving in or out of a particular SES level has on health or health behaviors.

As noted above, the association between socioeconomic status and mean levels of health or health behaviors appears to grow stronger with age (Fone et al., 2013; Karriker-Jaffe et al., 2013; Ross & Wu, 1996; Miech & Shanahan, 2000; Prus, 2007; Singh-Manoux et al., 2004; van de Mheen et al., 1998; Williams et al., 2013; Yen & Kaplan, 1998). We also observed in our dissertation analyses that variance effects were generally more apparent in adulthood. A strength of this dissertation is that we controlled for the linear effects of age (and gender) in level of and variance in health. Few studies have used such an approach (*e.g.*, Dinescu et al., 2015; Horn et al., 2015; McCaffery et al., 2009; Strachan et al., 2016) and just one has examined the interactive effect of age and a moderating environment (McCaffery et al., 2009). We note that in this dissertation we

did not test for the interactive effect of age and SES on level and variance in health, and it will be important for future research to include such a parameter. Future longitudinal research may also account for within- and between-measurement occasion changes in level and variance in health as a function of both age and socioeconomic status.

***The Dual Distribution Hypothesis.*** We discussed the dual distribution hypothesis (Tsang et al., 2017) in some detail in Chapter 6. Briefly, the dual distribution hypothesis posits that human phenotypes in which a portion of the trait distribution represents pathology can in fact be represented as the sum of two separate distributions. Many individuals fall on a normal distribution which is heavily influenced by genetic factors. The remaining individuals follow a distribution skewed in the direction of higher pathology and influenced predominantly by the nonshared environment. As noted above, we demonstrated in our study that socioeconomic status acts to pull individuals in from the tail of the health distribution representing pathology. As research on the dual distribution hypothesis develops and broadens by incorporating predictors of the ACE composition of the normal and skewed distributions composing the greater health distribution, future genetically informed SES–health gradient research might test the applicability of the dual distribution hypothesis to health phenotypes and their predictors.

***Phenotype-Environment Correlation and Levels of Analysis.*** The dual distribution hypothesis in and of itself is not enough to explain the nonshared environmental variance phenomenon we observed in the dissertation analyses. In Chapter 6, we proposed that phenotypic (and, more specifically, nonshared environmental) variance changes (*i.e.*, decreases) as a function of increasing SES are a sign that pathology is the outcome of a process and not of genetic determinism. That is,

our results suggest that pathological processes are individual processes that operate above and beyond genetic influences on health, and these processes are not correlated with family factors per se. It is absolutely true, however, that some individuals are more prone toward obesity, internalizing disorders, or substance use than other individuals, and that these phenotypes are influenced by genetic factors. At the same time, it is often the *phenotype*, and not the genotype, that is responsible for individuals selecting into protective or iatrogenic environments that reciprocally affect the phenotype, thereby furthering pathological (or protective) processes (Beam & Turkheimer, 2013; Beam, Turkheimer, Dickens, & Davis, 2015; Bronfenbrenner & Cici, 1994; Dickens & Flynn, 2001; Turkheimer, 2004; Turkheimer & Gottesman, 1996). Longitudinal research fitting models that account for phenotype-environment correlation (*r*PE) may be influential in demonstrating how pathology (exhibited primarily by those in the skewed distribution of a dually-distributed trait) develops and/or is maintained.

Related to *r*PE, we chose to conduct (moderated) bivariate Cholesky ACE decompositions of our data in order to test specific hypotheses regarding the SES–health gradient. We note, however, that the processes contributing to the variance changes we observed may be operating at a more fundamental level. For example, we observed that heritability (*i.e.*, the total proportion of variance in a phenotype accounted for by genetic factors) of health tends to expand with increasing socioeconomic advantage. The simplest explanation for this finding is perhaps conceptualizing the process as a comparison of zygosity-dependent differences in twin similarities. Indeed, future research may, in order to identify specific mechanisms of change, choose to analyze the SES–health gradient at this more fundamental level by exploring the influence of SES on

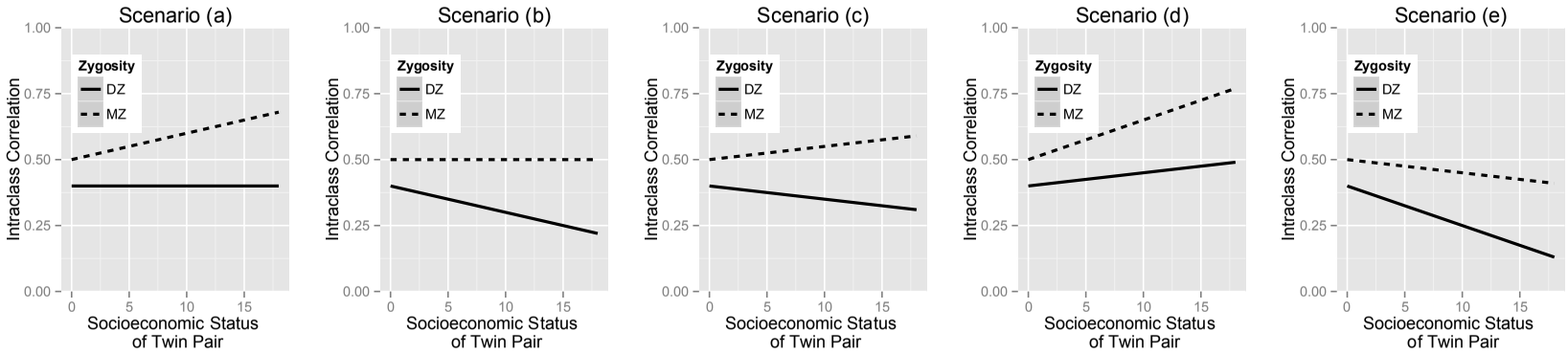


*Figure 11.1*. Hypothetical scenarios in which the comparison of zygosity-dependent differences in twin similarities leads to increased heritability as a function of socioeconomic status. In Scenario (a), the intraclass correlation of MZ twins (*r*MZ) increases, while the intraclass correlation of DZ twins (*r*DZ) remains stable with respect to SES. In Scenario (b), *r*MZ remains stable, while *r*DZ decreases. In Scenario (c), *r*MZ increases and *r*DZ decreases. In Scenario (d), both *r*MZ and rDZ increase, but *r*MZ increases at a faster rate. Lastly, in Scenario (e), both *r*MZ and *r*DZ decrease, but *r*DZ decreases at a faster rate. Although each of these scenarios represents a different hypothetical process by which the heritability of a phenotype may increase with respect to SES, the rate of increase in heritability is in fact identical in each case (equal to 0.18 unit increase per unit of SES).

MZ/DZ twin correlations and/or variances in health. Figure 11.1 illustrates several hypothetical MZ/DZ processes by which the heritability of health may increase with increasing SES. Importantly, the rate at which heritability increases is exactly equivalent in each of these scenarios (*i.e.*, heritability begins at 20% at the lowest SES level and increases to 56% at the highest). Scenario (a) demonstrates that heritability of health increases with SES because high SES forces MZ twins, relative to DZ twins, to be more similar to one another relative to lower socioeconomic environments (*i.e.*, MZ twin correlations are increasing with respect to SES while DZ twin correlations remain stable). It might instead be true that high SES fosters dissimilarity in DZ twins whereas MZ twin differences remain stable (*i.e.*, DZ twin correlations decrease with respect to SES while MZ twin correlations remain stable; Scenario (b)), or that each of these processes occurs simultaneously (*i.e.*, MZ twins grow more similar with respect to SES while DZ twins grow less similar; Scenario (c)). Scenarios (d) and (e) illustrate processes in which both MZ and DZ twins grow more or less similar, respectively, but at different rates, all the while still yielding the same linear increase in heritability. Each of these scenarios implies a very different mechanism by which SES impacts on standardized ACE variances in health, and implies that it is simply not enough—and potentially misleading—to conclude that heritability of health increases with increasing socioeconomic status.

***Standardized versus Unstandardized Variance in Health.*** The scenarios illustrated in Figure 11.1 extend naturally to absolute pair differences, which yield information about raw genetic variance in health rather than heritability per se. This is an important difference. Although we do argue that it is important to determine the relative

contributions of familial and environmental influences on a phenotype, we believe it perhaps is more important to examine the absolute contributions of these components. By definition, heritability is the proportion of variance in a phenotype that is attributable to genetic influences, and does not hold information about the magnitude of genetic variance contributing to the phenotype. Estimating raw additive genetic variance in health (as opposed to heritability) as a function of SES differentiates among several processes that yield increases in heritability. These hypothetical processes are illustrated in Figure 11.2, which shows raw variances in health as a function of SES in the top row, and the corresponding standardized variances in the bottom row. Scenario (a) shows raw additive genetic (A) variance increasing with respect to SES, and shared environmental (C) variance and nonshared environmental (E) variance remaining stable across all levels of SES, which corresponds to a heritability estimate—driven by increases in A variance—that increases with SES. In Scenario (b), all raw ACE variances are increasing, but the increase in heritability is driven by the fact that A variance increases at a faster rate than either C or E variances. Scenario (c) shows a different process for raw additive genetic variance. In this case, A variance remains stable (*i.e.*, is unchanging) with respect to SES, while C and E variances decrease; although there is an increase in heritability because of decreases in the total variance of health, this process is not being driven by changes in genotypic influences on health. In Scenario (d), both genetic and environmental variances are decreasing, but heritability is increasing because A variance decreases at a slower rate compared with C and E variances. Finally, Scenario (e) shows a case in which ignoring raw variances can mask can mask important processes that may be occurring (*e.g.*, changes in total variance). In this scenario, all ACE variances are

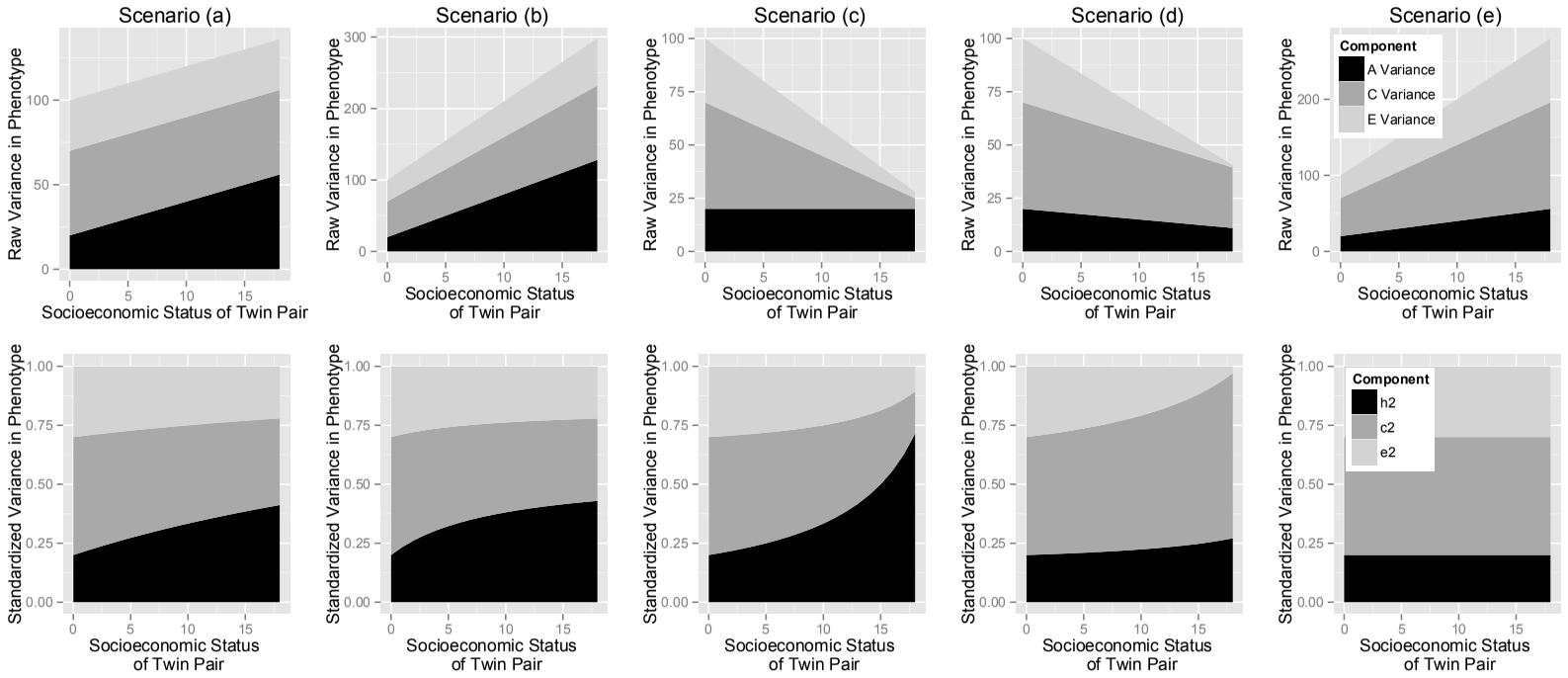


*Figure 11.2*. Hypothetical scenarios in which comparison of raw (top row) and standardized (bottom row) additive genetic (A), shared environmental (C), and nonshared environmental (E) variance components as a function of socioeconomic status may lead to different conclusions about the heritability of a phenotype. In Scenario (a), raw A variance increases while C and E variances remain stable, leading to increased heritability of the phenotype with respect to SES. In Scenario (b), all raw variances are increasing, with A variance increasing at the fastest rate, leading to an increase in heritability. In Scenario (c), A variance remains stable (*i.e.*, is non-changing) while C and E variances decrease, leading to an increase in heritability with respect to SES but no real change in the genetic influences on the phenotype. In Scenario (d), all variances are decreasing, with A variance decreasing the slowest rate, leading to increased heritability with respect to SES but a decrease in genetic influences on the phenotype. Scenario (e) shows how examining the heritability of a phenotype alone can mask changes in total variance. In this scenario, all variance components are increasing but their relative influences on the phenotype remain stable with respect to SES.

increasing, but at a rate proportional to the variance present at the lowest level of SES, yielding no observable changes in heritability despite increases in A variance. A noted strength of this dissertation is that we examined how socioeconomic status influences *raw* variance in health, and we were therefore able to identify that heritability of health increases (almost exclusively) because nonshared environmental variance decreases as a function of SES. The next step in research on the health variance effects produced by SES is to extend this design into the longitudinal, repeated measures domain.

***Parametric versus Nonparametric Gene×Environment Interaction.*** In our dissertation analyses, we fit traditional, parametric-based G×E models to the data (*e.g.*, Purcell, 2002; Medland et al., 2009). Specifically, we assumed that SES was linearly related to level and variance in health and health behaviors. This assumption may not always be accurate, however. Recently, Briley and colleagues (Briley, Harden, Bates, & Tucker-Drob, 2015) introduced a method for fitting nonparametric G×E models to data which uses local structural equation modeling (LOSEM; Hildebrandt, Wilhelm, & Robitzsch, 2009). Briefly, LOSEM uses locally weighted regression to fit a loess curve to model parameters as a function of a moderating variable (*e.g.*, socioeconomic status). The result is a smoothed line that is sensitive to data points only within a particular window of the moderator. This method has been shown to be useful in identifying interactions where they were previously believed to be absent (Briley et al., 2015). We suggest that it may also be useful in more closely identifying the range of the socioeconomic spectrum that may be most correlated with variance in health or health behaviors; that is, LOSEM may be a helpful tool for localizing where SES–health gradient interventions might be made. Currently, LOSEM supports only family-level

moderators (*i.e.*, moderators shared by members of a twin or sibling pair). As this method becomes more sophisticated, it may prove useful for the study of individual-level moderators as well, and would not require the assumption that socioeconomic status exerts equal influence on variance in health or health behaviors at all levels of SES. Indeed, we would hypothesize that SES exerts more influence on variance in (and perhaps level of) health and health behaviors at low levels compared with high levels of socioeconomic status.

***Relative Contributions of Compositional and Contextual SES.*** Another area of future research is considering the independent effects of socioeconomic status indicators on level and variance in mental health, physical health, and health behaviors. Specifically, fitting mediation models when using multiple indicators (for an example, see Horn et al., 2015) will yield information about the influence of compositional and contextual measures of SES relative to one another. As noted in Chapter 1, compositional and contextual measures of SES are weakly to moderately correlated (Demissie et al., 2000; Greenwald et al., 1994; Marra et al., 2011 ), but also have effects independent of one another (Kondo et al., 2009). Mediation models may help to elucidate important differences among types of SES indicators. Such models may also be important for informing public policy. Research demonstrating, for example, that neighborhood SES reduces environmental risk for chronic health conditions above and beyond the effects of compositional measures (such as education and income) could serve as a catalyst for funneling economic resources or government funds into deprived neighborhoods.

Related, future research might also focus on differences between relative (*i.e.*,

subjective) and absolute (*i.e.*, perceived) measures of socioeconomic status. Previous research suggests that subjective measures of SES may be more closely correlated with health than objective measures (Adler et al., 1994; Adler et al., 2000; Ostrove et al., 2000; Singh-Manoux et al., 2005). Importantly, however, we observed that one rubric for relative/subjective SES, income inequality, was nearly universally *unrelated* to mental health, physical health, and health behaviors, and where effects did exist, they were non-causal in nature. We note that the median income of our adult sample was between $50K-60K, and the majority of the sample was comprised of relatively privileged individuals (*e.g.*, predominantly white, greater than 75% having some college education). Although representative of the population of the State of Washington, the sample is not representative of the entire U.S. population; it is possible that a less homogenous sample would have yielded more pronounced effects for income inequality. Nevertheless, future research may focus on other measures of relative or subjective SES and their comparison with absolute measures, such as the ladder instrument (Adler et al., 2000).

***Exploratory Visual Analysis.*** An interesting lens through which future research might explore the SES–health gradient is exploratory visual analysis (Davis, Haworth, Lewis, & Plomin, 2012). This approach seems particularly relevant to public policy in that it would facilitate identification of geographic "hot spots" for SES effects on variance in health. In this method, twins' distances from a given location are used to weight the observed covariance matrix used in model estimation for that location:

$$w_i(x) = \frac{1}{\sqrt{d(x,\ x_j)}} \tag{11.1}$$

where $x$ represents a given location, $x_j$ represents the midpoint of the twins' locations,

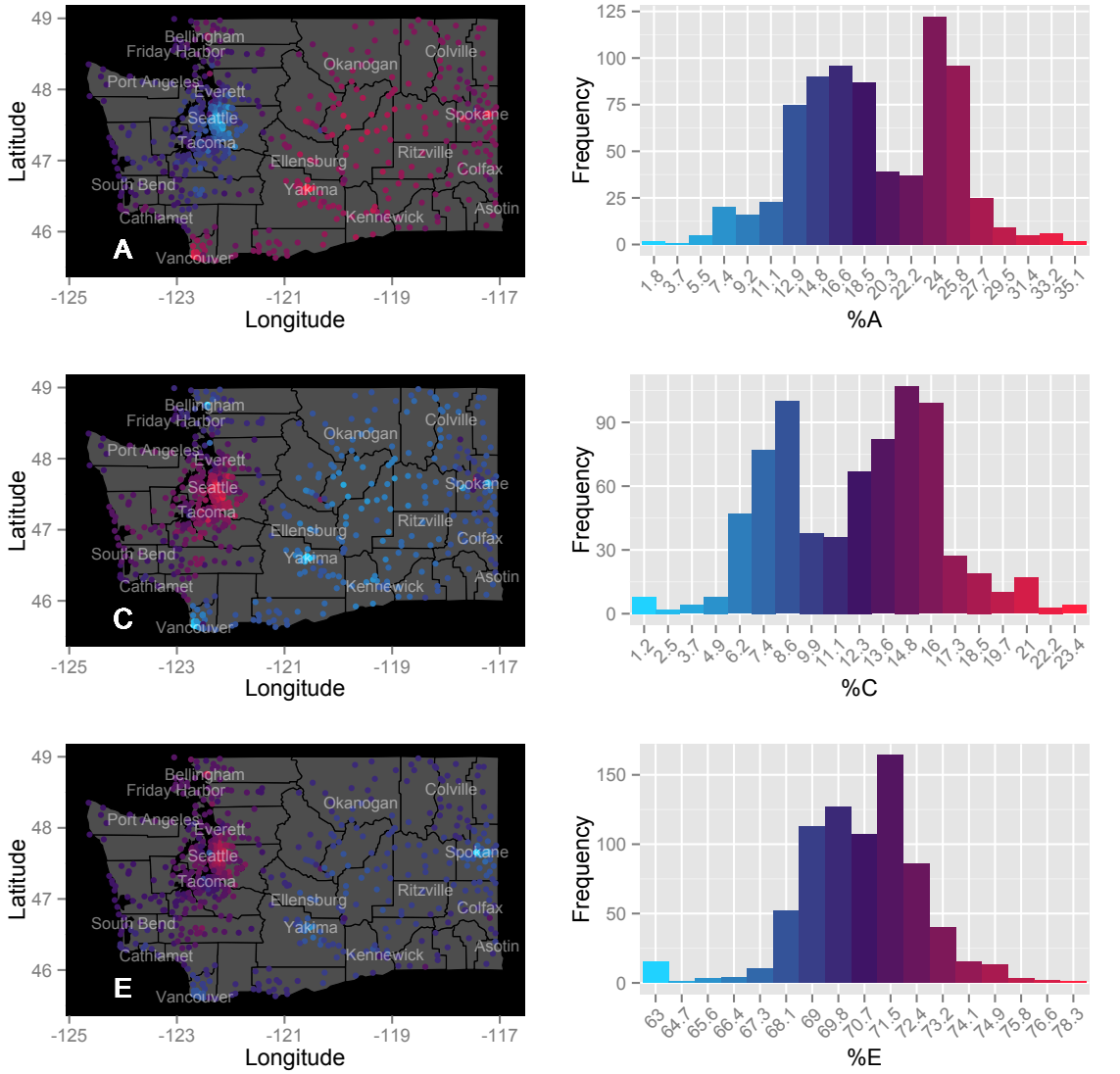


*Figure 11.3*. Exploratory visual analysis of self-rated health as a function of zip code in the State of Washington in a subsample of WSTR twins. The points on the map (left panels) are positioned in the center of a zip code and are colored according to the proportion of variance in self-rated accounted for by A (top), C (middle), or E (bottom). The frequency distributions in the right panel are color keys to the map and show the distributions of $a^2$, $c^2$, or $e^2$.

and $d$ represents the Euclidean distance between the location coordinates and the coordinates of the midpoint between members of a twin pair. The classical twin model can then be fit to each location's weighted covariance matrix and the results can be

placed on a map (c.f. Davis et al, 2012). To demonstrate what such an analysis looks like in the WSTR, we conducted a univariate biometric decomposition of variance in self-rated health as a function of zip code in the State of Washington (see Figure 11.3).

What is most striking about this plot is that self-rated health appears to show stronger environmental influences in urban environments (*e.g.*, Seattle, Tacoma) and is more heavily genetic in the more rural, desert portion of the state. This can be compared with a similar plot showing instead the mean neighborhood SES of a given zip code (see Figure 11.4). Where neighborhood SES is higher (which also happens to be in urban environments), environmental influences are more influential to self-rated health[29]. Currently, this method applies only to univariate analyses, does not allow for one to properly test interactive effects on variance components for moderators that are not shared between members of a twin pair (without averaging), and conducts no statistical testing (*e.g.*, cannot test whether the difference in the environmental effects on self-rated health in Seattle versus Spokane is statistically significant). Nevertheless, this exploratory method may contribute meaningfully to research on the SES–health gradient.

***Environment-Wide Association Studies.*** Some research suggests that individuals at the low end of the socioeconomic spectrum are disproportionately exposed to environmental health hazards (Berney et al., 2000). Likewise, we observed in our

[29] We note that in the dissertation analyses, neighborhood SES was related to small *increases* in heritability of self-rated health, whereas this figure suggests decreases in heritability as a function increasing neighborhood SES. This may be due in part to a number of factors. First, this analysis was conducted using a subsample of WSTR twins. Second, the means shown in this plot are weighted means rather than means only of individuals residing within that particular zip code. Third, in the dissertation analyses, we used a measure of neighborhood SES (the Area Deprivation Index) that was standardized across the entire United States, whereas this analysis used a measure standardized only within this subsample of WSTR twins (the Singh Index; Singh 2003). Finally, using zip codes for this type of analysis may in fact be too broad a geographic region, and may not reflect important differences that exist between neighborhoods or census tracts located within a given zip code.

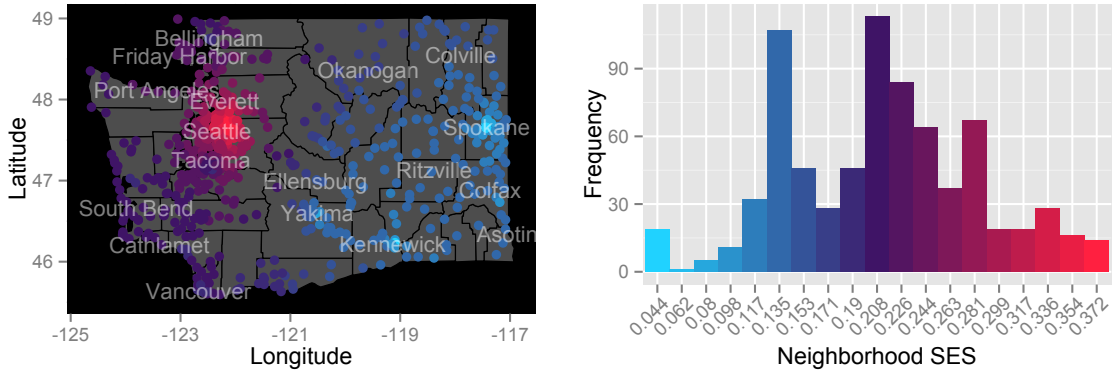


*Figure 11.4*. Exploratory visual analysis of mean neighborhood SES as a function of zip code in the State of Washington in a subsample of WSTR twins. The points on the map (left panels) are positioned in the center of a zip code and are colored according to the mean area deprivation score. The frequency distribution in the right panel is a color key to the map and shows the distribution of neighborhood SES.

analyses that low-SES individuals are at greater environmental risk for poor mental or physical health or negative health behaviors. A next step in understanding how SES impacts on nonshared environmental variance in health and health behaviors is to identify what environmental exposures seem to be exacerbated by socioeconomic deprivation. An obvious area of future research seems to lie in environment-wide association studies (EWAS; Patel & Ioannidis, 2014). EWAS are an extension of the widely employed genome-wide association study method (GWAS; see Bush & Moore, 2012, for a review), and explore the association between myriad environmental exposures and risk for disease. EWAS has already been employed for several health phenotypes, including type 2 diabetes (Hall et al., 2014; Patel, Bhattacharya, & Butte, 2010), blood pressure (McGinnis, Brownstein, & Patel, 2016), metabolic syndrome (Lind, Risérus, Salihovic, van Bavel, & Lind, 2013), all-cause mortality (Patel et al., 2013), and pre-term birth (Patel et al., 2014). The directive of EWAS is to identify the *exposome*, or the totality of

an individual's environmental exposures (Wild, 2005). The exposome "compliments the (epi)genome while providing a multitude of opportunities for intervention if exposures can be eliminated or minimized" (Louis & Sundaram, 2012, p. 2659). Identifying factors composing the exposome can then lead to more effective interventions for those most at risk for poor health (*i.e.*, low-SES individuals).

***Race, SES, and Intersectionality.*** A final and important area of future research we would like to highlight is the role of race and/or ethnicity in the SES–health gradient. Racial and ethnic minorities are overrepresented among low-SES groups in the United States (Williams, Mohammed, Leavell, & Collins, 2010). Further, racial inequalities in terms of health and longevity have also long existed (Braveman, Cubbin, Egerter, Williams, & Pamuk, 2010; Williams, 2012; Williams et al., 2010), and are observed even after accounting for socioeconomic disparities (Braveman, Cubbin, Egerter, Williams, & Pamuk, 2010; Williams et al., 2010). Some research suggests that the intersectionality between race and SES may have less of an impact on self-rated health compared with 30 years ago, but intersectionality effects do persist (Cummings & Jackson, 2008). A noted limitation of this dissertation is that we did not account for the role of race/ethnicity in our analyses. In addition, the WSTR, although representative of the State of Washington, is a predominantly white, relatively privileged sample. Future research might use samples that are more representative of the U.S. population, and should control not only for the main effects of race/ethnicity on health, but also its interaction with SES to predict health outcomes.

**Concluding Remarks**

At the outset of this dissertation, we aimed to determine how socioeconomic

status influences mental health, physical health, and health behaviors in Modern America. We discovered that health and health behaviors are largely related to one another via between-family (*i.e.*, non-causal) pathways. Further, we observed that higher socioeconomic status tends to causally reduce environmental risk for poor health or health behaviors. We also noted that this decrease in environmental variance coincided with a tendency for socioeconomic advantage to yield fewer extreme scores in the tail of the health or health behavior distribution marking pathology. Overall, results suggest that the SES–health gradient is far more nuanced than meets the eye.

So what will money buy in 21st century America? On a grand-scale level, the answer, fortunately or unfortunately, is nothing. For individuals at greatest socioeconomic and environmental risk for poor health, however, money may very well buy a one-way ticket toward better health and well-being.

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