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Nurture Net of Nature: Re-Evaluating the Role of Shared Environments in Academic Achievement and Verbal Intelligence

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Abstract

Prominent authors in the behavioral genetics tradition have long argued that shared environments do not meaningfully shape intelligence and academic achievement. However, we argue that these conclusions are erroneous due to large violations of the additivity assumption underlying behavioral genetics methods – that sources of genetic and shared and nonshared environmental variance are independent and non-interactive. This is compounded in some cases by the theoretical equation of the effective and objective environments, where the former is defined by whether siblings are made more or less similar, and the latter by whether siblings are equally subject to the environmental characteristic in question. Using monozygotic twin fixed effects models, which compare outcomes among genetically identical pairs, we show that many characteristics of objectively shared environments significantly moderate the effects of nonshared environments on adolescent academic achievement and verbal intelligence, violating the additivity assumption of behavioral genetic methods. Importantly, these effects would be categorized as nonshared environmental influences in standard twin models despite their roots in shared environments. These findings should encourage caution among those who claim that the frequently trivial variance attributed to shared environments in behavioral genetic models means that families, schools, and neighborhoods do not meaningfully influence these outcomes.

Keywords

Academic achievement; Verbal intelligence; Monozygotic twins; Nature/Nurture

1. Introduction

Three decades of behavioral genetics research largely concludes that shared environments play only a minor role in shaping individual outcomes, and that the appearance of this influence is attributable to gene-environment correlations. For instance, in *The Nurture Assumption*, Harris ([Harris, 1998]) argues that children are principally shaped by their parents through genetic pathways, and that socialization primarily takes place at the peer

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level. Similarly, in *The Limits of Family Influence*, Rowe (1995) argues that socialization research is founded on unsupported assumptions concerning the separability and relative importance of genetics and home environments. By ignoring the dual genetic and environment inheritance processes that potentially shape children's lives, these authors argue that sociologists and other social scientists have confounded environments with genetics, and have accordingly overstated the role of family, school, and neighborhood influence. These conclusions are broadly influential in psychology (e.g., Plomin, et al., 2001, Plomin and Daniels, 1987) and the popular discourse. Although these claims are not identical, these findings are often interpreted to mean that family life negligibly influences children's prospects (Harris, 1998, Rowe, 1995).

These are valid concerns – because genetics are rarely accounted for in sociological research on parental, neighborhood, and school influences on children, if genetic factors are related to shared environments and the outcomes, genetic confounding is a possibility. Because sociological and other social science research frequently concludes that these social environments are major determinants of educational prospects in early childhood (Alexander, et al., 2007, Fryer and Levitt, 2006, KewalRamani, et al., 2007), adolescence (Camara and Schmidt, 1999, Hedges and Nowell, 1999, Kobrin, et al., 2007) and beyond (Elman and O'Rand, 2004, Roscigno and Ainsworth-Darnell, 1999), it is important for sociological researchers to critically examine this literature to evaluate its conclusions.

We argue that shared and nonshared environments exert important influences on the academic achievement and verbal intelligence of adolescents in the United States. The common conclusion that shared environments are inconsequential for these outcomes, we argue, is attributable to two key assumptions of classical behavioral genetic models and related writings which have not received wide attention outside of that field. First, standard behavioral genetic models assume that genetic, shared environmental, and nonshared environmental influences are additive and separable – the *additivity assumption*.[1] Second, these models assume that objectively shared environments operate by making siblings more phenotypically similar – the *homogenizing assumption*. In other words, the homogenizing assumption occurs when shared environmental variance estimates (which measure non-genetic sources of sibling resemblance) are interpreted to reflect the variance explained by the objectively shared environment (factors to which siblings are commonly exposed; Goldsmith, 1993, Rutter, et al., 1999, Turkheimer and Waldron, 2000).[2] Furthermore, ongoing research on gene-environment interactions emphasizes that genetic and environmental influences are frequently non-additive, and research on socialization and the sociology of education emphasizes that objectively shared and nonshared environments are deeply intertwined in both their distribution and their effects. These non-additive processes can both have the result that objectively shared environments serve to differentiate, not homogenize, siblings. Because shared environmental components of behavioral genetic models reflect non-genetic sources of sibling homogeneity, this suggests that negligible

[1]Although behavioral genetics research is frequently concerned with identifying non-additive genetic effects, such as dominance and epistasis, this is not our intended meaning in this case.

[2]Because we wish to address the frequent (but not universal – see Plomin et al. 2013 for a more consistently careful interpretation) conflation of the effective and objective environment, we will not always use the term ‘shared environment’ in the manner in which behavioral geneticists typically do. See below for a full discussion of this issue.

estimates of effectively shared environmental influence may be misleading when they are interpreted to indicate that objectively shared environments are inconsequential.

We support these arguments using a regression model capable of estimating environmental effects net of genetic ones – a monozygotic twin fixed effects model. We use this model and data from the National Longitudinal Study of Adolescent Health (Add Health) to demonstrate: (a) the substantive importance of twin differences in behaviors and attitudes for academic achievement net of genetic influence, and (b) the interactive influence of a wide variety of home environmental characteristics with these nonshared environments. We conclude that both components are important determinants of academic achievement and verbal intelligence.

2. Background

2.1. "The Nature of Nurture"

The claim that shared environments are inconsequential (e.g., Harris, 1998, Rowe, 1995) for most child outcomes suggests that apparent shared environmental effects are in fact due to gene-environment correlations (rGE) whereby individuals with certain genes are more likely to find themselves in certain environments – in other words, rGE is "the nature of nurture" (Plomin, et al., 2013:108). For instance, suppose parents who encourage children's studying have higher genetic aptitudes for academic achievement which their children have partially inherited. These children may earn better grades on average due to their advantageous genetic characteristics whether or not their parents encourage their study habits. In this way, the appearance of an environmental effect may be created when in fact a genetic effect is at work.

2.1.1 Behavioral Genetics Studies of Intelligence—The common view that rGE biases sociological studies of environmental influences is clearly reflected in the contrast between behavioral genetic and sociological studies of intelligence and academic achievement. In contrast to the literature on the sociology of education, behavioral genetic research consistently finds that shared environments are responsible for the little of the overall variation in IQ. For instance, Nielsen (Nielsen, 2006) finds little shared environmental influence on adolescent verbal IQ using the Add Health dataset, as well as large genetic and nonshared environmental influences. Scarr and Weinberg (Scarr and Weinberg, 1978), using measures of the shared environment in a sample of 16-22 year-old adopted and biological children, similarly find negligible evidence for shared environmental effects on IQ. Many other behavioral genetic investigations of this matter have concluded similarly (Brody, 1992, Hunt, 1997). McGue (McGue, 1997) writes that, insofar as there is behavioral genetic differences in opinion on this matter, these are differences of degree (Daniels, et al., 1997, Feldman, et al., 2000). However, in recent years some researchers have examined how heritability estimates vary by major social environments (Boardman, 2009, Rowe, et al., 1999, Turkheimer, et al., 2003) and age (Haworth, et al., 2010).

2.1.2 Behavioral Genetics Studies of Academic Achievement—In contrast to studies of intelligence, the behavioral genetics literature on academic achievement outcomes is less uniform in its conclusions concerning the relative importance of genetic and

effectively shared and nonshared environments on academic achievement outcomes. As with studies of intelligence, many behavioral genetic studies of academic achievement find that genetic and nonshared environmental influences are responsible for the vast majority of the variation in academic achievement. For instance, in a study of nine-year-old twins, Haworth and colleagues (Haworth, et al., 2008) finds that genetics are responsible for 60% of the variation in science achievement, with nonshared environments responsible for nearly all the remaining variation. Other researchers have reported similar findings (Nielsen, 2006, Walker, et al., 2004).

However, recent research on this topic has increasingly found evidence for substantial effectively shared environmental influences on academic achievement. For instance, Lemelin and colleagues (Lemelin, et al., 2007) finds that the shared environment accounted for a plurality of the variance in four domains of cognitive school readiness in a sample of five-year-old twins, although the genetic and nonshared environment components also accounted for a substantial portion of the variance therein. Similar findings have been reported by a number of others (Petrill, et al., 2010, Thompson, et al., 1991). Studies of measured shared environmental effects on academic achievement also support this conclusion. For instance, Sacerdote (Sacerdote, 2007) studied a sample of Korean American children who were adopted, reasoning that correlations of children's outcomes with parental characteristics could only be due to shared environmental variance. This study found that children assigned to better educated and smaller families went further in school, and that objectively shared environments accounted for 14% of the variation in educational attainment. The proportion of variance attributable to the shared environment, however, appears to decrease with age (Dunn and Plomin, 1990, Walker, et al., 2004) in twin decomposition models.

2.1.3: Challenges for Sociology—These findings pose a challenge for researchers in the sociology of education, who are frequently concerned with environmental effects on academic outcomes. This literature has long identified a wide range of influences shared by siblings which are robustly associated with academic achievement and IQ outcomes in childhood and adolescence, such as family socioeconomic status (e.g., Fuligni, 1997, Grodsky, et al., 2008, Laureau, 2002, Laureau, 2003, Menaghan and Parcel, 1995), parental structure (e.g., Acs, 2007, Haveman and Wolfe, 1994, Manning and Lamb, 2003), sibling structure (e.g., Guo and Van Wey, 1999, Loeb and Bound, 1996, Steelman, et al., 2002), and school and neighborhood characteristics (e.g., Bradley and Taylor, 1998, Brooks-Gunn, et al., 1993, Coleman, et al., 1966, Crowder and South, 2003, DeLuca and Dayton, 2009, Jacob, et al., 2008). Researchers concerned with these matters must consider how we can leverage genetic and environmental data to better understand the validity of the “nature of nurture” and genetic confounding arguments.

2.2: The Importance of Nonshared Environments

It is important to note, however, that behavioral genetic argument typically concedes an important role for the nonshared environment – i.e., variance not explained by genetic or shared environmental factors (see below for further details). Research by Scarr and Weinberg (Scarr and Weinberg, 1978), Nielsen (Nielsen, 2006), Brody (Brody, 1992), and Hunt (Hunt, 1997) find both large genetic component of academic achievement and

substantial nonshared environmental effects. This is such a cornerstone of behavioral genetic theory, in fact, that it is a basis of Turkheimer's three laws of behavioral genetics: “First Law. All human behavioral traits are heritable. Second Law. The effect of being raised in the same family is smaller than the effect of genes. Third law. A substantial portion of the variation in complex human behavioral traits is not accounted for by the effects of genes or families” (Turkheimer, 2000:160).

Certainly behavioral geneticists and social scientists agree on the importance of factors not shared by siblings that influence academic achievement and are unlikely to be wholly genetic in origin, such as educational aspirations and expectations (e.g., Frank, et al., 2008; Muller and Ellison, 2001), academic effort (e.g., Aksoy and Link, 2000; Stinebrickner and Stinebrickner, 2008), eating breakfast (e.g., Pollitt and Mathews, 1998); delinquent behaviors (e.g., Staff, et al., 2008; Townsend, et al., 2007), peer influences (e.g., Frank, et al., 2008; Mouw and Entwisle, 2006), and extracurricular activities (e.g., De Graaf, et al., 2000; Guest and Schneider, 2003), and numerous other factors. As such, although this paper will assess nonshared environmental influences on verbal intelligence and academic achievement, the focus of the analysis and discussion will be placed squarely on the role of shared environmental influences.

2.3: Behavioral Genetics Methods: A Conceptual Overview

2.3.1: The Basics—To understand these findings and how they can best be evaluated, some background is in order. Behavioral genetics research is distinguished by its reliance on information concerning the genetic and environmental similarity between pairs of (non)relatives to separate genetic and environmental factors influencing an outcome of interest. Although much research uses adoption designs (Loehlin, et al., 2007) as well as other approaches, the workhorse of behavioral genetics research is the twin decomposition model (Boomsma, et al., 2002; Bouchard and Propping, 1993; Martin, et al., 1997; Plomin, et al., 2001).

This research strategy attempts to separate influences on an outcome variable into three components – genetic (heritability), shared environmental, and nonshared environmental influences. To fix ideas, define r_{MZ} as the correlation in a trait between monozygotic twins and r_{DZ} as the correlation in a trait between dizygotic twins. Following Loehlin (Loehlin, 1992), the correlation between MZ twins may be decomposed such that $r_{MZ} = h^2 + c^2$ (representing genetic and shared environmental components respectively) and the correlation for DZ twins may similarly decomposed, but as $r_{DZ} = ½h^2 + c^2$ because DZ twins have only half the genetic relationship by common descent of MZ twins. Solving terms, heritability may then be defined as $h^2 = 2(r_{MZ} – r_{DZ})$, and the shared environmental component as $c^2 = r_{MZ} - h^2$. The remaining variance in the trait is then labeled as the ‘nonshared environmental’ component e^2 (which also contains measurement error). In words, heritability is defined as twice the difference between MZ and DZ twins’ correlations, shared environment is any remaining non-heritability similarity between MZ twins, and the nonshared environment is a residual term.

2.3.2: The Equal Environments Assumption—The most commonly discussed, and probably least consequential, assumption of twin decomposition models is the equal environments assumption (EEA). The EEA requires that one view the comparison of MZ twins and DZ twins' similarity as a kind of natural experiment in which genetic relatedness varies and environmental similarity is known and fixed (as in comparisons of MZ and DZ twins to obtain heritability estimates), or in which genetic relatedness is fixed and environmental similarity varies (as in comparisons of children in which the mother gave some up for adoption and reared others). A number of authors (e.g., Horwitz, et al., 2003) have challenged the validity of these assumptions. MZ twins, they argue, do not inhabit the same social niche as DZ twins – compared to the latter, MZ twins' greater social and physical similarity results in a broader array of differential influences on their successes than may be attributed to their greater genetic similarity. Therefore, to the degree that these differential social niches also influence the outcomes studied, behavioral genetic methods which assume otherwise will tend to overstate genetic influences (Guo, 2005, Walker, et al., 2004).

However, a number of researchers (Conley, et al., 2013, Klump, et al., 2000, Koenig, et al., 2010, Walker, et al., 2004) have investigated whether this assumption is violated for different phenotypes, and usually find that it is not. To bias the model, two violations of the EEA must be met in a given study: (a) MZ and DZ twins must have differential similarity in key environmental measures; and (b) these environmental measures must be linked to the phenotype being studied. Most research critiquing behavioral genetics research on the basis of the EEA (Horwitz, et al., 2003) focus only on the first requirement while ignoring the second. Generally, any differences documented are insufficient to bias the analysis (Klump, et al., 2000, Koenig, et al., 2010). Nonetheless, re-evaluating the claims that home environments are inconsequential for academic achievement using methods that do not require the EEA will yield greater confidence in the results.

2.3.3: The Additivity Assumption—Another key assumption in these models is that the variance in a given outcome may be decomposed additively. In contrast, high profile work in the last ten years has suggested that many individual-level outcomes are the results of gene-environment interaction processes (Boardman, 2009, Caspi, et al., 2003), which cannot be directly accounted for in this framework. Not doing so could also overstate the influence of genetic factors in behavioral genetic models (Burt, 2009). Although behavioral genetics researchers have examined non-additive genetic models such as dominance (ADE) models (e.g., Maes, et al., 1997), gene-gene interactions (epistasis or emergenesis, e.g., Lykken, 2006, Purcell and Sham, 2004), and heritability-by-environment models (e.g., Boardman, 2009, Turkheimer, et al., 2003), these models have not been employed to examine the non-additive effects of objectively shared and nonshared environments. In contrast, modern socialization research adopts the ecological approach that the effects of families, schools, neighborhoods, and peers are fundamentally interdependent and causally related (Collins, et al., 2000). For instance, parents influence children's networks (Brown, et al., 1993, Parke and Bhavnagri, 1989) as well as their susceptibility to peer influence (Devereux, 1970, Fuligni and Eccles, 1993, Mounts and Steinberg, 1995). These processes also threaten the validity of additive twin decomposition models. As addressed below, this limitation of

previous behavioral genetics research is likely attributable to its analysis of effective environments, which cannot interact, rather than objective environments, which can.

Practically speaking, of course, it is impossible to correctly specify a priori all interrelationships between all relevant characteristics of the genome and different types of environments. The key point here is that twin model results on complex phenotypes such as academic achievement and intelligence should be viewed as informative starting points for further research (while bearing their limitations in mind), rather than the definitive word on these matters.

Furthermore, this is no mere methodological nuance – to assume that genetic, shared environmental, and nonshared environmental effects are additive and independent is a stark theoretical claim about the etiology of intelligence and academic achievement, which contrasts with much social scientific research highlighting complex interplay between these determinants of academic and intelligence outcomes (Maccoby, 2000). Although even the most ardent proponents of behavioral genetics will surely characterize this as a useful simplification of a very complex system, models based upon simplifications may cease to be useful when they are erroneously interpreted.

Figure 1 illustrates these points, mapping out some conceptual linkages between a phenotype and genetics, the shared environment, and the nonshared environment. Most behavioral genetics research underlying the claim concerning the unimportance of the shared environment assumes that only a subset of these pathways potentially exist: the main effects of genetics and the nonshared environment, and the correlations of genetic factors with the shared and nonshared environments. In contrast, most social science research emphasizes that environments are jointly distributed such that shared and nonshared environments will often be strongly correlated. Furthermore, socialization theory (Collins, et al., 2000) emphasizes that the effects of the shared and nonshared environment on a phenotype are interdependent, with the effects of sibling-specific environments depending on family-level environments and vice versa. Finally, ongoing research on gene-environment interplay emphasizes not only the importance of gene-environment correlations, but also gene-environment interactions, where environmental effects of both types may be genetically-dependent (and vice versa). In short, the structure that the twin decomposition model places on a dataset strongly implies a theoretical understanding of how environments and genetics influence a phenotype, to the exclusion of other pathways of influence.

2.3.4: The Homogeneity Assumption—Importantly, the behavioral genetics distinction between genetic, shared environmental, and nonshared environmental influences in twin decomposition models is concerned with the nature of environmental influences on siblings, not the nature of those environments. Shared environments in this approach are defined as nongenetic influences which make siblings more similar; nonshared environments are defined as nongenetic influences which make siblings more dissimilar. This form of the shared-nonshared environment distinction is known as the effective environment (Goldsmith, 1993, Rutter, et al., 1999, Turkheimer and Waldron, 2000). This definition is in contrast to the popular interpretation of shared environments as those to which all members of a sibship are exposed and nonshared environments as those to which only one member of

a sibship is exposed, which is known as the objective environmental distinction (Goldsmith, 1993, Rutter, et al., 1999, Turkheimer and Waldron, 2000). Theoretically, it is a strong assumption to assert that effective environmental definitions will correspond to objective environmental ones. This is the heart of the homogenizing assumption.

This distinction may seem trivial. However, the argument that home environments do not consequentially influence development frequently erroneously assumes that these two conceptualizations of the environment are equivalent (Turkheimer and Waldron, 2000). This is not obviously the case – shared environmental effects in twin decomposition models reflect any non-genetic influences that make twins more similar, which may or may not include any given elements of the objectively shared environment. For instance, home environments could have the effect of making twins less similar (Turkheimer and Waldron, 2000), perhaps via sibling niching processes (Feinberg, et al., 2005) and this effect would be attributed to the nonshared environment although this was an effect of the objectively shared environment. Importantly, effects of the objectively shared environment which operate by modifying the effects (Collins, et al., 2000) of the objectively nonshared environment in a way which made twins more dissimilar would also be attributed to the nonshared environment. By treating the effective and objective environments as equivalent, these arguments have contended that objectively shared environments are inconsequential on the basis of studies of the effective environment.

Given these ambiguities, any analysis which seeks to make claims about the effects of the objectively shared environment (including key relations and institutions such are parents, neighborhoods, and schools) should be supported by analyses of the objective, not effective, environment while still accounting for genetic influence. To date, this has not been a common practice in the behavioral genetics research tradition on which these claims have been founded.

3. Analytical Strategy

The analytical strategy employed in this study consists of two stages, corresponding to our interests in identifying the effects of shared and nonshared behavioral and environmental characteristics on academic achievement, net of the influence of genetic factors. (However, our focus is on the objectively shared environment.) Like many behavioral genetics researchers and others (Ashenfelter and Krueger, 1994, Behrman, et al., 2011, Branigan, et al., 2013, Taubman, 1976), we analyze a dataset of MZ twins, but employ a somewhat different strategy than has been typical, focused on the effects of objectively-measured shared and nonshared environments academic achievement and intelligence.Because it focuses on the effective, not objective, environment, standard behavioral genetic methods are not appropriate for doing so.

Instead, our approach to this important problem consists of two related tasks. The first task in this analysis is to identify the academic achievement effects of objectively nonshared variables – for instance, individual-specific behaviors, social environments, and attitudes. These correspond to the main effects of the nonshared environment depicted in Figure 1. In doing so, care must be taken to identify the effects of these variables free from spurious

influences. To date, the best method by which to do this is fixed effects models, wherein the stable characteristics of a cluster of observations are partialled out of the parameter estimation process by exploiting within-cluster variation in predictor and outcome variables only.

To formalize this discussion, consider a standard model for the academic achievement of two identical twins:

$$\boldsymbol{Y_{ij}}=\alpha+\beta_1\boldsymbol{X_j^s}+\beta_2\boldsymbol{X_{ij}^u}+\beta_3\boldsymbol{g_j}+\boldsymbol{e_{ij}} \quad (1)$$

Following standard behavioral genetic decomposition assumptions, $\mathbf{Y_{ij}}$ represents academic achievement, $\boldsymbol{X_j^s}$ represents a vector of shared environmental factors, $\boldsymbol{X_{ij}^u}$ represents a vector of nonshared environments, $\mathbf{g_j}$ represents genetic factors (which are shared by MZ twins), i indexes individuals, and j indexes twin groups.

It is also possible to extend this model to incorporate interactions between these components. Although in gene-environment interplay research this would typically involve an interaction between **g** and one of the **X** environmental terms, at present our primary interest is in the interaction of $\boldsymbol{X_{ij}^u}$ and $\boldsymbol{X_j^s}$ (the nonshared and shared environmental factors), resulting in the following model:

$$\boldsymbol{Y_{ij}}=\alpha+\beta_1\boldsymbol{X_j^s}+\beta_2\boldsymbol{X_{ij}^u}+\beta_3\boldsymbol{g_j}+\beta_4\boldsymbol{X_j^s}\boldsymbol{X_{ij}^u}+\boldsymbol{e_{ij}} \quad (2)$$

Turning back to equation (1), because genes and home environments are shared among identical twins, in a standard fixed effects model these terms are subtracted out, leaving only:

$$\left(\boldsymbol{Y_{ij}}-\overline{\boldsymbol{Y_j}}\right)=\alpha'+\beta_2\left(\boldsymbol{X_{ij}^u}-\overline{\boldsymbol{X_{ij}^u}}\right)+\boldsymbol{e_{ij}}' \quad (3)$$

where $\overline{\boldsymbol{X_{ij}^u}}$ is the mean value of measured individual environments in twin cluster j. Thus, this model uses only information on which twins differ to identify effects on academic achievement and verbal intelligence.

Because we are analyzing a sample of MZ twins, the method just described enables us to identify the effects of objectively nonshared factors on academic achievement while controlling for genetics and other objectively shared influences (eliminating the r(G,NSE) and r(SE,NSE) pathways in Figure 1 from the model). However, our primary goal is to estimate the influences of factors objectively *shared* by twins. Ideally we could directly estimate these effects as we do with those of objectively nonshared variables, but because by definition objectively shared environments do not vary within twin pairs, a standard fixed effects model of shared environmental effects cannot be estimated. Instead, we argue (as does Allison 2005) that statistically interacting the effects of nonshared variables with those of shared variables in a fixed effect model partially achieves this objective. Substantively, this amounts to testing a single pathway by which the shared characteristics of twins and their environments might influence academic outcomes – by modifying the influence of nonshared factors. This corresponds to the SE×NSE pathway in Figure 1.

To fix ideas, the following equation extends equation (2) to incorporate the environmental interaction element of our analytical strategy into a fixed effects regression:

$$\left(\boldsymbol{Y_{ij}} - \overline{\boldsymbol{Y_j}}\right) = \alpha' + \beta_2' \left(\boldsymbol{X_{ij}^u} - \overline{\boldsymbol{X_{ij}^u}}\right) + \beta_4 \left(\boldsymbol{X_{ij}^u X_j^s} - \overline{\boldsymbol{X_{ij}^u X_j^s}}\right) + \boldsymbol{e_{ij}}' \quad (4)$$

where $\left(\boldsymbol{X_{ij}^u X_j^s} - \overline{\boldsymbol{X_{ij}^u X_j^s}}\right)$ is the difference between twin i's interaction term and the cluster mean thereof. This term is not eliminated in a fixed effects model because it varies as a result of the nonshared portion of the interaction term. (As with main environmental effects, when the nonshared environmental variable has no within-cluster variance, this term will equal 0.) Fixed effects models of this type still rely only on information which varies within clusters to identify effects, eliminating potentially biasing effects from genetic factors. Succinctly put, equation (4) estimates the interactive effect of shared and nonshared environmental variables within a twin fixed effects model.

To illustrate, consider a twin pair who shared college-educated parents but study for different amounts of time (say, 3 and 5 hours per week). When the binary variable for parental college education is multiplied by their respective study habits values, these values are still not identical, and therefore will not be fully subtracted out when all variables are differenced from the cluster-specific mean – if parental college education is expressed as a binary variable, the interaction of study hours and whiteness for white twins will equal their study hours. Since $3 \neq 5$, this interaction will not drop out of the equation when there is within-pair variance in the nonshared variable. When the effect of this interaction term is estimated for the full sample of MZ twins, the effect will estimate the differential effect of studying for white twins compared with twins from other races or ethnicities on the dependent variable.

This analytical strategy offers several virtues. First, unlike twin decomposition models it requires no assumptions concerning the relative environmental similarities of MZ and DZ twins (i.e., no equal environments assumption). Second, this method does not require the homogenizing assumption or its equivalent – instead, environments are deemed shared or nonshared based solely on whether both twins are subject to them (i.e., the objective environment definition). Third, this approach permits direct interrogation of the additivity assumption underlying standard twin decomposition and most other behavioral genetic models. If the SE×NSE pathway in Figure 1 is widely influential on academic achievement and verbal intelligence, this could help to explain the apparent inconsequentiality of effectively-shared environments in models that make no allowance for this pathway.

3.1 Differentiating Objectively Shared and Nonshared Environments

Although the distinction between objectively shared and nonshared environments has thus far been assumed to be unproblematic, in practice these are frequently thorny distinctions. Family and school environments do not merely exert their influence on passive recipients – adolescents are both influenced by and construct their social worlds. With this caveat, however, we distinguish SEs and NSEs according to the distinctions typically used in the behavioral genetics literature on objective environmental distinctions – whether they are both equally subject to these environments. Non-relational characteristics of families, neighborhoods, and schools all meet this requirement. Dyadic relationships with others in

these environments do not, and therefore are treated as objectively nonshared environments along with other factors that differentiate twins in an objectively nonshared manner, such as attitudes and aspirations, social network characteristics, and academic, health, and delinquent behaviors. In borderline cases, variables with relatively high within-pair variability are considered nonshared.

This distinction does *not* mean that the objectively nonshared environments of twins cannot be correlated – indeed, it would be surprising if they were not. However, in our fixed effects model, we will identify the effects thereof using only the variance that differs within pairs.

3.2 Testing the Effects of Nonshared Variables

First, we conduct bivariate twin fixed effects regression analyses to capture the association of each measured, objectively nonshared independent variable with both dependent variables. Subsequently, we reassess the effects of these variables in multivariate[3] twin fixed effects regression analyses (in which the model is specified using all nonshared predictors) in order to assess the likelihood that these effects are spurious due to associations with other measured, nonshared variables.

For the bivariate estimates, we evaluate the statistical significance of each effect in the usual manner. For the multivariate estimates, however, we also conduct a test of the statistical significance of differences between the bivariate and multivariate regression coefficients. In other words, the bivariate fixed effects coefficient for the variable becomes the null hypothesis to be evaluated by the significance test for the multivariate analyses. However, since this is not the usual practice in sociological analyses (but see Fowler and Christakis, 2008, Freese and Powell, 2003), we also provide statistical evaluations of multivariate regression coefficients' difference from zero.

3.3 Testing the Effect of Shared Variables

We test the modifying effects of objectively shared variables using likelihood ratio tests by comparing the fully specified nonshared model (equation 3 above) to a model in which all nonshared variables are interacted with a single objectively shared variable (equation 4 above), and do so separately for each shared variable. Because the large number of tests this strategy produces raises multiple testing concerns, we assess the statistical significance of these tests using Bonferroni-adjusted critical p-values. To the degree that objectively shared environmental characteristics improve the fit of the interactive model compared to the multivariate nonshared model, these characteristics are deemed to significantly influence the effects of nonshared environmental variables net of genetics and multiple testing adjustments. Improved model fit is calculated using a likelihood ratio test comparing the multivariate nonshared environmental model (equation 3, presented in Table 3) with the interaction model in question (equation 4, presented in Table 4). Finally, the total effect of the shared environmental variable is calculated by taking the model-implied derivative of the dependent variable with respect to the shared environmental variable. In the interests of

[3]Although in behavioral genetics and related fields it is common to use the term 'multivariate' to refer to analyzing multiple dependent variables, in this paper we use this to refer to multiple independent variables, in accordance with the usual practice in sociology.

interpretability, this derivative is converted to its absolute value, then standardized: by the standard deviations of the dependent and shared environment variables in the case of continuous shared environments, and by the dependent variable only for binary shared environments. These effects are interpretable in terms of relative effect sizes only.

3.4 Fixed Effects and Within-Pair Variability

One final methodological note is in order. A major concern in this identification strategy is the possibility that there is insufficient within-pair variation to use a fixed effects model on this sample of MZ twins. Appendix A provides a descriptive analysis of this possibility, and shows that there is substantial variation on all nonshared variables in the twin sample.

4. Data and Methods

4.1 Add Health

The data source for this analysis is the National Longitudinal Study of Adolescent Health (Add Health). Add Health is a school-based study of the health-related behaviors of adolescents in grades 7-12 in the United States. The school sample was stratified by region, ethnic mix, size, urbanicity (urban/suburban/rural), and school type (public/private/parochial). In 1994, more than 90,000 adolescents from 134 schools completed the in-school questionnaire. All students who completed an in-school questionnaire, as well as those who did not complete a questionnaire but who were listed on a school roster, were eligible for selection into the in-home sample. The first wave of the in-home survey interviewed a total of 20,745 adolescents from May through December of 1995. The first wave in-home sample includes a core sample of 12,105 individuals representative of adolescents in grades 7 to 12 during the 1994-1995 school year in the United States. The in-home sample also includes over-samples of genetically related adolescents. At the time of the in-home interviews, a parental questionnaire was completed by one of the adolescent's parents (usually the mother) or guardians.[4]

During the collection of wave 1 of Add Health, investigators asked every adolescent in their sample whether they were a twin and, if so, whether they were an identical twin. On this basis, Add Health oversampled for identical twins as a part of their design, recruiting every identified MZ twin they encountered into their sample. The result was a subsample of 578 identical twins (289 pairs) within the much larger dataset. Although unrepresentative of the overall adolescent population, these twins hail from a large number of different schools and regions of the country. All measures are described in detail in Appendix B.

4.2 Measures

Following the sociology of education literature on academic achievement, we divide the determinants into a number of categories: behavioral and attitudinal characteristics, extracurricular activities, network structure, network content, demographic and family structural characteristics, neighborhood features, school characteristics, parental characteristics, and social support and parenting variables. In the objective shared-nonshared

[4]See http://www.cpc.unc.edu/projects/addhealth/design for further detail on the design of Add Health. Accessed August 31, 2009.

distinction we employ, the first four categories are considered nonshared and the last five are considered shared. Therefore we identify a series of measures available in Add Health which fall into each of these categories, treating these categories as sources of independent variables and employ differences in academic achievement and verbal intelligence as dependent variables.

4.2.1 Dependent Variables: Academic Achievement and Verbal Intelligence— To assess the influence of home environments on academic achievement, we analyze the determinants of self-reported GPA and the Peabody Picture Vocabulary Test score, a measure of verbal intelligence. GPA was constructed from self-reported mean letter grades for English, mathematics, science, and social studies in the past year. The use of self-reported grades has consistently been found to be a strong indicator of current academic achievement, with an average correlation with school reported GPA of 0.84 (Kuncel, et al., 2005). We also employ a measure of verbal intelligence, the Add Health Peabody Picture Vocabulary Test (PPVT), an abridged version of the full test developed for use in Add Health. Previous research has shown that PPVT scores are positively correlated with other intelligence scores for both children (Smith, et al., 1991) and college-age adults (Bell, et al., 2001).

4.2.2 Nonshared Measures—We draw upon a number of objectively nonshared measures to analyze their role in academic achievement differences between twins, motivated by the sociology of education literature. We employ five different measures of educationally-relevant attitudes and behaviors: adolescent college aspirations and perceived probability of attendance, parental educational aspirations for the adolescent, school effort, and skipping breakfast.[5] We also construct three different measures of delinquent behavior: an index of illegal substance use, a non-violent delinquency index, and a violent behavior index. We also employ two different measures of students' extracurricular school activities: a count of all clubs in which they participated, and a count of all academic clubs in which they participated.

Finally, we constructed a number of measures based on the Add Health in-school ego network data. Adolescents' network structures are captured as their network density, Bonacich centrality, and popularity (the number of friendship nominations they received). Network content measures capture nominated friends' characteristics and interactions with the adolescent, and include an index of average social interactions with one's friends, and a series of ego network averages (for school effort, the school difficulty index, delinquency, college probability, and perceived middle class income probability) and heterogeneity (in race and age).

4.2.3 Shared Measures—To assess the role of home environments in academic achievement, we draw upon a number of measures of parental characteristics, family structure, and parental behaviors. Family SES is assessed in two ways, using an indicator for

[5]Although some of these characteristics do not reflect the usual sociological understanding of an 'environment' as something external to the individual, they do meet the behavioral genetics definition of 'environments' as sources of phenotypic variation not attributable to genetic factors.

white collar parental employment and highest reported parental education. Family structure is measured in several ways using household roster data: a variable indicating whether one's primary parental respondent is married; the twins' place in the sibling birth order; twins' number of full siblings; siblings' age standard deviation; and the number of full sisters. Parenting and social support characteristics are also assessed using a variety of measures: a sum of two indicators for positive (and separately, negative) parent-child relationship characteristics; parental school involvement; parental control over children's activities; and an adolescent reported index of social support.

To assess the contributions of school characteristics, we include administrator-reported measures of school size, a private school indicator, an index of poor school quality, and the percentage of graduating students attending two- or four-year colleges. Additionally, based on student reports we construct two other school-level measures: an index of average school social cohesion and school-level racial network segregation. Finally, we include five measures of neighborhood characteristics: an index of poor neighborhood quality, the Census tract unemployment rate, the census tract standard deviation of home values, a parental measure of neighborhood social monitoring, and an indicator that the neighborhood is urban.

4.3 Missing Data

Due to normal survey rates of missingness and the small initial sample size for our analytical sample, we elected to multiply impute missing data using the ice (Royston 2005) program in Stata 10. Additionally, we drew on information from both identical twins to resolve differential twin reports on putatively shared influences, as indicated in Appendix B. When reports of shared categorical variables varied within twin pairs, both twins' values for that variable were assigned to one or other's reported value at random.[6] When reports of continuous or ordinal variables shared by twins differed, values for such variables were assigned to the average of their differential reports.[7]

5. Results

Table 1 provides descriptive statistics (mean, standard deviations, and range) for the objectively nonshared variables used in this study for the analytical, multiply imputed sample of MZ twins, side-by-side with the corresponding statistics for the full Add Health sample (minus the MZ twins). Table 2 provides the same statistics for objectively shared variables used in this study. Appendix A discusses supplementary analyses, reported in Tables 1 and 2, which reveal that the twin sample is substantively very similar to the full Add Health sample, suggesting that our results are unlikely to be heavily influenced by our analytical restriction to identical twins only. The variables for which t-tests indicated statistically significant differences between MZ twins and other adolescents are indicated by

[6]This was relatively rare, and only conducted on variables which are certainly shared among identical twins – gender, race, parental highest occupation, parental education, parental welfare/unemployment status, neighborhood urban status, neighborhood modal education, neighborhood unemployment, parental marital status, school size, private school status, and immigration status.

[7]Although some of these differential values result from differential imputation of missing values, in other cases this is due to differential measured responses. Siblings have been shown to differentially report such variables as how many siblings they have, and their parents' education – see Conley 2004 for a discussion.

an asterisk in Tables 1 and 2. We organize the presentation of these results around the answers to a series of key questions.

5.1 What are the effects of factors not shared by twins on academic achievement and ability?

Table 3 provides the results of a series of fixed effects models examining the association of our nonshared environmental variables with GPA and PPVT outcomes in the MZ twin subset of the Add Health data. The effects of each nonshared variable on both outcomes were modeled separately in a series of bivariate models. The bivariate results indicate that substance use is negatively related to GPA, as is delinquency, and that college aspirations and academic club participation are positively related to GPA. Because these coefficients are derived from an analysis of within-pair differences on GPA and these variables from identical twins, these effects are not attributable to unobserved heterogeneity in genetics or shared environments. No statistically significant bivariate associations with PPVT outcomes were found, however.

5.2 Are these associations spurious due to with the influence of other nonshared covariates?

Because these associations could still be spurious due to other, nonshared factors, we also fit a multivariate model including all of these nonshared predictors simultaneously. The association of substance use, non-violent delinquency, college aspirations, and academic clubs are not statistically significantly different from the bivariate estimates. However, the effects of delinquency, college aspirations, and particularly substance use are attenuated in the multivariate model, while the effect of academic clubs is enhanced. However, none of these coefficients are statistically significantly different from zero in the multivariate model. Similarly to the bivariate models, no coefficients of nonshared variables were found to have a statistically significant association with PPVT outcomes.

5.3 What are the moderating effects of shared environmental characteristics?

As discussed above, we analyzed the relevance of objectively nonshared variables for academic outcomes by interacting the effects of all objectively nonshared variables with these objectively shared variables, with a separate such model for each nonshared variable. The fit of these models were then compared to that of the multivariate nonshared models for the same dependent variable (depicted in Table 3). To clarify, when we depict shared environmental effects in Table 4, we are reporting the p-value associated with a likelihood ratio test comparing (a) a restricted model in which all nonshared, but no shared, variables were included in the independent variable specification, and (b) an unrestricted model in which all nonshared variables are simultaneously interacted with the shared variable. These effects are expressed as the absolute value of the standardized derivatives in all models.

Finally, because the large number of models this procedure produced raises multiple testing concerns, these likelihood ratio test results were compared to a Bonferroni-adjusted critical p-value. The results of this procedure are clear – many shared variables significantly modify the effects of nonshared variables on GPA and PPVT, even among MZ twins who share genes and many facets of the environment.

5.3.1 Demographic and Family Structure Effects—Several demographic and family structure variables have significant moderation effects on GPA and PPVT. Age, birth order, household size, and sibling age dispersion are all statistically and substantively significantly associated with both GPA and PPVT through these moderation effects. Furthermore, one's number of full siblings, non-white status, and immigrant status statistically and substantively significantly moderate the nonshared effects on GPA, but not PPVT. All of these variables have statistically significant moderation effects on GPA, and all but age and birth order do so for PPVT, as well. Overall the shared demographic characteristics appear to broadly influence GPA and PPVT scores through this interactive mechanism.

5.3.2 Neighborhood and School Effects—Furthermore, a number of neighborhood and school characteristics significantly modified the fit of the multivariate nonshared models as well. All measured neighborhood variables – the bad neighborhood index, neighborhood home value dispersion, social capital, unemployment, and urbanicity – statistically significantly modified the effects of nonshared variables on GPA. Similarly, all five neighborhood characteristics statistically significantly modified the effects of nonshared variables on PPVT. Finally, all of these estimated effects were substantively significant as well, with the exception of urbanicity moderator effects for PPVT. The effects for home value dispersion and unemployment percentages are not displayed because the estimated derivatives were larger than the ranges of the dependent variables in both cases.

5.3.3 Parental Characteristics Effects—Parental characteristics show evidence of important moderation effects as well. All estimated effects were statistically significant for both GPA and PPVT. Furthermore, a number of these associations are substantively large, especially parental health status and (for GPA) unemployment/welfare status. Parental risky behaviors show evidence of moderately substantively significant moderation effects, as well.

5.3.4 Social Support and Parenting Effects—Finally, the effects of parenting and social support have similarly broad effects on GPA and PPVT. All estimated effects are statistically significant, and several are substantively large. Positive parental relationships have a substantively large moderating effect on GPA, and negative parental relationships have a smaller but still substantial moderating effect on PPVT. Parental school involvement and control is similarly substantively associated with GPA, as is the social support index. In sum, parental and other social support and relationship quality appears to exert substantial moderating effects on GPA, and to a lesser degree on PPVT.

6. Discussion and Conclusion

It is a common conclusion in behavioral genetics that shared environmental effects on many important phenotypes, such as academic achievement and intelligence, are inconsequential. We argue that this is not the case, and that this conclusion stems from the sometimes uncritical use of a model that assumes that shared, nonshared, and genetic effects are additive, and that is often interpreted in a way that conflates the effective and objective environments. Although the models employed in this analysis are not suitable replacements for twin decomposition and related models for general purposes, they do show strong

interactive effects of measured shared and nonshared environments on academic achievement and verbal intelligence.

Theoretically, these results emphasize the complex web of causation that lead to the development of important outcomes like academic achievement. The key lesson from sociology and social psychology for this conclusion is that no environment (or gene) operates in a vacuum. For instance, family socioeconomic status is strongly correlated with the type of school one attends, the type of neighborhood in which one lives, and the sorts of peers by which one is surrounded. In addition to these features of the environment being interrelated, these characteristics also have interdependent effects on academic outcomes. Finally, it is likely that many of these environmental processes are interrelated and interdependent with genetic factors, as well, as extensively demonstrated in the literature on gene-environment interplay.

Furthermore, these results show evidence of theoretically-interesting patterns for the moderating effects of shared environmental characteristics. Since these are standardized coefficients, we can assess their effect sizes following Cohen's (1992) guidelines for correlation coefficients, broadened here to classify effects <0.2 as small, 0.2-0.4 as medium, and >0.4 as large. With these guidelines, some interesting patterns emerge. First, many shared environments exert large moderating effects on both verbal intelligence and academic achievement: birth order, household size, sibling age dispersion, and school quality, and several others show evidence of medium or large effects on both: age, neighborhood quality and social capital. Therefore we can conclude that key characteristics of families, schools, and neighborhoods exert substantively important moderating influences on both academic achievement and verbal intelligence. Still other shared environments exert no substantively important moderating effects: the sibling gender composition, parental marital status, school social cohesion, parental education, and parental risky behaviors. These variables may exert important main effects on these outcomes, but show little evidence of such influence through moderation of nonshared environments in this analysis.

Additionally, several shared environmental characteristics have substantively important moderating associations with one of our dependent variables, but not the other. Number of full siblings, non-white status, immigrant status, urban neighborhood, school size, positive parental relationship, parental school involvement, and social support all significantly moderate nonshared effects on GPA, but not PPVT. Only negative parental relationships show the opposite pattern of substantively moderating nonshared effects on PPVT but not GPA. Combined with the above observations, this suggests that, while many shared environments influence both verbal intelligence and academic achievement (perhaps influencing the latter through the former), many other shared environmental characteristics influence academic achievement alone, without corresponding associations with verbal intelligence. One can interpret these effects as combining with nonshared environments to structure the degree to which adolescents reach their academic potential. Thus, key shared environments such as family structure, race and immigration status, neighborhood and school characteristics, and parenting behaviors significantly moderate how nonshared environmental characteristics influence academic achievement without influencing verbal intelligence.

Variance decomposition models remain a compelling method to study the major parameters of key sociological and psychological outcomes for many purposes. Methodologically, however, they require assumptions on the relative environmental similarity of MZ and DZ twins and the additivity of environmental influences, and do not directly assess the influence of measured covariates on outcome variables. Theoretically, the frequent conflation of effective environments (how environments make siblings more or less similar) and objective environments (whether siblings are subject to the same environmental influences) is responsible for the conclusion that home and other objectively shared environments do not consequentially shape children's lives. Although we do not offer our statistical model as its replacement for general applications, we argue that our approach represents an alternative to variance decomposition methods for identifying the effects of objectively measured environmental factors net of genetic ones. Importantly, this method can identify the influence of nonshared and (interactively) shared environments on these same outcomes while controlling for genetic influences. Furthermore, this model can do so without the aid of the limiting assumptions of the decomposition model.

Nonetheless, the present study has a number of limitations of its own. Most notably, the limited size and representativeness of the sample restrict the external generality of our findings. Additionally, all associations studied in this analysis were cross-sectional, which limits our knowledge of the processes by which these associations arise.

Furthermore, although we control for the potential effects of genetics, our estimates of the effects of nonshared variables may nonetheless be biased by confounding with unmeasured variables. However, because genetic and other shared influences are fully controlled in this model, only unmodeled environmental or behavioral factors could produce such bias. Therefore, even this result would confirm the importance of nonshared influences (i.e., behaviors, attitudes, and peer groups) for academic achievement. Similarly, our analyses of the interactive effects of objectively shared and nonshared environments on GPA and PPVT may be subject to some degree of omitted variable bias, but this result would similarly reinforce our broader conclusion concerning the importance of the shared environment for these outcomes. Finally, the inability of our analytical strategy to capture the main effects of shared environments on GPA and PPVT is a limitation which should be extended in future research.

To conclude, we find that shared and nonshared environments and behaviors influence academic achievement and verbal intelligence, even when all unmeasured shared factors (such as genes) are statistically removed from the estimating equation. As such our results do not support the conclusions of research suggesting that objectively shared environments play a negligible role in academic achievement and verbal intelligence

Appendix

Table A1

MZ Pair Concordance Degree Percentage Distribution, in Standard Deviations

	Within-Pair Differences Percentage, in SDs					Total
	0	0-.5	.5-1	1-2	>2	
Academic Outcomes						
GPA, W1	13	31	35	19	2	100%
PPVT, W1	10	53	23	13	1	100%
Behavioral & Attitudinal						
Substance Use	61	--	12	19	8	100%
Delinquency	51	--	25	19	6	100%
College Aspirations	69	--	18	8	5	100%
College Probability	58	--	28	8	6	100%
No Breakfast	80	--	--	--	20	100%
Violence	40	25	22	8	5	100%
School Effort	68	--	--	--	32	100%
Parental Aspirations	24	23	23	20	11	100%
Extracurricular Participation						
All Clubs	17	28	24	23	8	100%
Academic Clubs	36	13	24	19	8	100%
Network Structure						
Density	3	29	21	25	22	100%
Centrality	6	36	21	25	11	100%
Popularity	9	33	26	22	10	100%
Network Content						
Interactions	6	34	25	27	9	100%
School Effort	5	28	22	27	18	100%
School Difficulty	1	29	23	29	18	100%
Delinquency	1	35	23	23	18	100%
Race Heterogeneity	13	28	22	24	13	100%
Age Heterogeneity	6	29	23	24	20	100%
College Probability	4	33	19	24	21	100%
Middle Class Probability	2	31	23	27	17	100%

NOTE: These calculations used standard deviations from the full Add Health sample, including twins and non-twins. Numbers in cells are percentages.

Table B1

Variable Construction Details

Variable (Original Variables)	Details
Academic Outcomes	
GPA (H1ED11/12/ 13/14)	Average letter grades on 4-point scale.
PVT (AH_PVT)	Add Health Peabody Picture Vocabulary Test
Behavioral & Attitudinal	
Substance Use (H1TO5/10/18/32/ 36/39/42)	Max(H1TO 5,10) + 2* H1TO 32 + 2* H1TO18 + 3*Max(H1TO36,39,42). Indicator variables for tobacco, marijuana, alcohol, and illegal drug use respectively.
Delinquency (H1DS1/2/4/7/ 8/9/10/12/13)	Average of indicator variables for: grafitti, property damage, stealing, running away, driving cars without permission, stealing >$50, breaking and entering, selling drugs, and stealing <$50.
Violence (H1DS5/6/11/14, H1FV5/7/8/9)	Standardized Chronbach's scale. In last 12 months: serious physical fights, hurting someone, use/threaten weapon, group fighting, any fighting, 'pulling' weapons, using weapons, carried weapon to school.
School Effort (S48)	In general, how hard do you try to do your school work well? 1= "I try very hard to do my best," 0=otherwise.
Parental Aspirations (H1WP11, H1WP15)	Sum of 1-5 scaled measures of how disappointed each parent would be if student did not attend college.
College Aspirations (H1EE1)	On a scale of 1 to 5, where 1 is low and 5 is high, how much do you want to go to college?
College Probability (H1EE2)	On a scale of 1 to 5, where 1 is low and 5 is high, how likely is it that you will go to college?
No Breakfast (H1GH23J)	Indicator variable for usually having nothing for breakfast.
Extracurricular Activities	
All Clubs (S44A1-33)	Count of all club/organization/sport participation in in-school questionnaire. Same as Acad. Clubs, plus: cheerleading/dance team, chorus/choir, other club, and all sports.
Academic Clubs (S44A1-13, S44A16, S44A30-33)	Count of organization participation with intellectual components: Languages, Book, Computer, Debate, Drama, FFA, History, Math, Science, Band, Orchestra, Newspaper, Honor Society, Student Council, Yearbook.
Network Structure	
Density (AXSDENS)	$D_i = \Sigma S/s(s-1)$, the proportion of ties between friends which are observed in the ego-network.
Centrality (BCENT10X)	Bonacich centrality score for ego-network.
Popularity (IDGX2)	Number of friendship nominations received.
Network Content	
Interactions (S34[A-E]-S43[A-E])	Sum of friend-specific dichotomous indicators of interactions: went to their house; hung out after school; spent time together last weekend; talked about a problem in last week; talked on telephone in last week.
School Effort (AXSS48)	Average of friends' response to, "In general, how hard do you try to do your school work well?" Scaled 1 to 4.
School Difficulty (AXSS46[A-D])	Average of friends' ratings of different difficulties – with teachers, paying attention, doing homework, getting along with other students.
Delinquency (AXSS59A-G)	Sum of friends' avg. responses (scaled 0 ("never") to 6 ("nearly everyday"): smoking, drinking, getting drunk, racing, dangerous behaviors, lying to parents, skipping school, getting in fights

Variable (Original Variables)	Details
Race Heterogeneity (EHSRC5)	$H_{iA} = 1-[\sum_1^n (R_k/en)^2]$, where R_k is count of nodes with race k in ego-network, en is nodes with valid data for race, and n is the total racial categories in the network.
Age Heterogeneity (EHSAGE)	$H_{iA} = 1-[\sum_1^n (A_k/en)^2]$, where A_k is count of nodes with age k in ego-network, en is nodes with valid data for age, and n is the total age categories in the network.
College Probability (AXSS45E)	Average of nominated friends' self-rated probability of attending college, scaled 0-8.
Middle Class Probability (AXSS45F)	Average of nominated friends' self-rated probability of attaining a middle-class income by age 30, scaled 0-8.
Demographics & Family Structure	
Age (H1GI1M, H1GI1Y, IMONTH, IYEAR)	Difference in Student-reported date of birth and interview date, in years
Full Siblings† (H1HR3A-T)	From household roster.
HH Female† (H1HR2A-T)	From household roster. Count of female siblings.
Birth Order† (H1HR15)	From household roster. =1 if first-born and increases thereafter.
HH Size† (H1HR2)	From household roster. Count of household residents.
Non-White (H1GI4, H1GI6)	=1 if not white or hispanic; =0 otherwise
Parents Married (PA10)	Parental-report; =1 if biological parents married; =0 otherwise.
Sibling Age SD† (H1HR[7/8]A-T)	From household roster. Standard deviation of full sibling ages.
Immigrant (H1GI11)	=1 if born outside U.S.; =0 otherwise
Neighborhood Characteristics	
Bad Neighborhood (PA33-34, H1IR10-11, H1IR14-15)	Weighted sum of following indicators: Litter is a problem (parent-report; weight=1), drug dealers/users are a problem (parent-report; weight=2), family doesn't live in single-unit home (interviewer-report, weight=1), house is not very well kept (interviewer-report, weight=1), buildings on street poorly or very poorly kept (interviewer-report, weight=1), interviewer felt concerned for safety in neighborhood (weight=2).
Home Value Disp. (BST90P28)	Contextual data. Tract-level variation in value of homes.
Social Capital (PA31-32)	Parental-report. Sum of responses (recoded – 1=definitely/probably/might not; 2=probably would, 3=definitely would): how likely tell neighbor if their child getting into trouble, how likely neighbor tell you if child in trouble.
Unemployment (BST90P23)	Contextual data. Tract-level unemployment rate.
Urban (BST90P01)	Contextual data. =1 if tract an urban area; =0 otherwise.
School Characteristics	
Bad School (A7,17,18A-F,19A-F,20A/C,22A/B)	Administrator-reported. Chronbach index of school characteristics: average class size; PTA membership % (reversed); dropout %; held-back %; student [below/above]-grade-level % (above reversed); academic track % (reversed); vocational track %.
Social Cohesion (S62B/E/I)	School-averaged student responses (on Likert scale) to: "I feel close to people at this school," "I feel like I am part of this school," and "I am happy to be at this school."
%College Attendance (A21)	% of seniors attending 2- or 4-year college next year.
Size (ASIZE)	Administrator-reported. =1 if >1000 students; =0 otherwise.
Race Segregation (SEG1RCE5)	School-level difference in expected (under independence) and observed cross-race friendship ties, standardized by expected cross-race ties.
Parental Characteristics	

Variable (Original Variables)	Details
Education (PA12)	Parent-reported highest parental education. Five categories: <HS, HS/Equiv., Sm Coll., BA/Equiv., >College.
Unemployed/Welfare (PA21, PB17, PA16)	Parent-reported. =1 if either parent unemployed and seeking work or on welfare; =0 otherwise.
White Collar† (S14, S20)	=1 if highest-ranked par. occ. white collar (professional, manager, technical worker, office worker, sales worker, or restaurant worker); =0 otherwise.
Health Status (PC49[A-F] _[2/3])	Parent-reported. Sum of indicator variables for both parents: obesity, migraines, allergies, asthma, alcoholism, and diabetes.
Risky Behavior (PA60-63, H1IR23)	Parent- and Interviewer-reported. Sum of indicator variables measuring infrequent seat belt usage, bing drinking frequency, smoking in household, and drinking frequency.
Social Support & Parenting	
Pos. Relationship (PC34A,D)	Parent-reported. Sum of dichotomous parent-child relationship characteristics: each =1 if “you get along well with [him/her],” and “you feel you can really trust [him/her]”; =0 otherwise.
Neg. Relationship (PC34C,E)	Parent-reported. Similar to above, for “you just do not understand [the adolescent]” and “[he/she] interferes with your activities.”
Par. School Involve. (PC25,26,28)	Parent-reported. Sum of indicator variables for talking with child about grades and school work, activities at school, and volunteering at school in the past week.
Parental Control† (H1WP2-7)	Sum of six indicator variables: Do your parents let you make your own decisions about: people you hang around with; what you wear; how much TV you watch; programs you watch; when go to bed; what you eat.
Social Support† (H1PR1-8)	Chronbach index on 1 (“not at all”) to 5 (“very much”) scale. How much: adults, teachers, parents, and friends care; family understand R; want to leave home (reversed); family has fun together, family pays attention.

NOTE: Many variable question wordings are not verbatim, but are abbreviated for space reasons. Full descriptions of all source variables are available in Add Health Wave 1 documentation. Variables are student-reported unless otherwise indicated.

†Averaged from both twin reports to capture shared environmental characteristics.

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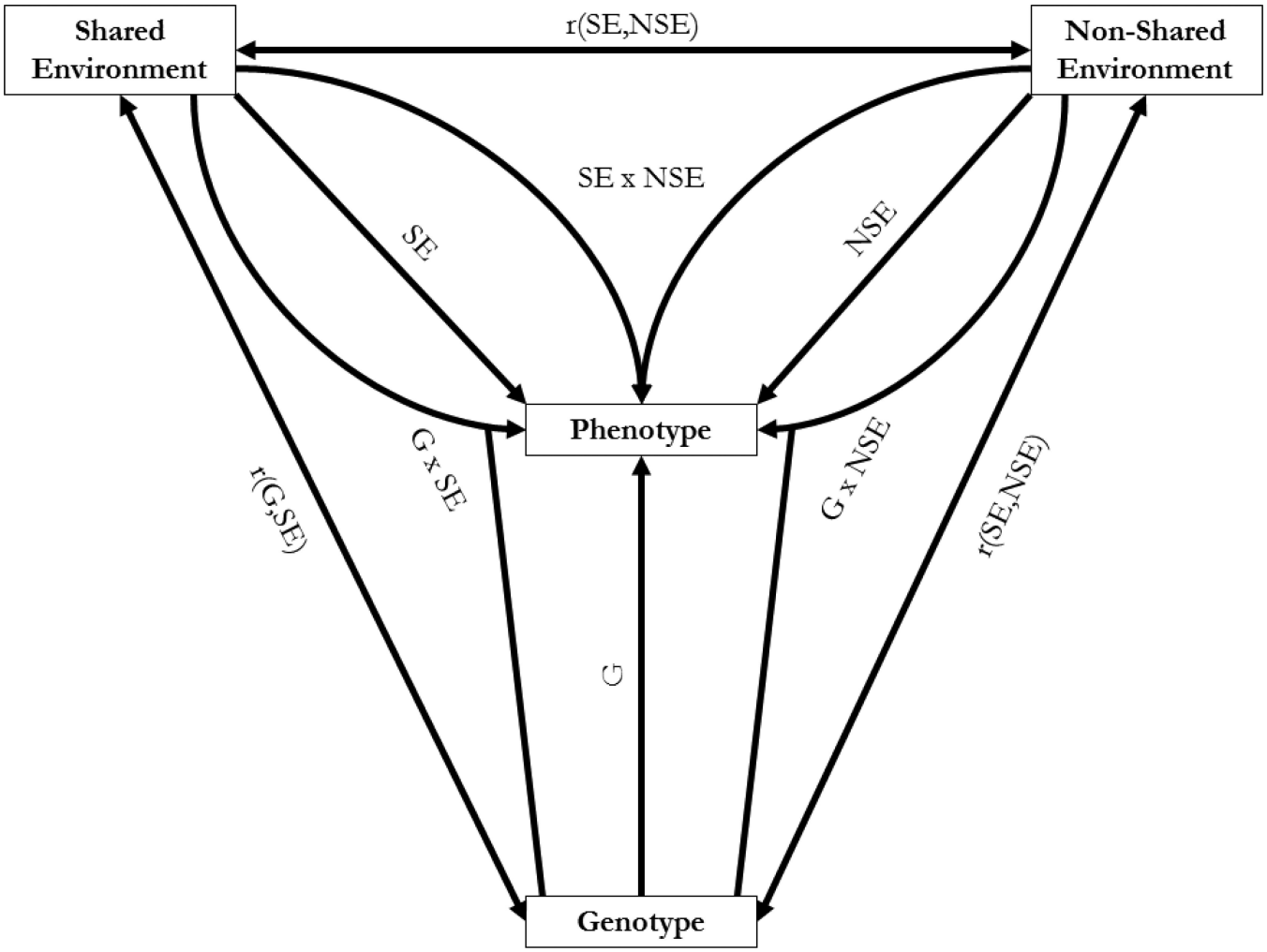

Figure 1.
Complex complex relations between genotype, environments, and phenotypes.

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Table 1

Descriptive Statistics, Non-shared Variables, by MZ Twin Status

	Twins (Imputed, N=578)					Full Sample (Unimputed, N=20,715)				
	Mean	**SD**	**Range**			**Mean**	**SD**	**Range**		
Academic Outcomes										
GPA *	2.84	0.76	1.0	-	4.9	2.75	0.77	1	-	4
PPVT	98.92	14.82	13	-	138	99.67	15.18	13	-	146
Behavioral & Attitudinal										
Substance Use	1.17	1.86	0	-	8	1.29	1.97	0	-	8
Delinquency	0.11	0.18	0	-	1	0.12	0.19	0	-	1
Violence	−0.01	0.72	−1.9	-	5.5	0.00	0.66	−0.5	-	6.3
School Effort *	0.48	0.50	0	-	1	0.38	0.49	0	-	1
Parental Aspirations	7.63	2.96	0	-	20	8.01	2.33	2	-	10
College Aspirations	4.44	1.03	1	-	5	4.42	1.04	1	-	5
College Probability	4.15	1.13	1	-	5	4.13	1.16	1	-	5
No Breakfast	0.19	0.39	0	-	1	0.20	0.40	0	-	1
Extracurricular Participation				-					-	
All Clubs	2.42	2.63	0	-	28	2.32	2.78	0	-	33
Academic Clubs	0.94	1.51	0	-	18	0.86	1.55	0	-	18
Network Structure				-					-	
Density	0.46	0.33	−1.2	-	2.4	0.41	0.20	0.1	-	1
Centrality	0.78	0.69	0.0	-	4.8	0.78	0.65	0.0	-	4.3
Popularity *	4.84	4.05	0	-	23	4.32	3.64	0	-	32
Network Content				-					-	
Interactions	12.46	10.48	−26	-	50	12.65	9.79	0	-	50
School Effort	3.22	0.62	0.6	-	5.5	3.24	0.41	1	-	4
School Difficulty	6.18	4.35	−14	-	27	6.29	2.62	0	-	16
Delinquency	8.06	7.32	−26	-	41	8.22	4.73	0	-	42
Race Heterogeneity	0.23	0.33	−1.5	-	1.6	0.26	0.23	0.0	-	0.8
Age Heterogeneity	0.39	0.32	−1.3	-	1.8	0.44	0.20	0.0	-	0.8
College Probability	6.43	2.44	−6	-	21	6.47	1.46	0	-	8

	Twins (Imputed, N=578)			Full Sample (Unimputed, N=20,715)		
	Mean	SD	Range	Mean	SD	Range
Middle Class Probability	5.12	2.22	−5 - 19	5.14	1.36	0 - 8

NOTE: MZ twin values which are outside the full sample range are the result of imputation procedures.

*MZ-Full Sample t-test $p<.05$

Table 2

Descriptive Statistics, Shared Variables, by MZ Twin Status

	Twins (Imputed, N=578)			Full Sample (Unimputed, N=20,715)		
	Mean	**SD**	**Range**	**Mean**	**SD**	**Range**
Demographics & Family Structure						
Age	16.21	1.55	13 - 20	16.16	1.72	11 - 21
Full Siblings*	3.22	1.13	1 - 9	2.61	1.50	1 - 15
HH Females*	0.84	0.77	0 - 3	0.55	0.78	0 - 8
Birth Order*	1.28	1.04	0 - 7.5	1.07	1.29	0 - 14
HH Size*	4.03	1.46	0 - 12	3.60	1.66	0 - 17
Non-White	0.46	--	0 - 1	0.48	--	0 - 1
Parents Married	0.59	--	0 - 1	0.59	--	0 - 1
Sibling Age SD*	1.81	1.75	0.0 - 9.0	1.55	2.08	0.0 - 50.5
Immigrant*	0.08	--	0 - 1	0.09	--	0 - 1
Neighborhood Characteristics						
Bad Neighborhood	5.69	2.32	3 - 14	5.40	2.25	3 - 14
Home Value Disp.	36024	26710	0 - 159580	38370	27144	4190 - 216343
Social Capital	4.31	1.29	2 - 6	4.40	1.30	2 - 6
Unemployment	0.07	0.06	0.00 - 0.34	0.08	0.06	0.00 - 0.59
Urban*	0.63	--	0 - 1	0.57	--	0 - 1
School Characteristics						
Bad School	0.18	0.26	0.0 - 1.1	−0.02	0.52	−1.8 - 0.9
Social Cohesion	2.31	0.60	0.3 - 4.5	2.46	0.20	1.6 - 3.1
%College Attend.*	61.56	21.74	5 - 130	63.72	22.19	5 - 100
Size*	0.57	0.50	0 - 1	2.35	0.72	1 - 3
Race Segregation	0.28	0.19	0.00 - 1.06	0.27	0.18	−0.00 - 0.75
Parental Characteristics						
Education						

	Twins (Imputed, N=578)			Full Sample (Unimputed, N=20,715)		
	Mean	**SD**	**Range**	**Mean**	**SD**	**Range**
<8th*	0.10	--	0 - 1	0.10	--	0 - 1
HS/Eq.*	0.20	--	0 - 1	0.16	--	0 - 1
Some College	0.26	--	0 - 1	0.28	--	0 - 1
≥Four Year Deg.	0.13	--	0 - 1	0.15	--	0 - 1
Unempd./Welfare	0.29	--	0 - 1	0.27	--	0 - 1
White Collar	0.64	--	0 - 1	0.66	--	0 - 1
Health Status	2.42	1.96	0 - 6	2.35	2.07	0 - 6
Risky Behavior*	1.19	1.09	0 - 4	1.07	0.99	0 - 4
Social Support & Parenting						
Pos. Relationship	1.01	0.75	0 - 2	0.94	0.83	0 - 2
Neg. Relationship	0.81	0.61	0 - 2	0.82	0.68	0 - 2
Par. School Involve.	1.76	1.01	0 - 3	1.56	1.11	0 - 3
Parental Control	2.21	1.13	−1 - 6	2.12	1.33	0 - 6
Social Support*	0.00	0.64	−1.0 - 1.6	0.00	0.60	−1.1 - 1.5

NOTE: MZ twin values which are outside the full sample range are the result of imputation procedures.

*MZ-Full Sample t-test p<.05

Table 3

MZ Twin Fixed Effects, Bivariate and Multivariate, for GPA and PPVT

	GPA		PPVT	
	Bivariate	**Multivariate**	**Bivariate**	**Multivariate**
Behavioral & Attitudinal				
Substance Use	−0.053 *	−0.018 #	−0.453	−0.426 #
Delinquency	−0.539 *	−0.466 #	−2.179	0.669 #
College Aspirations	0.126 **	0.102 #	0.453	0.181 #
College Probability	0.030	−0.012 #	0.184	0.163 #
No Breakfast	−0.133	−0.120 #	−1.174	−1.931 #
Violence	−0.062	0.053 #	−0.967	0.029 #
School Effort	0.092	−0.017	−1.038	−0.057
Parental Aspirations	−0.010	0.047 #	−0.115	−0.502 #
Extracurricular Participation				
All Clubs	0.032	−0.005 #	0.280	0.547 #
Academic Clubs	0.056 *	0.074 #	0.246	−0.108 #
Network Structure				
Density	−0.055	0.011	2.480	−0.146
Centrality	0.038	0.018 #	−0.271	0.910 #
Popularity	−0.011	−0.023 #	0.007	−0.409 #
Network Content				
Interactions	0.001	0.038 #	0.010	−1.737 #
School Effort	−0.014	−0.024 #	0.949	−0.103
School Difficulty	0.014	0.002 #	−0.155	0.031
Delinquency	−0.011	−0.068 #	−0.090	1.479 #
Race Heterogeneity	0.154	−0.010 #	−4.232	1.266 #
Age Heterogeneity	−0.156	−0.129 #	−2.410	−2.228 #
College Probability	0.004	0.178 #	0.019	−3.272 #
Middle Class Probability	0.015	−0.231 #	0.767	−0.717 #
Intercept	(Varies)	3.217	(Varies)	99.522

NOTE:

* p<.05

** p<.01

Statistically equivalent to bivariate estimates

Table 4

MZ Twin Fixed Effects Interactive Models, for GPA and PPVT, Bonferroni-Adjusted for Multiple Testing

	GPA		PPVT	
	|dy/dx|	**P**	**|dy/dx|**	**P**
Demographics & Family Structure				
Age	0.261	0.000 *	0.460	0.002
Full Siblings	0.407	0.000 *	0.059	0.000 *
HH Females	0.022	0.000 *	0.051	0.000 *
Birth Order	0.417	0.000 *	0.409	0.002
HH Size	0.496	0.000 *	0.574	0.000 *
Non-White	0.449	0.000 *	0.023	0.000 *
Parents Married	0.010	0.000 *	0.001	0.000 *
Sibling Age SD	0.727	0.000 *	1.223	0.000 *
Immigrant	1.449	0.000 *	0.074	0.000 *
Neighborhood Characteristics				
Bad Neighborhood	0.333	0.000 *	0.603	0.000 *
Home Value Disp.	--	0.001 *	--	0.000 *
Social Capital	0.707	0.000 *	0.235	0.000 *
Unemployment	--	0.000 *	--	0.000 *
Urban	0.630	0.000 *	0.032	0.000 *
School Characteristics				
Bad School	0.852	0.001 *	0.648	0.000 *
Social Cohesion	0.060	0.000 *	0.087	0.005
%College Attend.	--	0.000 *	--	0.000 *
Size	0.688	0.000 *	0.035	0.000 *
Race Segregation	--	0.000 *	--	0.000 *
Parental Characteristics				
Education				
<8th	0.131	0.000 *	0.007	0.000 *
HS/Eq.	0.049	0.000 *	0.003	0.000 *
Some College	0.069	0.000 *	−0.003	0.000 *
≥Four Year Deg.	0.055	0.000 *	0.003	0.000 *
Unemployed/Welfare	0.360	0.000 *	0.018	0.000 *
White Collar	0.071	0.000 *	0.004	0.000 *
Health Status	0.430	0.000 *	0.338	0.000 *
Risky Behavior	0.163	0.000 *	0.163	0.000 *
Social Support & Parenting				
Positive Relationship	0.760	0.000 *	0.188	0.000 *
Negative Relationship	0.133	0.000 *	0.394	0.000 *
Parental School Involvement	0.604	0.000 *	0.034	0.000 *
Parental Control	0.223	0.000 *	0.170	0.000 *
Social Support	0.362	0.000 *	0.068	0.000 *

NOTE: All likelihood ratio tests compared two models: (a) The reference model included all objectively non-shared variables; and (b) The expanded model added interactions between all non-shared variables and the indicated objectively shared variable. The Bonferroni-adjusted critical p-value for all model comparisons was $\frac{.05}{32} \approx .0015625$. Derivative values are replaced with a '--' mark if they were larger than the range of the dependent variable. Finally, all total effects coefficients are standardized. For dichotomous dependent variables, they are standardized by the dependent variable only.