ACHIEVEMENT AND ASCRIPTION IN EDUCATIONAL ATTAINMENT: GENETIC AND ENVIRONMENTAL INFLUENCES ON ADOLESCENT SCHOOLING

François Nielsen
Department of Sociology
University of North Carolina
Chapel Hill

Revised January 2006

Length: **13,225 words** with all materials

In memory of Bruce K. Eckland 1932 – 1999.

Direct all correspondence to François Nielsen, Department of Sociology, University of North Carolina, Chapel Hill, NC 27599-3210. Email francois_nielsen@unc.edu. Versions of this paper were presented at meetings of the American Sociological Association in San Francisco, CA, August 2004, and Research Committee 28 (Social Stratification) of the International Sociological Association in Neuchâtel, Switzerland, May 2004. The research described in this paper uses data from Add Health, a program project designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris, and funded by a grant P01-HD31921 from the National Institute of Child Health and Human Development, with cooperative funding from 17 other agencies. Special acknowledgment is due Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Persons interested in obtaining data files from Add Health should contact Add Health, Carolina Population Center, 123 W. Franklin Street, Chapel Hill, NC 27516-2524 (www.cpc.unc.edu/addhealth/contract.html). Data analysis was supported by a small grant from University Research Council at UNC-Chapel Hill. For help with the data, encouragement or advice I wish to thank Daniel Adkins, Ken Bollen, Tom DiPrete, Guang Guo, Kathi Harker Tillman, Kathie Harris, Rosemary Hopcroft, Mike Shanahan and Dick Udry. The ideas expressed are the sole responsibility of the author.

ACHIEVEMENT AND ASCRIPTION IN EDUCATIONAL ATTAINMENT:
GENETIC AND ENVIRONMENTAL INFLUENCES ON ADOLESCENT SCHOOLING

Abstract

The classic ("status attainment") model of educational and occupational attainment suffers from three related shortcomings when used as a tool for comparative social mobility research or policy oriented studies of social inequality: (1) model parameters are ambiguous measures of the strength of ascription versus opportunity for achievement; (2) the model is vulnerable to incomplete specification of family background; and (3) associations between background variables and attainment outcomes confound environmental and genetic influences. These issues can be addressed in part by using a model estimated from data on siblings who differ in their degree of biological relatedness that distinguishes between genetic and environmental influences on attainment. This ("behavior genetic") model estimates variance components due to influences of genetic endowment, shared (or common) family environment, and unshared (or specific) environment. One can interpret the relative size of the genetic component (heritability) as measuring opportunity for achievement in a system of stratification, and the relative size of the shared environment component (environmentality) as measuring the extent of social ascription. A multivariate behavior genetic model of adolescent verbal IQ, grade point average, and college plans is estimated using data from the AddHealth study for six types of adolescent sibling pairs living in the same household: MZ twins, DZ twins, full siblings, half siblings, cousins, and non-related siblings. Consistent with a large behavior genetic literature on cognitive and educational measures, results show large genetic components, relatively small shared environmental components, and large unshared environmental component for all three outcomes. The paper concludes that parameters of the behavior genetic model, unlike those of the status achievement model, can be used to compare mobility regimes across social systems, historical periods, and social contexts; the model thus constitutes a potentially important tool for comparative social mobility research.

ACHIEVEMENT AND ASCRIPTION IN EDUCATIONAL ATTAINMENT:
GENETIC AND ENVIRONMENTAL INFLUENCES ON ADOLESCENT SCHOOLING

INTRODUCTION

Recurring questions in the literature on social stratification and mobility involve evaluating the role of ascription versus opportunity in educational and socio-economic attainment: whether some societies are more open than others, whether the importance of ascription is waning in the course of industrialization, or whether a given subgroup of society enjoys fewer opportunities. The status attainment model introduced by Blau and Duncan (1967) is one of the principal tools used to address such questions in a comparative perspective (Breen and Jonsson 2005). Opportunity for achievement is typically associated with the effects on attainment of variables presumed to reflect inherent individual qualities and effort (e.g., cognitive ability, education); ascription or social reproduction is associated with family background characteristics (e.g., parental education, family SES). Such models have thus played a prominent role in public debates concerning the relative roles of achievement and ascription in educational and socio-economic attainment of individuals and groups.

The paper first presents a case that the status attainment model, as it is typically estimated from a sample of unrelated individuals most of whom were raised in their biological families is inadequate as a tool for comparing social mobility regimes due to three related shortcomings: (1) interpretations of model parameters as representing opportunity versus ascription are ambiguous; (2) the model is vulnerable to bias due to incomplete specification of family background; and (3) associations between explanatory variables and attainment outcomes confound environmental and genetic influences.

Second, some of the shortcomings of the attainment model can be alleviated by using an alternative, behavior model of attainment that explicitly distinguishes between genetic and environmental influences on educational or socio-economic outcomes. To illustrate this point I will use data on pairs of siblings who differ in their degree of biological relatedness to estimate a model of schooling involving three variables: verbal IQ, grade point average, and college plans. The model decomposes the variance in verbal IQ and the educational measures into components corresponding to effects of genetic endowment, shared family environment, and unshared (individual-specific) environment. This type of model springs from a research tradition that is well-established in other fields (such as mental health and psychology), but rarely represented in sociology (but see Lichtenstein et al. 1992; Rodgers et al. 2001). The results -- strong effects of genetic factors, relatively small effects of shared environment, strong effects of unshared environment -- may be surprising to sociologists but are quite typical of findings in the behavior genetic literature on cognitive ability and educational achievement, as well as on personality traits and social attitudes (e.g., Pinker 2002; Rowe 1994; Turkheimer 2000).

Finally I propose that behavior genetic models of attainment such as the one developed in this paper are important for comparative social mobility research because they provide consistent measures of the relative importance of opportunity and ascription in a system of stratification: strength of ascription is measured as the proportion of variation in attainment attributable to the shared environment ("environmentality") and opportunity as the proportion of variation attributable to genes ("heritability"). This view is not new; it has been expressed earlier by researchers in several fields, including sociology (e.g., Guo and Stearns 2002; Heath et al. 1985; Rowe 1994; Scarr-Salapatek 1971a, 1971b; Taubman 1995a; see also Table 6), but has not taken root in our field. To illustrate this perspective I will show how earlier research has used systematic differences in estimates of heritability and environmentality to assess differences in opportunity for achievement across groups and historical periods.

Subsequent sections discuss problems with the attainment model and the potential usefulness of a behavior genetic model of attainment. Later sections discuss the data, develop the empirical

model of adolescent schooling, present the results of the analysis, and finally discusses broader implications of the findings for comparative stratification research.

ACHIEVEMENT & ASCRIPTION

Following Blau and Duncan (1967) sociologists have used sets of recursive equations to describe the process of educational and occupational attainment of individuals in a system of stratification. A typical model of *status attainment* (as this type of research came to be known) might consist of three equations

(1) $RsIQ = f_1(FsOcc, FsEd)$
(2) $RsEd = f_2(RsIQ, FsOcc, FsEd)$
(3) $RsOcc = f_3(RsEd, RsIQ, FsOcc, FsEd)$

where IQ denotes a measure of cognitive ability, Ed is educational achievement, and Occ is a measure of occupational prestige; R and F refer to the respondent and the respondent's father, respectively (Duncan, Featherman, and Duncan 1972). The functions f_1, f_2, and f_3 are (typically) specified as linear. The model envisions the career of the individual as a process in which each level of attainment is a function of previous attainment and characteristics of the family of origin.

Blau and Duncan (1967) discovered an empirical pattern that would be often replicated in later research. First, the direct effect of FsOcc on RsOcc (controlling for RsEd) is small, from which they concluded that there is relatively *little social ascription*. Second, the direct effect of RsEd on RsOcc is large; from this they concluded that (1) *education serves to reproduce inequality* (as most of the correlation between FsOcc and RsOcc is indirect, through RsEd), and (2) there is *much opportunity for achievement* (as the major part of the correlation of RsEd with RsOcc is driven by the RsEd residuals, so that achievement appears as largely a function of unmeasured individual resources independent of family background). Overall Blau and Duncan interpret their findings as reflecting a trend of fading importance of ascription in modern industrial society.

The status attainment model was enthusiastically adopted by stratification researchers who saw in it a means of capturing opportunity for achievement (or openness) versus ascription. Opportunity is associated with effects of intermediate achievement variables (such as RsIQ and especially RsEd); ascription with effects of background variables (such as FsOcc or FsEd). With these substantive interpretations of the parameters, the status attainment model can be used as a comparative device to evaluate the relative openness of the stratification system in different societies or historical periods. The attainment model is a principal tool (together with other methodologies) in the collective work on comparative social mobility of Research Committee 28 of the International Sociological Association (e.g., Breen 2004; Breen and Jonsson 2005; De Graaf and Ganzeboom 1993; Ganzeboom, Treiman, and Ultee 1991; Rijken 1999; Shavit and Blossfeld 1993).

Because of the close association of its parameters with normatively loaded concepts of equal opportunity, merit, and social reproduction, the attainment model resonates with powerful moral and political themes of social justice and has become a centerpiece in the policy oriented discourse on social stratification (Olneck 1977: 151). In this normative vein, model parameters can be used to contrast the extent of meritocracy versus the enduring strength of social ascription in contemporary society. These substantive interpretations of the parameters have enjoyed a wide consensus, even among researchers who differ in their philosophical and political preferences.

The classic attainment model is unfortunately inadequate as a tool for comparative stratification research or for use in normative debates on social inequality because of shortcomings related to (1) interpretations of model coefficients in terms of opportunity versus ascription; (2) the possibility of bias in estimation due to incomplete specification of family background; and (3) confounding of environmental and genetic influences. These issues are discussed next.

Interpretation Issues

Interpretations of the effects of background (such as family SES) or intermediate variables (such as IQ or education) on dependent outcomes as measuring the relative roles of ascription (or social inheritance) and opportunity are ambiguous.[1] A given intermediate variable, such as IQ, may be viewed as reflecting in part social inheritance, rather than pure intrinsic talent. The difficulty in linking normative concepts to model parameters is exposed in particularly sharp relief in the debate surrounding Herrnstein and Murray (1994). The authors of *The Bell Curve* note that, controlling for SES of family of origin, cognitive ability (IQ) has a strong effect on a number of educational and socio-economic outcomes. They interpret this pattern (together with other evidence) as symptomatic of increasing opportunity in contemporary U.S. society, a claim that stirred a controversy about both the relative importance of cognitive ability as a factor in attainment and whether effects of this variable measure ascription or opportunity. Critics question the strength of ability effects and/or argue that IQ scores reflect social inheritance rather than native talent, so that IQ effects reflect social reproduction rather than opportunity for achievement (e.g., Fischer et al. 1996). This ambiguity of interpretation compromises the usefulness of the attainment model for comparative research.

Model Specification Issues

The second issue concerns the open-ended specification of family background and is also illustrated by the debate surrounding *The Bell Curve*. Herrnstein and Murray (1994) measure family background with a composite SES index based on parental education and income. Critics point out that the SES composite does not adequately control for all the relevant aspects of family background. Leaving important aspects of the family environment out of the regression model produces specification bias, which artificially inflates the apparent effect of cognitive ability and thus the evidence for opportunity. They contend that if all these factors were properly controlled the effect of ability would be reduced or disappear. Re-estimating some of Herrnstein and Murray's (1994) models with more detailed family background measures does reduce (but typically does not eliminate) the estimated effects of cognitive ability on educational and socio-economic outcomes (Fischer et al. 1996; Korenman and Winship 2000). [2,3]

This exchange illustrates a general problem with the classic attainment model: the task of controlling for family background with measured variables is inherently open-ended. There is no way to guarantee that all relevant aspects of the family environment have been included in the model, and therefore that the strength of ascription mechanisms has not been underestimated and opportunity overestimated. The behavior genetic model described later does provide an estimate of the overall impact of family background variables (measured or unmeasured) on the trait under study and thus contributes to alleviate this problem, although the particular model used in this

---

[1] Blau and Duncan's (1967: 163) identification of ascription with "the circumstances of a person's birth" and achievement with "consequences of [the person's] own actions taken freely – that is, in the absence of any constraints deriving from the circumstances of his birth or rearing" reflects the voluntarism of Linton's (1936) original distinction and strikes us today as involving problematic assumptions about free will (Pinker 's 2002 "ghost in the machine"; see also Eckland 1967: 193-194).

[2] Korenman and Winship (2000) also control for family background using a fixed-effects model based on siblings difference scores, following a tradition of research in sociology using data on siblings (Sewell et al. 2004: 45-57). A general problem with research in that tradition is that difference scores for ordinary (full) siblings include the effect of the fifty percent of genes that the siblings *do not* share, resulting in an inflated estimate of the *environmental* impact of the family. It is instructive in that regard to contrast the sociological literature reviewed by Sewell et al. (2004: 45-57) with the genetically-informed literature on family effects reviewed by Turkheimer and Waldron (2000).

[3] Fischer et al. (1996) also argue that cognitive ability is an *effect*, not a *cause*, of exposure to education exposure and thus do not include cognitive ability as an independent variable in their model of educational attainment (see also Winship and Korenman 1997).

paper is only a partial remedy since the model does not estimate the effects of specific, measured background characteristics.

Confounding of Genetic and Environmental Effects

Perhaps surprisingly, the issue of the role of genes in occupational mobility and the problem of confounding environmental and genetic influences was already addressed in the mainstream sociological literature almost forty years ago. In a remarkable paper Eckland (1967) argued that the social mobility research of his days (then mainly based on mobility tables) was flawed as it assumes, in estimating aggregate mobility, a null model in which sons from any category of origin are equally likely to reach any category of destination.[4] If the abilities to reach certain destinations are in part genetically determined and, as a result, unequally distributed among sons from different origins, it follows that certain categories of sons will be more likely to reach certain destinations; without control for these genetic effects, the resulting asymmetry will be falsely attributed to a lack of perfect mobility. Thus, Eckland claims, the degree of social mobility cannot be properly estimated without controlling for the association between origin and destination due to genetic transmission of abilities. It is worth noting the implication that in measuring social rigidity or ascription, any association between occupational achievements of father and son due to genetic causes should be somehow partialled out from the overall association; ascription is identified with non-genetic (presumably environmental) causes of inter-generational transmission of status.

In another landmark article Scarr and Weinberg (1978) presented results from a study of adopted children showing that the correlations between characteristics of adoptive parents and children's cognitive outcomes are very small, whereas the correlations between these outcomes and cognitive ability of the birth mother are larger, a pattern suggesting that effects of family background variables on achievement of children in biological families are largely due to genetic causes rather than the environmental mechanisms sociologists surmise. Such results imply that the association between father's education and the educational attainment of a biological child may reflect in part the genetic transmission from father to offspring of inherited abilities that enhance educational achievement, rather than purely environmental influences. It follows that the effect of father's education is not a good measure of *social* inheritance, or ascription.

An important implication of Eckland's (1967) and Scarr and Weinberg's (1978) papers is that *biological* inheritance produces associations between background variables (e.g., parental education and occupation) and respondent outcomes (e.g., IQ and education) that are conceptually distinct from, and should not be confounded with, associations due to environmental mechanisms of *social* inheritance (see also Eckland 1979). In the standard status achievement model estimated from respondents raised in their biological families effects of background variables on cognitive, educational and occupational characteristics of respondent are all confounded with genetic influences. By bringing attention to the potential role of genetic factors in the attainment process, these researchers also point to a potential solution to some of the inadequacies of the attainment model, a solution based on explicit recognition and modeling of genetic mechanisms.

THE ROLE OF GENES

Even though their articles were published in a mainstream sociology journal, Eckland's (1967) and Scarr and Weinberg's (1978) critique of the classical attainment model did not have much impact on the field. A small literature investigating the genetic bases of educational and occupational attainment did flourish briefly in the 1970s, involving economists and sociologists

[4] Eckland's (1967) article was an early manifestation of the ongoing revival of Darwinian thinking in the social sciences (Degler 1991: 224), all the more remarkable for appearing in sociology, the social science currently most resistant to biological thinking.

(see contributions in Taubman 1977). The work of Christopher Jencks and colleagues in sociology was a prominent part of that literature (Jencks et al. 1972; Jencks and Brown 1977; Jencks 1980, 1992). This research tradition persevered in economics (Behrman et al. 1980; Behrman, Pollack and Taubman 1995; Björklund, Jäntti and Solon 2005) but faded in sociology.[5] Meanwhile, since the early 1970s, a behavior genetic literature oriented mainly to the substantive fields of mental health, child development, and cognitive and personality psychology has experienced explosive growth (e.g., Plomin et al. 1997). This literature has produced a sophisticated statistical methodology to disentangle genetic and environmental influences on behavior using data on twins, on adopted children, and on other relatives (Neale and Cardon 1992; Neale and Maes, forthcoming; Rowe and Teachman 2001; Shanahan, Hofer, and Shanahan 2003), as well as an accumulation of findings on a substantial role of heredity in cognitive ability, personality, and educational and occupational outcomes (e.g., Baker et al. 1996; Guo and Stearns 2002; Heath et al. 1985; Lichtenstein et al. 1992; Rowe 1994; Rowe, Jacobson, and Van den Oord 1999; Rowe, Vesterdal, and Rodgers 1999; Tambs et al. 1989). These conclusions on the role of genes in behavior are finding their way to a broader public through popular books (Cohen 1999; Harris 1998; Pinker 2002).

Behavior genetic models partition the variance of a measurable trait (called a *phenotype*) into a component due to *genetic inheritance*, a component due to the *shared (*or *common, between-families) environment* of siblings (aspects of the family and the larger rearing environment that tend to make siblings reared together alike), and a component due to the *unshared (*or *specific, unique, within-family) environment* of a sibling (environmental factors that differ among siblings and tend to make them different). This decomposition of the phenotypic variance is the key to resolving the difficulties of the attainment model discussed earlier.[6]

First, in the behavior genetic model a clear distinction can be made between the relative roles of achievement and ascription. The shared environment component reflects the combined impact of such factors as social class, parental network of acquaintances, minority status, availability of reading materials at home, the quality of schools in the community, neighborhood characteristics, and other aspects of the rearing environment that constitute the common experience of siblings in a family and affect their outcomes (such as educational achievement) in a similar way. These are the background characteristics that stratification researchers presumably have in mind when they conceptualize mechanisms of social reproduction and the ascriptive assignment of status. The shared environment component of the total variance in outcome can be interpreted as a measure of ascription (Guo and Stearns 2002; Heath et al. 1985; Scarr-Salapatek 1971a, 1971b). As Rowe (1994: 33) writes: "This ratio [of shared environment to total phenotypic variance] has important policy implications, because it indicates how a phenotype might be changed by altering the rearing conditions of children with poor phenotypes to be like those of children with good ones. The greater the shared rearing estimate, the more change can be expected to follow from changing rearing conditions"; see Jencks (1980: 734) for a similar argument.

The genetic component, as it reflects the extent to which individuals are able to achieve their *genetic potential* for the trait, represents a measure of opportunity for achievement. In this interpretation the phenotype is viewed as resulting from the interaction of genetic endowment

[5] Reasons may include the considerable influence of Jencks's work combined with a widespread perception (perhaps encouraged by the difficulty of the technical parts of his work) that he had effectively ruled out any significant role of genes in the attainment process. In contrast Jencks (1992) sees himself as having been "on the side of genes" in his earlier work. Another factor may have been vigorous criticism by Goldberger (e.g., 1977, 1979) and the dominant anti-genetic *zeitgeist* in which this critique is rooted. Further discussion of this issue is beyond the scope of this paper.

[6] Many behavior genetic models also include a *genetic dominance* component capturing non-linear effects of allele combinations when a trait is affected by genes with dominant versus recessive alleles.

with the social environment and different social environments as more or less restrictive of the full expression of genetic potential. As Guo and Stearns (2002) write with respect to verbal IQ: "the premise of our analysis [is that] social conditions moderate the expression of biological or genetic predispositions. Different social conditions can result in a different level of genetic influences on a particular behavior (p. 885) … We define genetic potential for intellectual development as innate mental ability (p. 883)". Heritability measures realization of genetic potential and therefore opportunity for achievement.

The unshared environment represents a combination of measurement error and idiosyncratic environmental influences that affect the individual values of siblings on a trait in different ways; examples are birth order, a childhood disease that affects one sibling and not another, or association of siblings with different peer groups (Jensen 1997; Turkheimer and Waldron 2000).

Components of the total phenotypic variance can be compared across social settings, groups, or social systems. Such comparisons potentially inform the comparative sociology of social stratification and mobility, permitting statements on the relative degrees of social openness across societies and historical periods, and across groups and social contexts within a given society. The components are also meaningful conversation pieces in normative debates concerning social inequality.[7]

Second, the problem of incomplete specification of family background is rendered moot in the context of the behavior genetic model. The model measures the overall impact of the shared environment in "black box" fashion, without actually measuring, or even identifying, the variables involved. Thus it is no longer possible to spuriously overestimate the role of achievement variable such as IQ or underestimate the role of family background by leaving out of the model some important aspect of the environment. The type of behavior genetic models that I use in this paper, however, does not allow disaggregating the shared environmental components into effects of specific, measured background characteristics (such as SES), although other types of models do (Behrman et al. 1995; Loehlin, Horn, and Willerman 1997; Taubman 1995b; Waldman 1997). In any case the shared environment component represents an upper bound for the effect of *any* specific dimension of the rearing environment on the outcome.

Finally the behavior genetic approach, by distinguishing between variance components due to genes, shared environment, and unshared environment effectively disentangles the confounding of genetic and environmental influences that affects the classic attainment model.

Behavior genetics designs capable of separating genetic and environmental effects are variously based on comparisons of twins (monozygotic or fraternal, raised together or apart), of adopted and biological children, and on other family designs involving individuals who differ in their degree of relatedness and/or exposure to the same family environment.[8] The next two sections describe the data and the behavior genetic model of adolescent schooling.

DATA

Sibling Pairs in the National Longitudinal Study of Adolescent Health (AddHealth)

Data are from the first two waves of AddHealth, a school-based longitudinal study of adolescents in grades 7 to 12. Wave I was conducted in 1994-1995, and Wave II in 1996 (Bearman, Jones, and Udry 1997; Udry 1998). Pairs of siblings living in the same household were identified and if

[7] Comparison of behavior genetic estimates across contexts requires the assumption that the genetic variance for the trait is the same across the social contexts that are compared. Genetic variances might differ because of different degrees of population heterogeneity or histories of assortative mating. The assumption that the genetic variance is non-zero is *not* required: the few human traits with no genetic variance (e.g., the language one speaks and perhaps religious *denomination*) are appropriately characterized as purely ascriptive, in the sense that they are entirely determined by circumstances of birth.

[8] See Rodgers et al. (2001) for an excellent didactic example in sociology, and Rowe and Teachman (2001) for a survey of different behavior genetic designs; other examples in the sociological literature are Scarr and Weinberg (1978); Lichtenstein, Pedersen, and McClearn (1992); and Guo and Stearns (2002).

necessary a sibling was added to the sample to complete the pair. Pairs were classified as MZ (monozygotic twins, N=170), DZ (dizygotic twins, N=290), FS (full siblings, N=702), HS (half siblings, N=242), CO (cousins, N=105), and NR (non related, N=174). (The Ns are the numbers of sibling pairs actually used in the analysis, after the sample restrictions discussed below and cases lost due to missing data.) Most same-sex twins were determined to be monozygotic (MZ) or dizygotic (DZ) on the basis of their self-reported confusability of appearance (i.e., look like two peas in a pod as young children; confused by strangers; by teachers; by family members). Some twin pairs of uncertain zygosity were classified on the basis of molecular genetic markers (see Rowe and Jacobson 1998 for details). All pairs are used, even though data on an individual may be repeated (when the same individual is a member of both a twin pair, say, and of a pair of ordinary siblings), so that some of the pairs are not independent. Studies have shown that estimates from such data are unbiased, although not as precise as they would be under full independence; the result is that tests of goodness of fit are more likely to be rejected than they would be under independence (Eaves et al. 1999: 67).[9]

Measures

Verbal IQ (VIQ) measures verbal cognitive ability; it is the score on a test consisting of even-numbered items (87 items out of 175) in form L of the Peabody Picture Vocabulary Test (PPVT). The variable used in the analysis is AddHealth variable AH_PVT, which is age-standardized from the raw score and expressed on an IQ scale with mean 100 and standard deviation 15 (see also Neiss and Rowe 2000). *Grade Point Average (GPA)* is calculated from questions of the form "At the most recent grading period what was your grade in English or language arts?", "… in mathematics?", "… in history or social studies?", "… in science?". The four questions were asked in both Wave I and Wave II. Original codes were reflected to range from A=5 down to F=1. GPA is calculated as the average of available responses to the eight questions. *College Plans (CPL)* is calculated from the questions "On a scale of 1 to 5, where 1 is low and 5 is high, how much do you want to go to college?", and "…, how likely is it that you will go to college?". The questions were asked in both Wave I and Wave II. (Questions in Wave II added an answer category, coded 6, for respondents who already were in college.) CPL is the average of answers of available responses to the four questions.

Sample Restrictions

As respondents whose native language is not English are known to achieve lower scores on the English version of the PPVT on which VIQ is based, only blacks and non-Hispanic whites were included in the analysis. Preliminary analyses of sibling correlations also revealed a few outlying observations with very low VIQ scores (below 50). Such low scores are likely the result of either profound retardation or a survey artifact and inconsistent with the assumptions of polygenic inheritance and multivariate normality underlying model estimation; these observations were excluded from the data set.

Data Transformations

Average VIQ score is about 12 points less for blacks compared to whites, and about 2 points less for females compared to males. There are also significant race and sex differences in GPA, and a smaller sex (but not race) difference in CPL. Since siblings living in the same family are usually of the same race, mean race differences inflate correlations among all siblings, which tends to inflate estimates of shared environmental effects in behavior genetic models (Rowe, Jacobson, and Van den Oord 1999). Mean sex differences tend to increase variances for opposite-sex siblings relative to same-sex siblings and also distort genetic estimates. To control these

[9] There is much less overlap in the data used in this paper, compared to the multiple-pedigrees design used by Eaves et al. (1999).

differences while keeping the genetic analysis simple I standardized each variable within each of the four race by sex categories (black female, black male, white female, white male). There is substantial age variation among these adolescents in 7 to 12 grades. However, VIQ is already age-standardized, and associations of age with other measures, while statistically significant in this large sample, are substantively minute: the largest effect of age, on CPL, is .06. Thus age is not controlled further.

MODEL AND METHODS

The school attainment model is depicted in Figure 1. It is a structural equations model (SEM) containing both observed variables represented by squares and unmeasured, latent variables represented by circles.[10] The model can be viewed as a behavior genetic extension of a classical educational attainment model describing the recursive interrelationships of verbal IQ (VIQ), grade point average (GPA), and college plans (CPL).[11] Straight arrows between VIQ and GPA and CPL, and between GPA and CPL, represent elements of the classic path model relating the three variables in a recursive fashion. Thus, verbal IQ is assumed to affect both GPA and college plans; GPA also directly affects CPL. I will refer to this subset of effects as the *phenotypic* part of the model.

------ Figure 1 about here ------

The behavior genetic aspects are embodied in the remaining, latent variables. Measured variables for a given sibling are shown as functions of three genetic factors, A_1, A_2, and A_3 that are assumed uncorrelated *within* sibling. The first genetic factor, A_1, affects all three phenotypes VIQ, GPA, and CPL. The second genetic factor, A_2, affects only GPA and CPL, and the third, A_3, affects CPL only. This patterning of the paths is called a Cholesky factorization (Neale and Cardon 1992; Neale and Maes, forthcoming; see empirical examples in Emde and Hewitt 2001). The Cholesky structure is justified on both substantive and methodological grounds. Substantively, each genetic factor is viewed as a distinct (non-overlapping) set of genes. The first set, A_1, consists of all genes that affect all three measures of attainment, possibly with different strengths; the factor A_2 then consists of genes affecting GPA and CPL only; and finally A_3 consists of additional genes that affect CPL exclusively. Methodologically, the Cholesky structure is desirable because it corresponds to the unique (up to multiplications of the columns by –1) lower triangular matrix **X** of path coefficients such that $\mathbf{A}=\mathbf{X}\mathbf{X}'$, where **A** is the positive definite predicted covariance matrix of the genetic factors. Postulating the factor pattern **X** as Cholesky insures at once the uniqueness of **X** and positive definiteness of the predicted covariance matrix **A** (Neale and Maes, forthcoming).

Substantively the matrix $\mathbf{A}=\mathbf{X}\mathbf{X}'$ represents the genetic component of the total predicted covariance matrix of the measured variables, containing the inherited components of the phenotypic variances (on the diagonal) and inherited components of the *covariances* among the variables (off the diagonal). The Cholesky factorization represents a saturated model that can be constrained (by fixing parameters to zero) to yield substantively meaningful nested sub-models. The *common factor* sub-model is one in which the latent source of variation is reduced to a single common factor (e.g., the **X** matrix is reduced to a single column corresponding to A_1). In the *independent factors* sub-model, by contrast, each factor A_1, A_2, and A_3 affects only one of the observed variables, so the **X** matrix is diagonal. These sub-models can be statistically tested.

[10] I use the SEM approach in the multivariate analysis carried out for this paper. There is an alternative regression-based behavior genetic methodology appropriate for univariate analysis called DF analysis (DeFries and Fulker 1985; Kohler and Rodgers 2001).

[11] For simplicity the model is shown with measured dependent variables, represented in squares; an alternative approach would be to distinguish between latent true scores for VIQ, GPA, and CPL (in circles) each linked to its respective indicator (in a square) by a fixed path equal to the square root of the indicator's estimated reliability (see Loehlin 2004).

Each genetic factor for one sibling is correlated with the corresponding factor for the other sibling by a quantity *k* corresponding to the degree of relatedness of siblings, i.e. the proportion of genes that they share by common descent. MZ twins have the same genes, so their genetic factors are identical and *k* is always equal to 1.0. When mating is random (a strong assumption that will be relaxed later) DZ twins and full siblings share half their genes, so $k = .5$. Genetic correlations for the remaining sibling types are then $k = .25$ for half siblings; $k = .125$ for cousins; and $k = 0$ for non related siblings. It is the ability to use genetic theory to specify in advance the association between genotypes of siblings -- an idea going back to Fisher (1918) -- that gives behavior genetic models the leverage to disentangle genetic from environmental effects.

Latent factors C_1, C_2, and C_3 represent the *shared* or *common* environment of siblings, which corresponds to the rearing environment as usually understood by sociologists, consisting of such variables as social class or family SES, quality of schools in the community, ethnic culture, family connections, etc., that affect siblings reared in the same environment in the same ways and thus contribute to make them phenotypically *similar*. The shared environment is decomposed into a Cholesky structure involving three orthogonal factors C_1, C_2, and C_3, in the same way as the genetic factor structure. The *C* factors are assumed uncorrelated *within* sibling; as the shared environment is assumed to affect each sibling in the same way, the correlation of each *C* factor *across* siblings is fixed to 1.

Finally, latent factors E_1, E_2, and E_3 represent the *unshared* or *unique* environment of each sibling, consisting of sibling-specific experiences that contribute to make siblings phenotypically *different* from each other. Such differentiating environmental influences might include parental preference, birth order, influences of different teachers or peers, or a disease affecting one sibling but not the other. In this model the specific environment also includes errors of measurement in the variables. The unshared environment too is modeled as a Cholesky structure composed of three factors uncorrelated within sibling. The sibling-specificity assumption is implemented by fixing to 0 the correlation of each factor across siblings.

The model is estimated by deriving mathematically the expected covariance matrix of the observed variables for each type of sibling pairs as a function of model parameters.[12] Each matrix has dimension 6 by 6, with rows and columns corresponding to the observed phenotypes for each sibling (i.e., VIQ_1, GPA_1, CPL_1, VIQ_2, GPA_2, CPL_2 where subscripts denote siblings in a pair). The parameters of the model are then estimated simultaneously for the 6 types of siblings by minimizing the discrepancies between expected and observed covariance matrices according to the maximum likelihood (ML) criterion (Bollen 1989; Loehlin 2004). The SEM program Mx was used (Neale et al. 2003). Each 6 by 6 covariance matrix provides $(6x7)/2 = 21$ statistics (variances or covariances), so there is a total of 126 statistics over the 6 groups of sibling pairs. The full model contains 21 path coefficients to be estimated (6 for each Cholesky factorization plus 3 for the phenotypic model), so there are 105 df remaining to test the fit of the model.

RESULTS

Following the recommended strategy, model parameters are estimated from covariance matrices rather than from correlations (Loehlin 2004; Neale and Maes, forthcoming). However, it is instructive to look at correlations to get a sense of the information used to estimate the variance components is based. Correlations for the six types of sibling pairs are shown in Table 1. Correlations across siblings for the same variables are shown in bold type. Comparing MZ and DZ twins in the top panel of the table it appears that cross-sibling correlations for MZ twins (below the diagonal) are high, .724, .660 and .663 for VIQ, GPA, and CPL, respectively. The corresponding correlations for DZ twins (above the diagonal) are smaller, typically about half that

[12] The derivation of the model and the Mx script used in the analysis are available from the author at http://www.unc.edu/~nielsen/sf05/.

for MZ twins except for CPL: .356, .332, and .264.[13] A classic estimator of heritability (the proportion of the variance in a trait associated with the genotype) is twice the difference between the correlations of MZ and DZ twins. Thus one can estimate heritability as 2(.724 - .356) = .736 for VIQ, 2(.660 - .332) = .656 for GPA, and 2(.663 - .264) = .798 for CPL. These heritability values are not incompatible with those found in other studies of cognition-related outcomes for adolescents and young adults (e.g., Plomin and Petrill 1997). At the other extreme of relatedness, correlations for non-related siblings living in the same household (third panel of Table 1, above diagonal) are estimates of the pure impact of the shared environment of siblings on the outcomes; their small sizes (.063, .080 and .190) constitute a preliminary hint that the shared environment is not a strong determinant of these variables in this population, except perhaps for CPL.

------ Table 1 about here ------

The full model is denoted BACE, as it specifies direct paths relating observed variables (contained in matrix B), in addition to a full Cholesky structure associated with each latent component A, C, and E. Fit statistics for the BACE model are shown on the first line of Table 2. Unfortunately the B matrix in the BACE model is not identified; the reason is that the ACE part of the model completely accounts for the observed variables and their correlations, so B cannot improve the fit.[14] B can be estimated in a simplified model with no genetic component, a single shared environment factor C_1 affecting the observed variables and three uncorrelated latent variables E_1, E_2, and E_3 representing the unshared environment of VIQ, GPA, and CPL, respectively. (C_1 is equivalent to a pair-specific fixed effect affecting each observed variable.) The model is labeled BC_1E_d to indicate the shape of the matrices involved, with C reduced to a single column and E to a diagonal matrix.

------ Table 2 about here ------

The fit statistics for the BC_1E_d model are shown on line 2 of Table 2. The χ^2 is 555.888 for 117 df ($p<.001$), which is not a satisfactory fit. (With SEMs the goal is to obtain a *non-significant* model.) However with the large number of pairs in the data set (total N=1683) any discrepancy between expected and observed covariances tends to be significant. The RMSEA is a measure of fit that adjusts for degrees of freedom (df) and for the sample size (McDonald 1989; Steiger and Lind 1980). At .112 RMSEA is below the threshold of .10 that represents a good fit. To check the significance of B the model on line 3 of Table 2 drops B from the model. This results in a χ^2 increase of 89.657 for 3 degrees of freedom, a highly significant deterioration in fit. Thus the path coefficients relating the three observed variables of the model are significant in the context of this simplified specification of the latent structure with no genetic influences. One reason for the poor overall fit of Models 2 and 3 is that the absence of genetic component implies identical covariance matrices for all six groups of sibling pairs, a pattern that is clearly inconsistent with the data (Table 1).

χ^2 for the full ACE model is 153.930 with 105 df ($p=.001$), which at first sight is not a satisfactory fit. However RMSEA is .042, below the threshold of .05 corresponding to a very good fit. Fixing to zero the shared environment structure C (model AE) causes a highly significant increase in χ^2 of 25.139 for 6 df ($p<.001$); therefore the shared environment structure cannot be dropped from the model. Fixing the genetic structure A (model CE on line 6) produces a highly significant increases in χ^2 of 142.273 with 6 df ($p<.001$); thus the genetic structure cannot be excluded either.

Models 7 to 11 test alternative specifications of the internal structure of the A, C, and E matrices, specifically whether the lower triangular matrix can be replaced by a simpler model

[13] For CPL the DZ correlation (.264) is less than half the correlation for MZ twins (.663/2=.332). This pattern suggests the presence of a dominance effect. This possibility is addressed later.

[14] BACE produces the same estimates and the same fit as the ACE model (see line 4 of Table 2) for the behavior genetic part, but the estimated coefficients of B shift with changes in the initial values of the coefficients, indicating underidentification (Neale and Maes, forthcoming).

consisting of a diagonal matrix (three uncorrelated factors, each affecting a single outcome) or by a single column (a single factor affecting all three outcomes). Reducing A to a single column (model A_1CE, called the *common factor* model) or to a diagonal matrix (model A_dCE, called the *independent factors* model) leads to unacceptably large χ^2 increases (68.014 and 34.209 with 3 df, respectively). Factors in the A matrix can be thought of as sets of genes. Thus the tests on lines 7 and 8 show that the genetic structure can neither be reduced to a single set of genes affecting all three outcomes (A_1CE), nor to three uncorrelated sets of genes, each affecting a single factor (A_dCE). Likewise reducing the shared environment structure C to a diagonal matrix representing three independent factors (model AC_dE) results in a significant increase in χ^2 (13.507 for 3 df, p=.004). However reducing C to a single column representing a single shared environment factor affecting all three variables (model AC_1E) produces a non-significant increase in χ^2 (4.877 for 3 df, p = .181). Finally, combining a common factor structure for the shared environment with a diagonal structure for the unshared environment (model AC_1E_d) produces a slightly refined model that does not fit significantly worse than ACE (χ^2 increase 11.505 for 6 df, p = .074) or AC_1E (χ^2 increase 6.628 for 3 df, p = .085), and therefore becomes the favored model. AC_1E_d is favored over AC_1E for its simplicity, despite a slightly larger AIC value: AIC is –62.565 for AC_1E_d versus –63.193 for AC_1E.[15] Estimated parameters for the two models hardly differ.

------ Table 3 about here ------

------ Figure 2 about here ------

Table 3 and Figure 2 show standardized path coefficients for the favored AC_1E_d model. The favored model AC_1E_d represents shared environmental influences acting on VIQ, GPA and CPL as a single latent factor affecting all three outcomes, which one might perhaps identify with a "privilege" factor capturing the cognitive and academic advantage shared by siblings due to their rearing environment. The unshared environment, by contrast, is represented as separate factors, each one affecting a single measure. It is the behavior one would expect if the unshared environment consisted largely of measurement error, and unmeasured causes of differences between siblings that behave statistically like measurement errors. This is somewhat surprising theoretically, since one would expect that some unshared influences (e.g., perinatal damage affecting one sibling but not the other) would affect all three variables in similar ways. Effects of the genetic factors tend to be the largest (.202 to .738), and effects of the shared environment the smallest (-.041 to .371), with effects of the unshared environment in between (.572 to .609).

------ Table 4 about here ------

Table 4 shows the proportions of the total expected variances and covariances of the observed variables that are explained by the latent factors. For example, the figures .536, .669, and .600 on the diagonal in the genetic factors panel represent proportions of variance explained by genetic factors, i.e. heritabilities (conventionally denoted h^2). Estimated heritability is high for GPA (.669) and for CPL (.600), and somewhat lower for VIQ (.536). The figures .137, .002, and .030 on the diagonal in the shared environment panel represent proportions of variance explained by shared environmental factors, i.e. environmentalities (denoted c^2). Environmentality is substantial for VIQ (.137) but almost nil for GPA and CPL (.002 and .03, respectively). Finally, the diagonal figures .327, .329, and .370 in the unshared environment panel indicate substantial levels of unshared environmental variance for all three outcomes. Recall that the unshared environment here includes residual variance.

Off-diagonal elements in Table 4 represent the proportion of the *covariance* of two variables explained by a given set of factor. The figures suggest that the associations between VIQ and GPA and between GPA and CPL are entirely due to genetic causes, while the association between VIQ and CPL is about 70% genetic in origin. (Figures greater than 1.0 are an artifact due to the

[15] Akaike's Information Criterion (AIC) is a measure of model fit adjusted for degrees of freedom such that a smaller value indicates a better fit.

slight negative off-diagonal estimates in the shared environment panel.) Thus associations among these cognitive and educational measures seem largely due to genetic factors.

------ Table 5 about here ------

Figures in Table 5 are the squared values of the standardized coefficients of Table 3. As the latent factors are independent within sibling, heritabilities, environmentalities, and specificities (shown in the Total column) are obtained as the sums of the squared paths, according to the rules of path analysis. Table 5 suggests that the high heritability of the measures is due to largely independent genetic factors, i.e. that the genes associated with high VIQ, high GPA, and high CPL constitute substantially non-overlapping sets. This impression is confirmed by computing the estimated correlations among the genetic components of the variances in the three outcome variables (Neale and Maes, forthcoming). The calculations (not shown) yield genetic correlations of .431 for VIQ by GPA, .261 for VIQ by CPL, and .551 for GPA by CPL. This picture of partially independent genetic factors is not consistent with the idea that school success reflects a single "academic ability" factor; rather, it suggests that scores on VIQ, GPA, and CPL might be associated with imperfectly overlapping sets of psychological traits, each with a partially independent genetic etiology. Specifically one might speculate that the qualities responsible for high GPA and high CPL are affected not only by genes enhancing cognitive ability, but also by genes independently affecting non-cognitive traits, such as working habits or conscientiousness, that enhance both school success (as measured by GPA) and educational expectations.

Heritabilities and environmentalities are principal substantive properties of the model. Table 5 shows tests of significance for these parameters using ML-based confidence intervals.[16] All estimates are significant, in the sense that the ML 95% confidence interval does not include zero, except for the environmentality parameter for GPA. Thus the hypothesis cannot be rejected at the .05 level that GPA is entirely explained by a combination of genetic factors and individual-specific, unshared environmental factors of a kind that cause siblings to be *different* from each other. Environmentality for CPL also comes close to non-significance. These results do not suggest strong impacts of shared environmental factors on the schooling process for these adolescents in U.S. schools at the end of the 20th century.

ELABORATIONS

Additional analyses and discussions elaborating on various aspects of the behavior genetic model were not included in this paper to save space. This supplementary material[17] addresses the following issues. (1) Whether the shared environment is more similar for DZ twins compared to full siblings, and for MZ twins compared to DZ twins. (2) Model estimation using twins only (MZ and DZ) rather than the 6 different types of siblings. (3) Effects of allowing assortative mating on parameter estimates. (4) Possibility of a genetic dominance effect in the model for CPL.

DISCUSSION: HERITABILITY, ENVIRONMENTALITY, AND COMPARATIVE STRATIFICATION RESEARCH

In this section I discuss the implications of the finding that variation in cognitive and educational measures is in large part genetic and the idea that heritability (as a measure of opportunity) and environmentality (as a measure of ascription) can be treated as macro-social variables characterizing the mobility process in different groups, social systems or historical periods.

High Heritability / Low Environmentality of School Measures

[16] ML-based confidence intervals are considered superior to those based on estimated standard errors (Neale et al. 2003; Neale and Miller 1997).

[17] Available from the author at http://www.unc.edu/~nielsen/sf05/.

Empirical results shown earlier suggest that the three measures of schooling are highly heritable, relatively unaffected by the shared environment, and substantially affected by unshared environments. Furthermore, the shared environment seems to affect all three measures as a single latent "privilege" factor, whereas genetic influences are better represented as partially independent sets of genes specific to each outcome.

This kind of findings is, perhaps surprisingly, far from isolated. The view that cognitive ability and educational success have a substantial genetic basis and are little affected by the shared environment has become commonplace in mainstream psychology (Brody 1992; Gottfredson [1994] 1997; Neisser et al. 1996; Sternberg and Grigorenko 1997; especially Hunt 1997). As McGue (1997) notes, the scientific opposition to these conclusions now consists of studies using behavior-genetic models tuned to produce lower heritability estimates (e.g., Daniels, Devlin and Roeder 1997; Feldman, Otto and Christiansen 2000). There is also evidence for a role of genes in the determination of earnings (Behrman et al. 1995; Björklund, Jäntti and Solon 2005), personality traits, and some social attitudes (Rowe 1994). These findings have moved Turkheimer (2000) to proclaim three "laws" of behavior genetics, namely that (1) all human behavioral traits are heritable, (2) the effect of being raised in the same family is smaller than the effect of the genes, and (3) a substantial portion of the variation in complex human behavioral traits is not accounted for by the effects of genes or families. Pinker (2002: 372-399) reckons that the three laws may be "the most important discoveries in the history of psychology (p. 372)".

The most controversial implication of findings of a major role of genetic endowment in educational and socio-economic success is a concern that this knowledge could be misinterpreted to make inequality of school outcomes seem "natural, just, and immutable" and to justify opposition to efforts at improving school performance. For this reason Goldberger (1979) suggests abandoning the enterprise of estimating genetic variance components of educational and socio-economic achievement (see also Goldberger 1977; Kamin and Goldberger 2002).[18] Taubman (1995a), declining Goldberger's advice, argues that behavior genetic models are valuable in providing a measure of opportunity for achievement. As Behrman and Taubman (1995) write: "The share of the observed variation in schooling that is attributable to across-family variability in environment [environmentality] provides a measure of inequality of schooling opportunity" (p. 250). An instructive parallel is provided by the field of mental health, where Heston's (1966) initially controversial finding of an important genetic factor in the etiology of schizophrenia, far from promoting the status quo, eventually proved liberating in disposing of the traditional environmental theory attributing the disease to defective mothering and in encouraging the development of ever more effective drug treatments (see also Plomin et al. 1997: 70-71).[19]

The discovery of a substantial genetic component in educational and socio-economic attainment may prove liberating, too. Knowledge that environmental influences (including parenting style) have limited impact on the way children turn out may relieve the anxiety of parents without causing them to begin abusing or neglecting their children (Harris 1998; Pinker 2002; Rowe 1994). On the other hand, heritability and environmentality estimates refer to a specific population, characterized by an existing range of environments; these estimates give no guidance on how difficult it would be to change the trait through environmental manipulation *outside* the existing range (Jencks 1980, 1992: 92-119).[20] Thus high heritability does not imply

[18] See Pinker (2002) on "the fear of inequality" and "the fear of imperfectability" in reaction to findings of a substantial biological basis of human behavior.

[19] Conclusive evidence of a genetic basis for schizophrenia was obtained by behavior genetic methods related to those used in this paper long before a specific gene for schizophrenia was first identified in 2003 (*Science* 2003).

[20] Although it stands to reason that environmental interventions outside the normal environmental range are more likely to be expensive, impractical, or unethical.

that all environmental policies are ineffective (Eckland 1979). Likewise, it is not because socio-economic success has a genetic basis that inequality in socio-economic outcomes is desirable; to conclude otherwise is to commit the naturalistic fallacy (Pinker 2002: 162-163). Time will tell how discoveries about the genetic basis of behavior will be received by sociologists, but it seems unlikely that the accumulated evidence for a major role of genes in socio-economic outcomes will be easily overthrown or ignored.

Behavior Genetic Parameters as Macro-social Variables

A cornerstone principle of behavior genetics is that heritability or environmentality are not fixed properties of a trait but population statistics, and thus inherently dependent on the specific social context. Results presented earlier must be so qualified as pertaining to (non-Hispanic) adolescents in US schools at the end of the 20th Century. For comparative social mobility research the parameters of the behavior-genetic model may be viewed as macro-social variables describing the nature of social mobility in a system of stratification. Heritability h^2 indexes opportunity for achievement (realization of native potential) and, conversely, environmentality c^2 measures inequality of opportunity due to differences in rearing environments (ascription or social reproduction). The analysis reported in this paper describing one society at a particular point in history represents a single "case" from a macro-sociological comparative perspective, or for purposes of policy-oriented evaluations (since such evaluations also imply a comparison between an existing social system and a potential one considered more or less desirable on normative grounds).

The promise of the behavior genetic approach as a tool of comparative social mobility thus depends on securing comparable heritability and environmentality estimates for educational or socio-economic outcomes in different social systems or for subgroups within a social system, in order to score these macro-social units on a scale of ascription versus opportunity. While such a systematic comparative corpus does not exist at present, one can already combine estimates from a number of published studies to construct a "demo" of what genetically informed comparative social mobility research might look like.

------ Table 6 about here ------

Table 6 assembles published estimates of heritability and environmentality for cognitive or socio-economic attainment measures in different social contexts. Comparisons of the behavior genetic parameters can be made either within study (between different groups or social contexts, or different aspects of attainment) or between studies. In both types of comparisons the general hypothesis is that realization of genetic potential and thus heritability will be lower -- and environmentality correspondingly higher -- in social contexts that are either disadvantaged or less advanced on a social-developmental dimension (Guo and Stearns 2002; Heath et al. 1985; Scarr-Salapatek 1971a, 1971b).

Despite being obtained from behavior genetic models that differ in their particulars, estimates in Table 6 exhibit for the most part a systematic pattern consistent with the notion that disadvantaged or less advanced contexts offer fewer achievement opportunities than contexts with opposite characteristics. Heath et al.'s (1985) pioneering study in Norway found that heritability of educational attainment is relatively low (41%) for both males and females born before 1940, who would have been exposed to the more elitist traditional educational system in that country. For two cohorts of males born later heritability rises to 74% and 67%, reflecting greater openness of the system; for females, however, heritability remains lower for these two cohorts (45% and 38%) suggesting that achievement opportunity has increased for males but not for females. A similar scenario emerges from the study of IQ, educational attainment, and occupation in Norway by Tambs et al. (1989). Here heritability is higher for the younger than for the older group, reflecting again less social ascription affecting the younger group. The same inter-generational pattern obtains in the study of educational attainment in Australia by Baker et al. (1996), but the pattern is not as marked in the Norwegian data analyzed by Lichtenstein et al.

(1992). Rowe, Vesterdal, and Rodgers (1999), using US data on young adults, find heritability to be higher for IQ and educational attainment (64% and 68%, respectively) than for hourly wages (42%) (see also Rowe, Jacobson and Van den Oord 1999). Guo and Stearns (2002) show that heritability of verbal IQ for adolescents in AddHealth is lower when a parent is unemployed than when no parent is unemployed (42% versus 54%), and for black as compared to white adolescents (58% versus 72%); lower heritability is again associated with fewer opportunities in disadvantaged social environments. Finally Turkheimer et al. (2003) find higher h^2 and lower c^2 for IQ in high-SES environments, compared to low-SES ones.

However cursory, this partial survey of published estimates of heritability and environmentality for educational and occupational outcomes already suggests some of the contextual variables on which genetically-informed comparative stratification research might focus: historical period and country -- as these factors proxy for the nature of the mobility regime; relative socio-economic advantage, race, ethnicity and sex -- as these characteristics define groups facing more or less opportunity; and age -- both as it defines cohorts subjected to different mobility regimes and as it may otherwise affect parameters of the behavior genetic model.[21]

CONCLUSION

The status attainment model has been used to evaluate the strength of ascription versus equality of opportunity in systems of stratification, but this use of the model is problematic. Estimating ascription from the size of family background effects and opportunity from the size of cognitive ability and education effects is misleading because attribution of the effects of variables to ascription or opportunity is arbitrary, because the model is vulnerable to misspecification of family background, and because effects of background and achievement variables are all confounded with genetic influences. Behavior genetic models estimated from twins and other family data can disentangle ascription from achievement by controlling for genetic influences and by providing an overall ("black box") estimate of shared environmental influences that does not require explicit measurement (or even knowledge) of the variables involved. The impact of the shared environment (environmentality) may be interpreted as a measure of ascription, and heritability as a measure of opportunity for achievement. A multivariate behavior genetic model of verbal IQ, grade point average and college plans estimated from data on adolescent sibling pairs who were in grades 7 to 12 of US schools in 1994-95 shows that variation in all three measures of educational attainment has a large genetic component and a relatively smaller shared environmental component. These findings suggest high levels of educational opportunity for adolescents in US schools at the end of the 20th Century.

A cursory overview of published estimates of the parameters of behavior genetic models of attainment-related variables illustrates the possibilities of a comparative sociology of stratification systems using heritability and environmentality parameters as fundamental measures of ascription and opportunity for achievement in a system of stratification. In this view heritability and environmentality are not fixed properties of a trait; they are properties of the stratification system that are *expected* to vary across societies, historical periods and social contexts. Parameters of the genetic model are estimated, not as permanent properties of a trait such as intelligence, but as a descriptive feature of the social context that may tell us something valuable, say, about the way men and women fare differentially in the educational system in Norway after World War II, or how members of disadvantaged social or racial groups are less able to realize their native potential in the US at the end of the 20th Century.

One last comment about the broader methodological context of the analysis presented in this paper is needed. The models used in the paper are based on the assumption of *polygenic inheritance*, i.e. that the phenotype under study is affected by a number of genes, each of which

[21] A major discovery of behavior genetics in the last two decades is that heritability of cognitive ability increases, and environmentality declines, from childhood to late adulthood (Plomin and Petrill 1997).

has a relatively small effect on the phenotype (Fisher 1918). Another, rapidly developing category of behavior genetic models focuses on individual loci, either to evaluate the contribution of a specific gene (segregation analysis) or to locate on the genome genes with large effects, using genetic markers (linkage analysis). These methods have produced major findings, such as genes associated with schizophrenia or Alzheimer's disease. The success of the gene-finding methods is dependent on the existence of genes with large effects on a trait; only then can the effect of a specific gene be detected against the background of other genetic and environmental influences. The possibility of identifying genes with large effects is methodologically attractive, since part of the genetic contribution is then associated with an explicitly measured variable (i.e., presence or absence of a given allele or marker); effects of other genes are treated as a residual, latent genetic factor. There is a great deal of finality in the identification of a gene with a major effect on a given trait; when the trait in question is a physical or mental disorder, identification of the responsible locus may also facilitate development of a treatment.[22]

Traits of interest in social mobility research, such as cognitive ability, dimensions of personality and measures of socio-economic achievement may, or may not, be affected by genes of major effect that stand out against the polygenic noise of other genes and the additional din of environmental effects. When found, rare genes of large effect may not explain a large proportion of the genetic variance of a trait in the population. Gene-finding methodologies may one day identify genes responsible for educational and socio-economic achievement (thus resolving the latent genetic component into observed DNA measures). However to the extent that genetic influences on these outcomes are truly polygenic, structural equations models based on the assumption of polygenic inheritance like those estimated in this paper may prove useful for a long time to come (Neale and Maes, forthcoming).

[22] Note that the same logic would apply to identification of a specific property of the environment (shared or unshared) with a large effect on the trait.

REFERENCES

Baker, Laura A., Susan A. Treloar, Chandra A. Reynolds, Andrew C. Heath, and Nicholas G. Martin. 1996. "Genetics of Educational Attainment in Australian Twins: Sex Differences and Secular Changes." *Behavior Genetics* 26 (2): 89-102.

Bearman, Peter S., Jo Jones, and J. Richard Udry. 1997. *The National Longitudinal Study of Adolescent Health: Research Design* [WWW document]. URL: http://www.cpc.unc.edu/addhealth

Behrman, Jere R., Zdenek Hrubec, Paul Taubman, and Terence J. Wales. 1980. *Socioeconomic Success: A Study of the Effects of Genetic Endowments, Family Environment, and Schooling*. Amsterdam, Netherlands: North-Holland.

Behrman, Jere R., Robert A. Pollack, and Paul Taubman. 1995. *From Parent to Child: Intrahousehold Allocations and Intergenerational Relations in the United States*. Chicago, IL: University of Chicago Press.

Behrman, Jere R. and Paul Taubman. 1995. "Is Schooling 'Mostly in the Genes'? Nature-Nurture Decomposition using Data on Relatives." Pp. 249-267 in *From Parent to Child: Intrahousehold Allocations and Intergenerational Relations in the United States*, edited by Jere R. Behrman, Robert A. Pollack, and Paul Taubman. Chicago, IL: University of Chicago Press.

Björklund, Anders, Markus Jäntti and Gary Solon. 2005. "Influences of Nature and Nurture on Earnings Variation: Preliminary Results from a Study of Various Sibling Types in Sweden." Pp. 145-164 in *Unequal Chances: Family Background and Economic Success*, edited by Samuel Bowles, Herbert Gintis, and Melissa Osborne Groves. New York: Russell Sage.

Blau, Peter M. and Otis Dudley Duncan. 1967. *The American Occupational Structure*. New York: Wiley.

Breen, Richard, ed. 2004. *Social Mobility in Europe*. Oxford: Oxford University Press.

Breen, Richard and Jan Jonsson. 2005. "Inequality of Opportunity in Comparative Perspective: Recent Research on Educational Attainment and Social Mobility." *Annual Review of Sociology* 31: 223-243.

Bollen, Kenneth A. 1989. *Structural Equations With Latent Variables*. New York: Wiley.

Brody, Nathan. 1992. *Intelligence*. (2d edition.) New York: Academic Press.

Cohen, David B. 1999. *Stranger in the Nest: Do Parents Really Shape Their Child's Personality, Intelligence, or Character?* New York: Wiley.

Daniels, Michael, Bernie Devlin, and Kathryn Roeder. 1997. "Of Genes and IQ." Pp. 45-70 in *Intelligence, Genes, and Success: Scientists Respond to 'The Bell Curve'*, edited by Bernie Devlin, Stephen E. Fienberg, Daniel P. Resnick, and Kathryn Roeder. New York: Springer-Verlag.

DeFries, John C. and D. W. Fulker. 1985. "Multiple Regression Analysis of Twin Data." *Behavior Genetics* 15: 467-473.

Degler, Carl N. 1991. *In Search of Human Nature: The Decline and Revival of Darwinism in American Social Thought*. New York: Oxford University Press.

De Graaf, Paul M. and Harry B. G. Ganzeboom. 1993. "Family Background and Educational Attainment in the Netherlands for the 1891-1960 Birth Cohorts. Pp. 75-99 in *Persistent Inequality: Changing Educational Attainment in Thirteen Countries*, edited by Yossi Shavit and Hans-Peter Blossfeld. Boulder, CO: Westview Press.

Duncan, Otis Dudley, David L. Featherman, and Beverly Duncan. 1972. *Socioeconomic Background and Achievement*. New York: Seminar Press.

Eaves, Lindon, Andrew Heath, Nicholas Martin, Hermine Maes, Michael Neale, Kenneth Kendler, Katherine Kirk and Linda Corey. 1999. "Comparing the Biological and Cultural Inheritance of Personality and Social Attitudes in the Virginia 30,000 Study of Twins and Their Relatives." *Twin Research* 2: 62-80.

Eckland, Bruce K. 1967. "Genetics and Sociology: A Reconsideration." *American Sociological Review* 32: 173-194.
Eckland, Bruce K. 1979. "Genetic Variance in the SES-IQ Correlation." *Sociology of Education* 52: 191-196.
Emde, Robert N. and John K. Hewitt, eds. 2001. *Infancy to Early Childhood: Genetic and Environmental Influences and Developmental Change*. New York: Oxford University Press.
Falconer, D. S. and Trudy F. C. MacKay. 1996. *Introduction to Quantitative Genetics*. Burnt Mill, England: Longman.
Feldman, Marcus W., Sarah P. Otto, and Freddy B. Christiansen. 2000. "Genes, Culture, and Inequality." Pp. 61-85 in *Meritocracy and Economic Inequality*, edited by Kenneth Arrow, Samuel Bowles, and Steven Durlauf. Princeton, NJ: Princeton University Press.
Fischer, Claude S., Michael Hout, Martín Sánchez Jankowski, Samuel R. Lucas, Ann Swidler, and Kim Vos. 1996. *Inequality by Design: Cracking the Bell Curve Myth*. Princeton, NJ: Princeton University Press.
Fisher, Ronald A. 1918. "The Correlation Between Relatives on the Supposition of Mendelian Inheritance." *Transactions of the Royal Society Edimburgh* 52, Part 2: 399-433.
Ganzeboom, Harry, Donald Treiman, and Wout C. Ultee. 1991. "Comparative Intergenerational Stratification Research." *Annual Review of Sociology* 17:277-302.
Goldberger, Arthur S. 1977. "Twin Methods: A Skeptical View." Pp. 299-324 in *Kinometrics: Determinants of Socioeconomic Success Within and Between Families*, edited by Paul Taubman. Amsterdam, Netherlands: North-Holland.
Goldberger, Arthur S. 1979. "Heritability." *Economica* 46: 327-347.
Gottfredson, Linda S. 1997. "Mainstream Science on Intelligence: An Editorial with 52 Signatories, History, and Bibliography." In *Intelligence and Social Policy* [special issue], edited by Linda S. Gottfredson. *Intelligence* 24: 13-23.
Guo, Guang and Elizabeth Stearns. 2002. "The Social Influences on the Realization of Genetic Potential for Intellectual Development." *Social Forces* 80 (3): 881-910.
Harris, Judith Rich. 1998. *The Nurture Assumption: Why Children Turn Out the Way They Do.* New York: Free Press.
Heath, Andrew C., K. Berg, Lindon Eaves, M. H. Solaas, L. A. Corey, J. Sundet, P. Magnus, and W. E. Nance. 1985. *Nature* 314 (25 April): 734-736.
Herrnstein, Richard J. and Charles Murray. 1994. *The Bell Curve: Intelligence and Class Structure in American Life*. New York: Free Press.
Heston, Leonard L. 1966. "Psychiatric Disorders in Foster Home Reared Children of Schizophrenic Mothers." *British Journal of Psychiatry* 112: 819-825.
Hunt, Earl. 1997. "Nature vs. Nurture: The Feeling of *vujà dé*." Pp. 531-551 in *Intelligence, Heredity, and Environment*, edited by Robert J. Sternberg and Elena L. Grigorenko. New York: Cambridge University Press.
Jencks, Christopher. 1980. "Heredity, Environment, and Public Policy Reconsidered." *American Sociological Review* 45 (October): 723-736.
Jencks, Christopher. 1992. *Rethinking Social Policy: Race, Poverty, and the Underclass*. Cambridge, MA: Harvard University Press.
Jencks, Christopher and Marsha Brown. 1977. "Genes and Social Stratification: A Methodological Exploration with Illustrative Data." Pp. 169-233 in *Kinometrics: Determinants of Socioeconomic Success Within and Between Families*, edited by Paul Taubman. Amsterdam, Netherlands: North-Holland.
Jencks, Christopher, Marshall Smith, Henry Acland, Mary Jo Bane, David Cohen, Herbert Gintis, Barbara Heyns, and Stephan Michelson. 1972. *Inequality: A Reassessment of the Effect of Family and Schooling in America*. New York: Basic Books.

Jensen, Arthur R. 1997. "The Puzzle of Nongenetic Variance." Pp. 42-88 in *Intelligence, Heredity, and Environment*, edited by Robert J. Sternberg and Elena L. Grigorenko. New York: Cambridge University Press.

Kamin, Leon J. and Arthur S. Goldberger. 2002. "Twin Studies in Behavioral Research: A Skeptical View." *Theoretical Population Biology* 61:83-95.

Kohler, Hans-Peter and Joseph Lee Rodgers. 2001. "DF-Analyses of Heritability with Double-Entry Twin Data: Asymptotic Standard Errors and Efficient Estimation." *Behavior Genetics* 31 (2): 179-191.

Korenman, Sanders and Christopher Winship. 2000. "A Reanalysis of The Bell Curve: Intelligence, Family Background, and Schooling." Pp. 137-178 in *Meritocracy and Economic Inequality*, edited by Kenneth Arrow, Samuel Bowles, and Steven Durlauf, eds. 2000. Princeton, NJ: Princeton University Press.

Lichtenstein, Paul, Nancy L. Pedersen, and Gerald E. McClearn. 1992. "The Origins of Individual Differences in Occupational Status and Educational Levels: A Study of Twins Reared Apart and Together." *Acta Sociologica* 35:13-31.

Linton, Ralph. 1936. *The Study of Man.* New York: Appleton-Century.

Loehlin, John C. 2004. *Latent Variable Models: An Introduction to Factor, Path, and Structural Equation Analysis*. (4th edition.) Mahwah, NJ: Lawrence Erlbaum Associates.

Loehlin, John C., Joseph M. Horn, and Lee Willerman. 1997. "Heredity, Environment, and IQ in the Texas Adoption Project." Pp. 105-125 in *Intelligence, Heredity, and Environment*, edited by Robert J. Sternberg and Elena L. Grigorenko. New York: Cambridge University Press.

McDonald, R. P. 1989. "An Index of Fit Based on Non-centrality." *Journal of Classification* 6: 97-103.

McGue, Matt. 1997. "The Democracy of the Genes." *Nature* 388: 417-418.

Neale, Michael C., Steven M. Boker, Gary Xie, and Hermine H. Maes. 2003. *Mx: Statistical Modeling*. (6th edition.) VCU Box 900126, Richmond, VA 23298: Department of Psychiatry.

Neale, Michael C. and Lon R. Cardon. 1992. *Methodology for Genetic Studies of Twins and Families*. Dordrechts, Netherlands: Kluwer Academic Publishers.

Neale, Michael C., and Hermine H. Maes. Forthcoming. *Methodology for Genetic Studies of Twins and Families*. (Draft 2nd edition.) Dordrecht, Netherlands: Kluwer Academic Publishers.

Neale, Michael C. and M. B. Miller. 1997. "The Use of Likelihood-based Confidence Intervals in Genetic Models." *Behavior Genetics* 27: 113-120.

Neiss, Michelle, and David C. Rowe. 2000. "Parental Education and Child's Verbal IQ in Adoptive and Biological Families in the National Longitudinal Study of Adolescent Health." *Behavior Genetics* 30 (6): 487-495.

Neisser, Ulric, Gwyneth Boodoo, Thomas J. Bouchard, Jr., A. Wade Boykin, Nathan Brody, Stephen J. Ceci, Diane F. Halpern, John C. Loehlin, Robert Perloff, Robert J. Sternberg, and Susana Urbina. 1996. "Intelligence: Knowns and Unknowns." *American Psychologist* 51 (2): 77-101.

Olneck, Michael R. 1977. "On the Use of Sibling Data to Estimate the Effects of Family Background, Cognitive Skills, and Schooling: Results from the Kalamazoo Brothers Study." Pp. 125-162 in *Kinometrics: Determinants of Socioeconomic Success Within and Between Families*, edited by Paul Taubman. Amsterdam, Netherlands: North-Holland.

Pinker, Steven. 2002. *The Blank Slate: The Modern Denial of Human Nature*. New York: Viking.

Plomin, Robert, John C. DeFries, Gerald E. McClearn, and Michael Rutter. 1997. *Behavioral Genetics*. (3d edition.) New York: Freeman.

Plomin, Robert, and Stephen A. Petrill. 1997. "Genetics and Intelligence: What's New?" Pp. 53-77 in *Intelligence and Social Policy* [Special issue], edited by Linda S. Gottfredson. *Intelligence* 24 (1).

Rijken, Suzanne. 1999. *Educational Expansion and Status Attainment: A Cross-national and Over-time Comparison*. (Ph.D. Dissertation.) Utrecht, Netherlands: ICS/Utrecht Universiteit.

Rodgers, Joseph Lee, Hans-Peter Kohler, Kirsten Ohm Kyvik, and Kaare Christensen. 2001. "Behavior Genetic Modeling of Human Fertility: Findings from a Contemporary Danish Twin Study." *Demography* 38 (1): 29-42.

Rowe, David C. 1994. *The Limits of Family Influence: Genes, Experience, and Behavior*. New York: Guilford.

Rowe, David C. and Kristen C. Jacobson. 1998. *National Longitudinal Study of Adolescent Health: Pairs Code Book* [web document].
URL: http://www.cpc.unc.edu/addhealth/codebk1.html/pairs.zip

Rowe, David C., Kristen C. Jacobson, and Edwin J. C. G. Van den Oord. 1999. "Genetic and Environmental Influences on Vocabulary IQ: Parental Education Level as Moderator." *Child Development* 70: 1151-1162.

Rowe, David C. and Jay Teachman. 2001. "Behavior Genetic Research Designs and Social Policy Studies." Pp. 157-187 in *America's Families and Children: Research Needed in the Coming Millenium*, edited by A. Thornton. Ann Arbor, MI: University of Michigan Press.

Rowe, David C., Wendy J. Vesterdal, and Joseph L. Rodgers. 1999. "Herrnstein's Syllogism: Genetic and Shared Environmental Influences on IQ, Education, and Income." *Intelligence* 26(4): 405-423.

Scarr, Sandra, and Richard A. Weinberg. 1978. "The Influence of 'Family Background' on Intellectual Attainment." *American Sociological Review* 43: 674-692.

Scarr-Salapatek, Sandra. 1971a. "Unknowns in the IQ Equation." *Science* 174: 1223-1228.

Scarr-Salapatek, Sandra. 1971b. "Race, Social Class, and IQ." *Science* 174: 1285-1295.

Science. 2003. "Breakthrough of the Year: The Runners-Up." *Science* 302 (19 Dec): 2039-2045.

Sewell, William H., Robert M. Hauser, Kristen W. Springer and Taissa S. Hauser. 2004. "As We Age: A Review of the Wisconsin Longitudinal Study, 1957-2001." *Research in Social Stratification and Mobility* 20:3-111.

Shanahan, Michael J., Scott M. Hofer, and Lilly Shanahan. 2003. "Biological Models of Behavior and the Life Course." Pp. 597-622 in *Handbook of the Life Course*, edited by J. T. Mortimer and Michael J. Shanahan. New York: Plenum.

Shavit, Yossi and Hans-Peter Blossfeld, eds. 1993. *Persistent Inequality: Changing Educational Attainment in Thirteen Countries*. Boulder, CO: Westview press.

Steiger, J. H. and J. C. Lind. 1980. "Statistically-based Tests for the Number of Common Factors." Presented at the annual Spring Meeting of the Psychometric Society in Iowa City.

Sternberg, Robert J. and Elena L. Grigorenko, eds. 1997. *Intelligence, Heredity, and Environment*. New York: Cambridge University Press.

Tambs, K., J. M. Sundet, P. Magnus, and K. Berg. 1989. "Genetic and Environmental Contributions to the Covariance Between Occupational Status, Educational Attainment, and IQ: A Study of Twins." *Behavior Genetics* 19:209-222.

Taubman, Paul, ed. 1977. *Kinometrics: Determinants of Socioeconomic Success Within and Between Families*. Amsterdam, Netherlands: North-Holland.

Taubman, Paul. 1995a. "On Heritability." Pp. 245-248 in *From Parent to Child: Intrahousehold Allocations and Intergenerational Relations in the United States*, edited by Jere R.

Behrman, Robert A. Pollack, and Paul Taubman. Chicago, IL: University of Chicago Press.

Taubman, Paul. 1995b. "Measuring the Impact of Environmental Policies on the Level and Distribution of Earnings." Pp. 269-285 in *From Parent to Child: Intrahousehold Allocations and Intergenerational Relations in the United States*, edited by Jere R. Behrman, Robert A. Pollack, and Paul Taubman. Chicago, IL: University of Chicago Press.

Turkheimer, Eric. 2000. "Three Laws of Behavior Genetics and What They Mean." *Current Directions in Psychological Science* 5: 160-164.

Turkheimer, Eric, Andreana Haley, Mary Waldron, Brian D'Onofrio and Irving I. Gottesman. 2003. "Socioeconomic Status Modifies Heritability of IQ in Young Children." *Psychological Science* 14: 623-628.

Turkheimer, Eric and Mary Waldron. 2000. "Nonshared Environment: A Theoretical, Methodological, and Quantitative Review." *Psychological Bulletin* 126:78-108.

Udry, J. Richard. 1998. *The National Longitudinal Study of Adolescent Health (Add Health), Waves I & II, 1994-1996* [machine-readable data file and documentation]. Chapel Hill, NC: Carolina Population Center, University of North Carolina at Chapel Hill.

Waldman, Irwin D. 1997. "Unresolved Questions and Future Directions in Behavior-Genetic Studies of Intelligence." Pp. 552-570 in *Intelligence, Heredity, and Environment*, edited by Robert J. Sternberg and Elena L. Grigorenko. New York: Cambridge University Press.

Winship, Christopher, and Sanders D. Korenman. 1997. "Does Staying in School Make you Smarter? The Effect of Education on IQ in *The Bell Curve*." Pp. 215-234 in *Intelligence, Genes and Success: Scientists respond to "The Bell Curve"*, edited by Bernie Devlin, Stephen Fienberg, Daniel Resnick, and Kathryn Roeder. New York: Springer Verlag.

Table 1 – Correlations for six groups of siblings (N = number of sibling pairs)

MZ twins (below diagonal, N=170) -- DZ twins (above diagonal, N=290)						
	VIQ_1	GPA_1	CPL_1	VIQ_2	GPA_2	CPL_2
VIQ_1	--	.239	.172	**.356**	.047	.035
GPA_1	.277	--	.273	.132	**.332**	.062
CPL_1	.290	.378	--	.136	.105	**.264**
VIQ_2	**.724**	.308	.239	--	.245	.136
GPA_2	.182	**.660**	.322	.308	--	.292
CPL_2	.325	.362	**.663**	.374	.393	--
Full siblings (below diagonal, N=702) -- Half siblings (above diagonal, N=242)						
	VIQ_1	GPA_1	CPL_1	VIQ_2	GPA_2	CPL_2
VIQ_1	--	.183	.236	**.310**	-.103	.183
GPA_1	.295	--	.434	.092	**.278**	.081
CPL_1	.212	.380	--	.111	.102	**.204**
VIQ_2	**.411**	.189	.148	--	.127	.203
GPA_2	.163	**.360**	.245	.265	--	.295
CPL_2	.133	.226	**.332**	.252	.406	--
Cousins (below diagonal, N=105) -- Non related siblings (above diagonal, N=174)						
	VIQ_1	GPA_1	CPL_1	VIQ_2	GPA_2	CPL_2
VIQ_1	--	.292	.182	**.063**	-.101	.013
GPA_1	.171	--	.372	-.066	**.080**	-.007
CPL_1	.061	.206	--	.099	.169	**.190**
VIQ_2	**.354**	.127	.007	--	.253	.155
GPA_2	.090	**.104**	-.013	.191	--	.180
CPL_2	.238	.207	**.121**	.271	.224	--

Note: Figures in bold type correspond to correlations for the same variable across siblings. VIQ = verbal IQ; GPA = grade point average; CPL = college plans. Subscripts 1, 2 denote siblings in a pair.

Table 2 – Model comparisons

	Fit statistics					Tests			
Model	χ^2	df	p	AIC	RMSEA	Test	$\Delta\chi^2$	Δ df	p
1. BACE	153.930	105	.001	-56.070	.042				
2. BC_1E_d	555.888	117	.000	321.888	.112				
3. C_1E_d	645.546	120	.000	405.546	.126	3 vs. 2	89.657	3	.000
4. ACE	153.930	108	.002	-62.070	.041				
5. AE	179.069	114	.000	-48.931	.046	5 vs. 4	25.139	6	.000
6. CE	296.203	114	.000	68.203	.079	6 vs. 4	142.273	6	.000
7. A_1CE	221.945	111	.000	-.055	.064	7 vs. 4	68.014	3	.000
8. A_dCE	188.139	111	.000	-33.861	.054	8 vs. 4	34.209	3	.000
9. AC_dE	167.437	111	.000	-54.563	.043	9 vs. 4	13.507	3	.004
10. AC_1E	158.807	111	.002	-63.193	.040	10 vs. 4	4.877	3	.181
11. AC_1E_d[a]	165.435	114	.001	-62.565	.044	11 vs. 4	11.505	6	.074
						11 vs. 10	6.628	3	.085

Note: B = phenotypic paths; A = genetic paths; C = shared environment paths; E = specific environment paths; A_d, C_d, E_d: off diagonal elements of A, C, or E fixed (independent factors model); A_1, C_1: lower triangular matrix A, C reduced to single column vector (common factor model).
[a] favored model

Table 3 – Standardized path coefficients for genetic, shared environmental, and specific environmental factors for favored AC_1E_d model (maximum likelihood estimates)

	Genetic factors			Shared environment			Specific environment		
	A_1	A_2	A_3	C_1	C_2	C_3	E_1	E_2	E_3
VIQ	.732			.371			.572		
GPA	.352	.738		-.041	0		0	.574	
CPL	.202	.376	.646	.172	0	0	0	0	.609

Note: VIQ = verbal IQ; GPA = grade point average; CPL = college plans.
0 denotes a coefficient fixed to zero

Table 4 – Variance and covariance components: Proportions of total predicted variance or covariance associated with genetic, shared environment, and specific environment factors for favored AC_1E_d model

	Genetic factors			Shared environment			Specific environment		
	VIQ	GPA	CPL	VIQ	GPA	CPL	VIQ	GPA	CPL
VIQ	.536			.137			.327		
GPA	1.063	.669		-.063	.002			.329	
CPL	.699	1.021	.600	.301	-.021	.030			.370

Note: VIQ = verbal IQ; GPA = grade point average; CPL = college plans.

Table 5 – Squared standardized path coefficients and 95% maximum-likelihood confidence intervals for total genetic effects (heritabilities $a^2 = h^2$), total shared environmental effects (environmentalities c^2), and total specific environmental effects (specificities e^2) for favored AC_1E_d model

Squared genetic paths				Total (a^2 or h^2)	95% CI	
					Lower	Upper
VIQ	.536			.536	.408	.649
GPA	.124	.545		.669	.585	.725
CPL	.041	.142	.418	.600	.493	.677
Squared shared environmental paths				Total (c^2)	Lower	Upper
VIQ	.137			.137	.060	.217
GPA	.002			.002	.000	.043
CPL	.030			.030	.001	.093
Squared specific environmental paths				Total (e^2)	Lower	Upper
VIQ	.327			.327	.269	.397
GPA		.329		.329	.275	.394
CPL			.370	.370	.310	.441

Note: VIQ = verbal IQ; GPA = grade point average; CPL = college plans. Entries are squared standardized path coefficients for the effects of latent factors on observed variables; the sums of squared paths for a set of latent factors estimate heritabilities (a^2 or h^2), environmentalities (c^2), and specificities (e^2) of the observed variables

Table 6 – Estimated heritability (h^2), environmentality (c^2) and unshared environment variance (e^2) for attainment-related variables in various social groups or contexts

Study	Context and data	Measure of attainment	Comparison group	h^2	c^2	e^2
Heath et al. 1985	Norway; twins, parents	educational attainment	♂ & ♀ b. bef. 1940	41	47	12
			♂ b. 1940-49	74[a]	8	18
			♂ b. 1950-60	67[a]	20	13
			♀ b. 1940-49	45[a]	41	14
			♀ b. 1950-60	38[a]	50	12
Tambs et al. 1989	Norway, twins b. 1944-60	occupation	youngest	43[b]		
		educ. attain.	youngest	51[b]		
		IQ	youngest	66[b]		
		occupation	oldest	16[c]	6	78
		educ. attain.	oldest	10[c]	62	28
		IQ	oldest	37[c]	45	18
Lichtenstein et al. 1992	Norway, twins & adopted	educational attainment	young ♂	35	21	44
			old ♂	30	12	39[d]
			young ♀	20	9	38[d]
			old ♀	12	46	21[d]
Baker et al. 1996	Australia	educational attainment	♂ ♀ b. bef. 1950	57	24	19
			♂ ♀ b. aft. 1950	82	18	- [e]
Rowe, Vesterdal and Rodgers 1999	US (NLSY); full & half sibs.	IQ		64	23	13
		educ. attain.		68	18	14
		hourly wages		42	8	49
Guo and Stearns 2002	US, MZ, DZ, FS, HS, CO, NR adolescent siblings	verbal IQ	unemployed parent	42	39	19
			no unemployed parent	54	22	24
			Black	58	19	23
			white	72	-1	29
Turkheimer et al. 2003	US, 7-year old twins	WISC IQ	low SES	10	58	32
			high SES	72	15	13

Note: [a] figure includes genetic dominance component; [b] average of 3 groups, average for c^2 and e^2 not given; [c] authors comment "this [oldest] sample is small and the estimates are unstable" (p. 209); [d] figures do not add up to 100 because of additional "correlated environmental variance" component; [e] estimate correcting for phenotypic assortative mating for educational level.

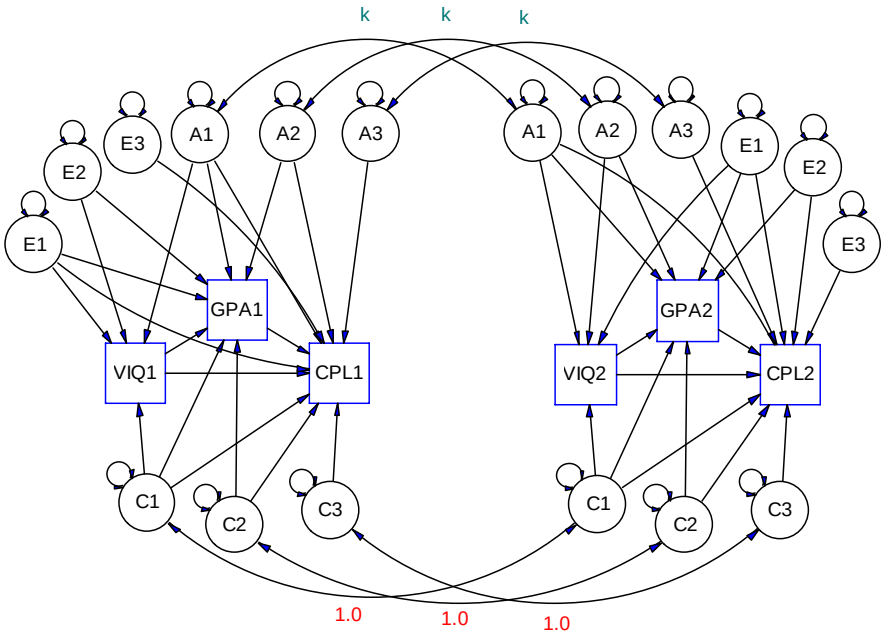

Figure 1 – Full (BACE) model of adolescent schooling.
Note: VIQ = verbal IQ, GPA = grade point average, CPL = college plans. Each side (left or right) of the model corresponds to one of the siblings in a pair. Latent variable sets A_1, A_2, and A_3; C_1, C_2, C_3; and E_1, E_2, E_3 correspond to Cholesky (triangular) factorizations for genetic factors, shared environment factors, and unshared environment factors, respectively (see text for discussion). Each genetic factor A_j is correlated across siblings by a quantity *k* representing the degree of relatedness of siblings (1.0 for MZ; .5 for DZ and FS; .25 for HS; .125 for CO; 0 for NR); each shared environmental factor C_j is assumed perfectly correlated (r=1) across siblings. Variances of all latent variables are set to 1.0.

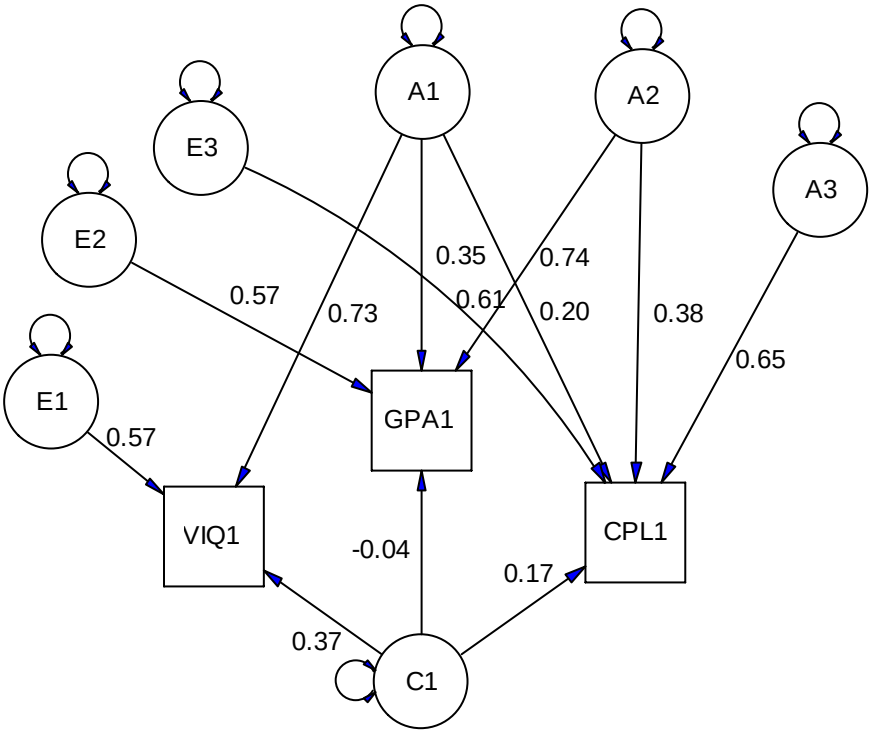

Figure 2 – Standardized path coefficients for favored AC_1E_d model.
Note: VIQ = verbal IQ, GPA = grade point average, CPL = college plans. A_1, A_2, A_3 = genetic factors; C_1 = shared environment factor; E_1, E_2, E_3 = unshared environment factors. Only one sibling is shown.