

**NATURE AND NURTURE EFFECTS ON CHILDREN'S
OUTCOMES: WHAT HAVE WE LEARNED FROM STUDIES OF
TWINS AND ADOPTEES?***

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I. Introduction and Overview

A fundamental question in social science has long been the degree to which children's outcomes are influenced by genes environment, and the interaction of the two. One sensible way to attempt to separate out the effects of genes and environment is to examine data on twins or adoptees since we may be able to make plausible assumptions about the genetic relationships between identical versus fraternal twins or between parents and their adoptive and non-adoptive children.

I begin this chapter by reviewing the methods used by psychologists and behavioral geneticists to identify the effects of nature and nurture and I summarize some of the key results from this large literature. I discuss the assumptions underlying the behavioral genetics model and explain some of the challenges to interpreting the results. I use these issues of interpretation to motivate why economists and sociologists have used a different approach to measuring the impact of environment on children's outcomes. And I discuss the results from the recent literature in economics on environmental versus genetic determinants of children's education, income and health. Finally I try to bring the results from both literatures together to address the issues of what we do know, what we don't know and whether any of this work has implications for social policy or other research on children's outcomes.

Behavioral geneticists have estimated the "heritability" of everything from IQ to "shrewdness" to alcoholism. Their most frequently cited result is that genetic factors explain about 50 to 60 percent of the variation in adult IQ while family environment explains little of the variation in adult IQ¹. This finding is incredibly robust (see Devlin et al [1997]). But researchers' interpretation of the finding is all over the map. Harris [1998] uses the finding of almost no effect from family environment as a key piece of

¹ Studies of young adoptee's IQ find significant effects of family environment, though still only 1/3 as large as the genetic effects. See Cardon and Cherny [1994]. These effects of adoptive family environment appear to be attenuated in adulthood and get even smaller in old age. Plomin et al [2001].

evidence for her thesis that parents do not have a direct effect on their children's outcomes. And both Hernnstein and Murray [1994] and Jensen [1972] interpreted the lack of measured effect from family environment to mean that policies aimed at improving the home and school environment of children are likely to have small impacts on outcomes.

Jencks et al [1972], Jencks [1980] and Goldberger [1977] provide a series of reasons why such strong interpretations may be unwarranted. First of all, understanding the determinants of IQ is different than understanding the determinants of educational attainment, income and health. Second, the assumptions of the behavioral genetics model are likely tilted towards overstating the importance of genes in explaining variation in outcomes. Positive correlation between family environment and genes raises the heritability estimate. Third, family environment is likely endogenous and may depend heavily on genes (Jencks [1980], Scarr and McCartney [1983], Dickens and Flynn [2001]). This endogeneity makes any simple nature nurture breakdown difficult to interpret. Fourth, noting that variation in a given outcome for some population has a large genetic component is very different from saying that the outcome is predetermined or cannot be changed by interventions. Genetic effects can be muted just as environmental effects can be. To take Goldberger's example, a finding that most of the variation in eyesight is due to genes does not imply that we should stop prescribing eyeglasses for people. The use of eyeglasses may add enormous utility for people (and offer an excellent return on investment), regardless of what fraction of eyesight is measured as being environmental.

In other words, knowing what fraction of the existing variance is environmental does not tell us whether a given environmental intervention is doomed to failure or success. Imagine a state with uniformly mediocre schools. Perhaps in that population, school quality doesn't explain any of the variation in student outcomes. But there may be great benefits from introducing a new school with motivated peers, high financial resources and high teacher quality. It is critical to bear in mind that the variance breakdown only deals with variation in the sample. Mechanically, expanding a sample to

encompass a broader range of environment (eg considering children in both Africa and the US as opposed to the US alone) will increase the variation in inputs and outcomes and likely the proportion of the variation in outcomes that is due to environment.

The precision and specificity with which one must properly interpret behavioral geneticists' estimates lead Jencks [1972] to write that "indeed our main conclusion after some years of work on this problem is that mathematical estimates of heritability tell us nothing about anything important." In his analysis Goldberger [1978] summed up by noting that "such conclusions [of high genetic influence on schooling] are unwarranted and indeed the entire effort is misguided." Feldman and Otto [1997] note that obtaining a plausible decomposition has proven notoriously elusive and that we do not know the correct model to use for any given trait.

These warnings from these highly esteemed authors from three separate disciplines (sociology, economics and biology) should be enough to make other social scientists realize that we cannot simply take the estimates from behavioral genetics and plug them into a causal model of children's outcomes or to predict the effect of some policy intervention. In fact, economists are probably right to ignore the importance of genetic factors when studying any particular policy or law change since a policy can have meaningful or tiny effects (or could represent a great or terrible return on investment) regardless of the measured heritability of the outcome.

What then do we learn from behavioral geneticists' estimates of the relative contribution of genes, family environment and non-shared environment? We are getting a breakdown of the variance of the outcome in the current population, assuming a particular structural model. In the case of adoption studies, heritability is a measure of how much more biological siblings resemble each other relative to adoptive siblings. Similarly in the case of twin studies, heritability is a measure of how much more outcomes for identical twins are correlated relative to outcomes for fraternal twins or other siblings. See the next section for the algebra. If heritability estimates were labeled as the additional correlation in outcomes that is associated with being identical rather

than fraternal twins, there might be less misinterpretation and less obsession with these numbers.

Such a variance breakdown may be worth something to social scientists to the extent that they want a best estimate as to whether genetic variation is particularly important in determining an outcome. Even if the functional form of the behavioral genetics model is terribly simplified or wrong, the model might still deliver useful relative rankings of how much variation in genes contributes to variation of different outcomes (e.g. height versus age at first marriage.) The breakdown of outcome variance into variance contributed by genes, family environment and non-shared environment is an ambitious goal, but one that comes with many caveats and questions of interpretation.

Economists and sociologists have suggested several ways to reframe the question so as to use adoption data to estimate some of the causal impacts from family environment without having to know the true model by which outcomes are determined and without having to deliver a complete nature, nurture breakdown. This line of research consists of regressing child outcomes on parental characteristics, i.e. using the more standard approach within economics. For example Plug and Vijverberg [2003] and Sacerdote [2007] regress adoptee's years of schooling on mother's years of schooling, family income and family size. The advantage of using regression is that it tells us which specific parental inputs are most correlated with child outcomes and slope of the relationships.

Certainly one cannot necessarily take these regressions coefficients as causal due to measurement error, endogenous relationships among the right hand variables, and unobservables. But these regressions provide a starting point for understanding which parental inputs matter and how they matter much even in the absence of a genetic connection between parents and children. We can then compare the observed coefficients on parental inputs that we find for adoptees to those that use other sources of variation in family characteristics. For example, Sacerdote [2007] finds little evidence for a direct effect of parental income on adoptees' income and education and this is

generally consistent with the work of Mayer [1997] and Blau [1999].² And one can compare the effects of family size found in adoption studies to those found by Black Devereux and Salvanes [2005b] and Angrist, Lavy and Schlosser [2005] who use the birth of identical twins and sex preferences as an exogenous shock to family size.

One can also generate separate transmission coefficients for adoptees and non-adoptees by regressing the child's outcome on that of the parent. See Björklund Lindahl and Plug [2006] and Björklund Jäntti and Solon [2007] for transmission coefficients of income (education) from parents to adoptees and nonadoptees. This enables one to see how transmission varies when there is and is not a genetic link to the parents. This work also carries the advantage of providing comparability between existing estimates of transmission coefficients from parents to children such as those in Solon[1999], Zimmerman [1992] and Mazumder[2005].

Several bottom lines emerge from my summary of the nature and nurture literature. First, economists who are not already familiar with the literature are generally surprised by how much genes seem to matter, or more precisely stated, how much less adoptees resemble their adoptive parents and siblings than do nonadoptees.³ Second, the estimated effects of family environment on adoptee outcomes are still large in some studies and leave tremendous scope for children's outcomes to be affected by changes in family, neighborhood or school environment. And the importance of family environment can rise significantly when the model is made more flexible.

Third, the precise breakdowns of variance provided by behavioral genetics are subject to a number of important issues of interpretation. Findings of little variance contribution from family environment to variance in children's IQ or educational attainment might be partially generated by the model's assumptions and in any case do not necessarily imply that interventions in environment will not affect child outcomes or that parents actions do not have a direct impact on child outcomes. Finally, the

² Mayer [1997] does find evidence for an effect of parental income on college attendance.

³ In the twins literature, one might say that economists are often surprised by how much more similar are outcomes for identical twins relative to fraternal twins.

importance of adoptive family characteristics for adoptee outcomes varies significantly depending on which outcomes are considered. For example, college selectivity and drinking habits seem to be significantly more influenced by adoptive family characteristics than are test scores.

Ultimately the evidence is quite consistent with the simplistic and widely held view that both nature and nurture matter a great deal in determining children's outcomes. Parental characteristics matter a great deal even in the absence of any genetic connection to their children. A more deeply informed view will also recognize that certain measured parental effects or transmission coefficients from parents to children drop significantly when one considers adoptees rather than children raised by their biological parents. However, that fact does not negate any of the findings of researchers who measure directly the causal effects of changing school, neighborhood and family environment on outcomes.

II. The Behavioral Genetics Model⁴

In the simplest version of the model, child outcomes (Y) are produced by a linear and additive combination of genetic inputs (G), shared (common) family environment (F) and unexplained factors, which the BG literature often calls non-shared or separate environment, (S). This implies that child's educational attainment can be expressed as follows

$$(1) \quad \text{Child's years of education (Y)} = G + F + S$$

The key assumptions here are that nature (G) and shared family environment (F) enter linearly and additively. Separate environment (S) is by definition the residual term

⁴ Large portions of the text here are copied from Sacerdote [2007].

and is uncorrelated with G and F. In the simple version of the model one further assumes that G and F are not correlated for a given child. On the surface, this seems like a strange assumption and one that could perhaps be defended for some subsets of adoptees but not for children being raised by their biological parents. At a deeper level, behavioral geneticists often take F to represent that portion of family environment which is not correlated with genes and they assume that G represents both the effects of gene and gene-environment correlation. The correlation between G and F can be modeled explicitly. If F itself is endogenous, modeling becomes very difficult. With these caveats in mind, one can already see that the BG breakdown into genes versus family environment is not necessarily an easily interpreted decomposition.

With the assumptions of no correlation between G,F, and S, taking the variance of both sides of equation one yields:

$$(2) \quad \sigma^2_Y = \sigma^2_G + \sigma^2_F + \sigma^2_S$$

Dividing both sides by the variance in the outcome (σ^2_y) and defining $h^2 = \sigma^2_g / \sigma^2_y$, $c^2 = \sigma^2_f / \sigma^2_y$, and $e^2 = \sigma^2_s / \sigma^2_y$ yields the standard BG relationship:

$$(3) \quad 1 = h^2 + c^2 + e^2$$

The variance of child outcomes is the sum of the variance from the genetic inputs (h^2 or heritability), the variance from family environment (c^2) and the variance from non-

shared environment (e^2), i.e. the residual. From this starting point, a variety of variances and covariances of outcomes can be expressed as functions of h , c , and e . The sample moments can then be used to identify these underlying parameters. Consider first the relationship for two adoptees. If one standardizes Y, F, G, S to be mean zero variance one, the correlation in outcomes between two adoptive siblings equals

$$(4) \quad \text{Corr}(Y_1, Y_2) = \text{Cov}(Y_1, Y_2) = \text{Cov}(F_1, F_1) = \text{Var}(F_1) = c^2.$$

The correlation in outcomes between two nonadoptive siblings equals

$$(5) \quad \text{Corr}(Y_1, Y_2) = \text{Cov}(G_1 + F_1 + S_1, G_2 + F_2 + S_2) = \text{Cov}(G_1 + F_1, 1/2G_1 + F_1) = 1/2 h^2 + c^2.$$

This assumes that non-adoptive siblings share half of the same genetic endowment and the same common environment (See Plomin et. al [2001] for a discussion). Thus one can recover the full variance breakdown (h^2, c^2, e^2) from just the correlation among adoptive and biological siblings. By comparing (4) and (5) we see that $h^2 =$ twice the difference in correlations in the outcome between the adoptive and biological siblings. This is the "double the difference" methodology frequently referred to in text books or discussions of the BG model (See Duncan et al [2001]).

Now consider the correlation in outcomes between two identical twins versus the correlation for two fraternal twins. Identical twins are assumed to share all of the same genes and the same family environment and hence their correlation in outcomes is

$$(6) \quad \text{Corr}(Y_1, Y_2) = \text{Cov}(G_1 + F_1 + S_1, G_2 + F_2 + S_2) = \text{Cov}(G_1 + F_1, G_1 + F_1) = h^2 + c^2.$$

The algebra for the fraternal twins is the same as the algebra for any two biological (nonadoptive) siblings and hence the same $1/2 h^2 + c^2$ we had in the preceding paragraph. By subtracting (5) from (6) and multiplying by 2, we see that h^2 is twice the difference in

correlations between identical and fraternal twins. Thus the twins literature has its own "double the difference" methodology.

Of course one need not stop at finding the analytical solutions for the correlations for just twins, adoptive siblings and full siblings. One can also write down the equations for correlations in outcomes between children and parents, grandchildren and grandparents, between first cousins etc. This general model is known as Fisher's Polygenetic Model. Behrman and Taubman [1989] provides a great illustration in that the authors present formulae for the phenotypic (outcome) correlations among 16 different possible pairings of relatives.

Note that as we add more pairings of different relatives, we can incorporate additional parameters and potentially make the model more realistic. (We can add flexibility and identify additional structural parameters.) Goldberger [1977] provides examples of models which allow for gene-environment correlation and use correlations among twins reared together, twins reared apart, adoptive siblings, and the parent-child correlations for twins and adoptees. Frequently the structural models employed are overidentified (because there are more pairings of relatives than parameters) in which case the estimation chooses parameters which minimize the sum of squared errors between the sample moments and the fitted values of the sample moments.

In the case of Behrman and Taubman's [1989] model, they allow both for the possibilities of assortative mating and for the effects of dominance versus additive genes. Assortative mating is the notion that couples may positively select on phenotypes (outcomes), ie. mating is non-random which means that siblings may have more than 50% of the same genes. Dominant gene effects are identified separately from additive effects by comparing correlations in outcomes across types of relatives that would have the same genetic connection under an additive system but need not if dominant effects are present. For example, suppose we had data for full siblings, half siblings and identical twins and we assume that all sibling pairs receive the same common environment. Think of the difference in correlation between full siblings and half siblings as identifying the

effects of genetic connection. Under an additive system, genetic effects will cause exactly twice as much resemblance in identical twins as is caused among full siblings. If identical twins resemble each other MORE than twice as much as implied by the other sibling types, one could attribute this "additional" component to the dominance effects of genes.⁵ Behavioral geneticists may also want to allow for the interactive effects of different genes which is known as *epistasis*. By modeling the correlation among even more pairings of relatives (beyond the three types of sibling pairs mentioned above), one can have additive genetics effects, dominance effects and epistasis in the same model.

Note that there are at least three other ways in which the structural modeling can be further extended. First one might assume that the same underlying process gives rise to several different outcomes. For example, one might have two verbal test scores on the same set of individuals. One could treat these as yielding two sets of sample moments which can be used to identify the same underlying parameters. Alternatively, one might have a panel of test score outcomes for the same group of relatives over time and one could posit that the relationship between siblings' outcomes is changing over time in a specific way. See Cardon and Cheny [1994]. Finally one can also allow for different levels of gene-environment correlation among different relatives (e.g. fraternal and identical twin pairs need not be modeled as having correlation of 1.0 in their family environments.)

An important assumption in this modeling is that the various samples used to estimate the relevant covariances have the same underlying variance in genes and family environment. If for example, adoptive families have a restricted range of family environments (Stoolmiller [1999]), then it may not make sense to combine covariances from adoptive siblings, non-adoptive siblings and twins all in the same estimation. Furthermore, the variance breakdown obtained using adoptees may not apply to the general population.

⁵ Goldberger [1977] page 331 offers the following intuition: "If the individual genes have non-additive effects, then it is only the additive part of the effect that makes for parent-child resemblance. The non-additive part of the effect does contribute to the resemblance of siblings, who may happen to receive the same gene combination."

In practice, the results are sensitive to which relative pairs are modeled in the analysis. As noted above, a great deal of weight is often placed on the difference in correlations of outcomes for adoptive siblings versus full siblings or for fraternal versus identical twins. However, if we instead compare parent-child (i.e. intergenerational) correlations to sibling correlations, we get a different answer for h^2 , c^2 , e^2 than if we compare across sibling types. Solon [1999] notes that the sibling correlation equals the sum of the intergenerational correlation squared plus other shared factors, all of which are nurture based. Björklund and Jäntti [2008] points out that sibling correlations in many outcomes are typically much higher than intergenerational correlations. And they show that this fact combined with the BG model implies a large nurture based component to outcomes.

III. Canonical Results from the Behavioral Genetics Literature

As noted earlier, the most voluminous and heavily cited part of the BG literature measures the contributions of genes and family environment to IQ. There are numerous summaries of this IQ literature including Goldberger [1977], Bouchard and McGue [1981], Devlin [1997], Jencks et al [1972], and Taylor [1980]. Table I shows the mean of the estimated correlations in each of these meta-studies along with the number of individual studies incorporated.

Devlin, Daniels and Roeder reviewed 212 different studies of the IQ of twins. The mean correlation in IQ for studies of pairs of identical was .85. The correlations for fraternal twins were similar to correlations for other siblings and averaged .44. One can see immediately that in a simple model this will generate a high estimated heritability; if one assumes that identical twins are twice as genetically related as other full siblings and have twice the correlation in outcomes, equations (5) and (6) would lead one to conclude that all the explained variance is genetic. Goldberger (1977) and Jencks et al [1972] each reviewed a number of twins studies of IQ. The studies they review yielded similar

results. In the case of Jencks [1972], the correlation in IQ for identical twins is .86 versus .54 for other siblings.

Bouchard and McGue [1981] examined a large number of twin studies and adoption studies. The adoption studies find significant correlation in IQ between adoptive siblings. The median correlation in IQ for adoptive siblings is .29 while the correlation for biological siblings raised together is .45. However, many of these studies are for adoptees less than age 18. Studies of older adoptive and biological siblings have found that the correlation in IQ among adoptees tends to fall significantly in adulthood while the correlation for biological siblings grows. Plomin et al [1997, 2001].

Table II translates these sibling correlations into the behavioral genetics decomposition of variance in IQ into portions attributable to variance in genes, family (common) environment and separate environment. The twins designs find that a high proportion of explained variance in IQ is due to genes and very little is due to family environment. Averaging over more than 200 studies, Devlin et al show the average finding is that 49 percent of the variance is genetic and 5 percent is attributable to family (common) environment. The Bouchard and McGue summary of correlations for twins finds similar results, namely that 54 percent of variation is genetic and 4 percent is due to family environment. Non-shared environment (what economists would call the residual or unexplained variance) accounts for a substantial 40-50% of the variation in IQ.

The adoption studies find a larger proportion of variance in IQ attributable to family environment. Cardon and Cherny's [1994] examination of nine year olds in the Colorado Adoption Project found that 16 percent of the variation in IQ is attributable to family environment and 60 percent is due to genes. The Bouchard and McGue summary of IQ correlations for adoptees implies that 29 percent of the variation is due family environment and 32 percent is due to genes. Averaging over the studies in Goldberger's [1977] literature summary which includes both twin and adoption correlations, I find that 22 percent of the variation in IQ is due to family environment and 58 percent is due to genetic effects.

There is a disconnect between the twin and adoption literatures with regard to the importance of family environment. One way to partially resolve this contradiction is to appeal to the findings that family environment effects on adoptees are greatly attenuated in adulthood and that heritability rises with age (Pedersen et al [1992] and McClearn et al [1997]). However, another reasonable explanation is that applying the simple version of the behavioral genetics model to pairs of identical and fraternal twins will overstate heritability if identical twins face environments more similar than that faced for other siblings (Feldman and Otto [1997]).⁶ Or identical twins might affect each other's environment more than do fraternal twins. Recall from section II that *any* factors which make outcomes for identical twins more similar than outcomes for fraternal twins are assigned to genetic effects. The assumption of the structural model is that sibling pairs raised in the same household have the same correlation in family or common environment. One could imagine that parents and teachers would be even more likely to expect or demand similar performance from siblings who are identical twins. Parents may be more likely to provide similar environmental experiences for identical twins. In decomposing sources of earnings variation, Björkland Yäntii and Solon [2005] find that allowing different types of sibling pairs to have different amounts of correlation in family environment greatly lowers the estimated heritability and raises the estimated impacts from family environment.

In Table III, I summarize the existing behavioral genetics studies of variance in years of education. There are far fewer BG studies of education and earnings than of IQ, and the most widely known studies are those done by economists and sociologists. Behrman and Taubman [1989] uses data on twins and their relatives from the National Academy of Science / National Research Council sample. They compute years of schooling correlations for 16 different pairs of relatives and fit the parameters of their model to match the predicted correlations with the sample correlations. Consistent with twins studies of IQ that find high heritability, Behrman and Taubman find that genetic

⁶ Scarr and Carter-Saltzman [1979] provide some evidence that identical and fraternal twins do have similar correlations in family environment.

effects explain 88 percent of the variation in schooling.⁷ Family environment explains little or none of the variance in schooling. Scarr and Weinberg [1994] examine adoptees and find that family environment explains 13 percent of the variation. However, this study is based on only 59 adoptive sibling pairs. Teasdale and Owen [1984] have 163 pairs of adoptees and find that variance family environment explains 5 percent of the variation in schooling.

Overall, to the extent that behavioral geneticists have performed nature-nurture decompositions using years of schooling as the outcome, the findings have mirrored the findings of the much larger IQ literature. Genetic effects play a large role while there is only a small role for family environment. That statement is tempered a bit by the Behrman, Taubman and Wales study and Scarr and Weinberg study, though that study had only 59 pairs of adoptive siblings. A different but equally valid interpretation of the results in Table III would be to say genetic effects clearly matter a great deal in determining schooling, but that the portion attributable to family environment changes significantly depending on how one specifies the structural model.

In Table IV I switch the outcome of interest to earnings and I report results from two different studies. Björkland, Jäntti and Solon [2005] used a large sample of siblings, twins and adoptees from the Statistics Sweden and Swedish Twin Registry. They derive formulae for the predicted correlations among nine different sibling types. They use weighted least squares to choose parameters to best fit the sample correlations to the predicted correlations from the models. One of the key results from this study is that it matters a great deal whether or not one constrains all sibling types reared together to have the same degree of correlation in family (common) environment. With such a constraint (Model 1), genes explain 28 percent of the variance in earnings and family environment explains 4 percent.⁸ By adding three additional parameters to allow for differing correlations in family environment among sibling pairs (Model 4), the importance of

⁷ The earlier Behrman Taubman and Wales [1975] study used the same data set of twins, but found lower heritability of schooling. This may be precisely because of the different way the two studies modeled correlation in family environment.

⁸ These are the numbers for brothers. For sisters, the comparable numbers are 24.5 percent genetic and 1 percent common environment.

family (common) environment rises to 16.4 percent and the genetic effects fall to 19.9 percent.

Table V shows the results from Loehlin's [2005] summary of the behavioral genetics literature on the determinants of personality traits. Like the IQ research, this is a rich literature and Loehlin considers hundreds of studies. He reports average correlations between parents and children for the most commonly measured aspects of personality, namely extraversion, agreeableness, conscientiousness, neuroticism, and openness. With regard to the determinants of personality traits, the literature has reached even more of a consensus than with regard to IQ. The first column is for the correlations between parents and children when children are raised by their biological parents. Correlations range from .11 to .17. When we consider adoptees and adoptive parents in column 2, the correlations almost disappear, falling to an average of .036. Column 3 reports correlations in traits for adoptees and their biological parents. Here the correlations rise almost to the levels seen in column (1), ie for the children raised by their biological parents. This evidence (which again is a summary of hundreds of studies) is striking and certainly points strongly in the direction of genes being an important determinant of personality traits.

As a final outcome of interest, I graph in Figure I some of the data from the Grilo and Pogue-Geile [1981] meta study of correlations in weight, height and body mass index among full siblings raised together, adoptive siblings, and twins. Adoptive siblings have almost no correlation in body mass index. Full siblings raised together have a correlation of about .32. Interestingly fraternal twins show similar levels of correlation to other sibling pairs. The correlation in BMI jumps to .72 for identical twins.⁹

IV. Critiques and Challenges to Interpretation of the Behavioral Genetics Results on IQ and Schooling

⁹ I report the body mass index correlations which combine data for both same and mixed gender pairs of siblings. It would look only moderately different if I controlled for gender.

BG results with respect to IQ appear to be quite robust in finding that the genetic effects account for 50 to 60 percent of the variance in adult IQ. In the twins studies and the studies of adult adoptees, family environment accounts for almost none of the variance.¹⁰ Behrman and Taubman [1989] and Teasdale and Owen [1989] find no role for family environment in explaining years of schooling. What is one to make of these findings? One approach is to accept this finding as not only an accurate estimation of the BG model, but also as having important causal meaning and predictive power for interventions which might affect child test scores, educational attainment or income. This is the approach of Jensen [1973] and Murray [2004] who are pessimistic about the ability of social policy to affect inequality of income and schooling.

This view is unsatisfying not only because it makes one unpopular at dinner parties, but more importantly because such conclusions about the real weakness of family influences and other forms of environment like school quality seem to contradict everyday experience. And it is hard to reconcile a view of minimal effects of shared environment with the extensive investments that many parents and school systems make in their children.¹¹ For example, there is a widespread belief that certain charter schools and certain Catholic schools have large treatment effects on test scores and high school graduation rates. These beliefs have been subsequently confirmed by very careful empirical work on the treatment effects from these schools. See Hoxby and Murarka[2007], Evans and Schwab [1995], and Neal[1997].

One way to handle the apparent contradiction is to note that some BG estimations (particularly those using adoption data for younger adoptees) find a significant role for shared environment in determining income, IQ and education. Perhaps many of the well measured treatment effects of interventions are working through the 15-20% role

¹⁰ Some of the studies of younger adoptees find that up to 16% of the variation in child IQ is attributed to family environment (Cardon and Cherny [1994]). This clearly leaves the question of family environment effects on test scores open to interpretation. Nonetheless both Harris [1998] and Plomin et al [2001] p. 176 sum up the literature by stating that effects of family environment on IQ are modest and get smaller or disappear with age.

¹¹ I am assuming here that a large part of school quality is shared between siblings, which strikes me as a reasonable assumption. Parents may of course invest in children for reasons besides producing higher income and levels of education.

assigned to shared environment. In the large Devlin et al meta study for twins data, the consensus number for the variance explained by family environment is 5%. But the older literature summary by Goldberger [1977] implies an average percent explained by family environment of 22%.

A different approach to reconcile the observed environmental effects on test scores and schooling with the BG decomposition is to note that behavioral geneticists may be working only within a restricted range of environments that are actually observed in the United States or in some other society. Stoolmiller [1999] emphasizes this point and presents corrections for this "restriction of range" problem.

This is certainly a correct and incredibly important point. A variance decomposition only deals with the variance observed in the data. In Sacerdote [2007], I study Korean American adoptees who were placed as infants with US families. Clearly if I extended the sample to included adoptees who remained in Korea, then the variance of educational attainment would rise substantially as would (in all likelihood) the portion attributable to environmental factors such as home country. A less extreme example might be to say that while Catholic schools may represent shared environment and may have large effects, Catholic schools are not an important source of variation in the BG studies being considered.

A third and popular reaction to the key BG findings is that one needs to somehow fix the BG structural model so that it not only delivers more plausible estimates of the effects of shared environment, but can also explain other facts such as the Flynn effect. Flynn [1999] notes that IQ scores tend to rise over time. Dickens and Flynn [2001] present an elegant model in which environment responds endogenously to genetic endowments. This can explain a number of facts including the Flynn effect and possibly why the effect of adoptive parents on adoptee's IQ diminishes in adulthood. The Björklund, Jäntti Solon [2005] decomposition for earnings finds that heritability falls significantly once they allow for different shared environment correlations among identical twins relative to fraternal twins. Following this logic, one could either conclude

A) that the whole enterprise is not robust to modest changes in how we write down the structural model or B) once we make the model a bit more flexible we arrive at a fairly accurate and useful structural model.¹²

More generally there is a sizable literature that points out that gene environment interactions or the endogeneity of environment will cause the BG model to understate the importance of shared environment and overstate the importance of genetic factors. See Ridley [2003] or Turkheimer [1997]. Turkheimer et al. [2003] makes the point that nonlinearities in the relationship between genetic factors and outcomes can cause the BG model to overstate heritability. In particular they find that measured heritability is lower for children in less advantaged families. Lizzeri and Siniscalchi [2007] point out that if parents are behaving optimally, the learning process for adoptees and nonadoptees will likely differ and that this can lead behavioral genetics' estimates to greatly overstate heritability.

I suggest another approach to understanding the BG results on IQ, schooling and income. This approach follows that of Jencks et al [1972], Goldberger [1977] and Duncan et al [2001]. Rather than further trying to "fix" the BG model, let's just accept that this is a simple structural model with strong assumptions and that the model may not be able to deliver causal, out of sample predictions about the effects of the type of environmental interventions of interest to social scientists. The facts from the BG work are that nonadoptive siblings (identical twins) resemble each other much more on certain outcomes than do adoptive siblings (fraternal twins). Clearly that suggests that genes matter a lot. We need not proceed from this fact to a full decomposition of outcome variances into the effects of genes which we do not observe and a single index of shared environment which we do not observe. And if we do implement such a decomposition, one needs to keep in mind that we are decomposing variance within the sample that we have; the causal effects for interventions outside of this range may be bigger or smaller than effects implied by the decomposition. And finally if even if we had the ultimate

¹² To the extent that all we have done is add parameters to the model until we get a decomposition that fits our priors, the approach would not be useful for making predictions or explaining other puzzles.

decomposition, it is unclear that it could be used to make out of sample predictions about the effects of policy changes or the degree to which a shock to an individual will affect her children.

V. Enter the Economists and Sociologists: Treatment Effects and Regression Coefficients

Economists tend to be much more interested in the associations and causal relationships among variables that we do observe, such as parental income and children's schooling. And we tend to study children's health, income, education, marital status and happiness as the outcomes of interest rather than IQ scores and personality traits.

Rubin's causal model [1974] provides an excellent framework for understanding and clarifying what is meant by a "causal effect" or a "treatment effect." According to Rubin (and many empirical economists), in order to measure a causal effect there needs to be an identifiable intervention that could be implemented or not implemented. The causal effect of the treatment on outcome Y for unit i is the difference in potential outcomes that will occur with versus without the treatment being applied. Thus one wouldn't measure the causal effect from being black or female since that it is not a treatment one could apply or withhold. Similarly, one cannot interpret the BG variance decomposition in a strict causal sense since one cannot literally alter the subjects' genes. Nor can one actually move the family environment of a twin or an adoptee by a standard deviation of the BG index of shared (family) environment since this index is a theoretical concept and not observed.

I take this point very literally in Sacerdote [2007] in which I reduce the problem to estimating the causal effect from an adoptee being assigned to one type of family versus another. For example, I calculate the effects on adoptee's educational attainment from being assigned to a family in which both parents have college degrees and there are three or fewer children in the family. More formally I estimate:

$$(7) E_i = \alpha + \beta_1 * T1_i + \beta_2 * T2_i + Male_i + A_i + C_i + \varepsilon_i$$

Where E_i is educational attainment for child i , $T1_i$ is a dummy for being assigned to a family with three or fewer children and high parental education, $T2$ is a dummy for being assigned to a family that either has three or fewer children OR has one or more college educated parents, A_i is full set of single year of age dummies, and C_i are a full set of cohort (year of adoption) dummies. The omitted category are children assigned to large families in which neither parent has a college education.

This has the clear disadvantage of only identifying the effect of a discrete jump in family characteristics like parental education that have more variation than simply "college degree or not." However, the advantage is that the result is very easy to explain and interpret. β_1 is the causal effect on outcomes from being assigned to a particular family type. The family type includes size, parental education and all the observables and unobservables correlated with those two characteristics.¹³ In such an analysis, there is no attempt to make broader statements about the effects of genes or an overarching index of family environment.

Random assignment of adoptees to families plays a critical role in this analysis. It is the lack of correlation between adoptee pre-treatment characteristics and adoptive family characteristics that allows one to give β_1 a causal interpretation.

More broadly, economists and sociologists have used regression to estimate the effects of child and adoptive family characteristics on adoptee outcomes. Examples of this include Plug and Vijverberg [2003], Scarr and Weinberg [1978] and Sacerdote [2002]. A typical equation estimated is of the form:

¹³ For example, the adoptive families that are large and in which neither parent has a college education may have very different unobserved characteristics than the other families. The quality of the school system might be different or the amount of time spent reading to children might be different, β_1 and β_2 will incorporate effects from such unobserved characteristics.

$$(8) E_i = \alpha + \beta_1 * \text{MomsEd}_i + \beta_2 * \text{DadsEd}_i + \beta_4 * \text{Log(Family Income)}_i + \beta_5 * \text{Birth Order}_i + \beta_6 * \text{Male}_i + \varepsilon_i$$

Here E_i represents adoptee i 's years of education while MomsEd_i and DadsEd_i represent adoptive mother and adoptive father's years of education. If one had similar measures for the biological mother and father, those could clearly be added to the equation as well.

This approach loses the bare simplicity of the treatment effects approach in equation (7) but gains a great deal in allowing the reader to think about *which* adoptive family (or biological family) characteristics are most correlated with adoptee outcomes and how steep the slopes are. Social scientists have long used regression to attempt to separate out the effects of different right hand side variables. Clearly selection, measurement error, collinearity and unobservables can potentially bias β_1 - β_4 away from the true treatment effects. But these caveats are well understood and presenting the results in the form of regression coefficients is transparent.

Furthermore, the use of regression coefficients in studying the effects of adoptive family characteristics allows a direct comparison of the results to other studies that attempt to hone in on particular and exogenous shock to family environment. For instance Blau [1999] and Meyer[1997] present evidence that shocks to income itself have only small effects on child education and income. The results from adoption studies appear to confirm this finding (see the following section).

The final and most common approach used by economists is to calculate transmission coefficients of various outcomes from adoptive and biological parents to adoptees. A transmission coefficient takes the form:

$$(4) E_i = \alpha + \delta_1 * E_{Mi} + \gamma * X_i + \varepsilon_i$$

where E_i and E_{Mi} are adoptive child's and adoptive (or biological) mother's education respectively and X_i could be a vector of controls for child gender or age. δ_1 captures the degree to which additional years of education for the mother are transmitted to the child. Again, the great advantage of this approach is that economists already know a great deal about these transmission coefficients. And there is a large literature on transmission coefficients for education and income in general populations. See Solon [1999] and Mazumder [2005].

Calculating transmission coefficients from adoptive parents to adoptees allows us to understand how these transmission coefficients change (are lessened?) when we remove the genetic connection between children and the parents raising them. One can see again why some assumption of random assignment of children to families becomes important. If selection of children into families creates significant positive or negative correlation between the genetic endowments of children and parents, then knowing the transmission coefficient for the adoptees becomes less useful because genetic effects are driving part of δ_1 .

Similarly calculating δ_1 between adoptees and their biological parents is potentially very interesting. This allows us to understand how much of the transmission process remains even when the parents are not involved in raising the child.

VI. Results from Economics on Adoptees

I start by presenting the results on transmissions coefficients since these are the most commonly used tool of economists studying nature and nurture effects. Arguably the best paper on transmission of education and income to adoptees is Björklund Lindahl, Plug [2006] which uses a very large sample of Swedish adoptees who were placed with families. This paper literally uses the census of all Swedish adoptees who were born during 1962-1966 (roughly 5,000 adoptees) and a 20% sample of nonadoptees born during the same time period.

Key results from the Björklund Lindahl Plug study are reproduced in Table VI. This table contains transmission coefficients *from* adoptive and biological parents *to* adoptees and nonadoptees. The outcomes considered are years of schooling, a dummy for completing four years of university, annual earnings, and annual income. The first two rows are for nonadoptees, i.e. children raised by their biological parents. For the nonadoptees, we see transmission coefficients of earnings in the range of .24 which are similar to those for single years of income found in the existing income transmission literature. See Solon [1999], Haider and Solon [2006], and Mazumder [2005]. The transmission coefficients for education of .24 also are similar to those found in the OLS specifications in other studies including Black et al [2005]. Note that whether one uses the father's education or the mother's education on the right hand side of the regression, the coefficients are nearly identical.

There are several remarkable facts about the results for adoptees. First, there is strong transmission of years of schooling (or university status) from both the adoptive parents and the biological parents. Furthermore when one considers the effects of the adoptive and biological fathers, the coefficients are roughly equal in magnitude. Transmission of years of schooling from biological fathers to adoptees has a coefficient of .113 and transmission from adoptive fathers to adoptees is .114. And the two transmission coefficients for adoptees add up to .227 which is roughly equal to the .240 transmission coefficient of schooling for the nonadoptees.

This apparent additivity of the transmission from biological parents and nurturing parents is extremely interesting and can be seen in roughly five of the six columns in Table VI. For example, transmission of income from an adoptee's biological father is .06 and .17 from adoptive father's and this adds up to .23. The transmission coefficient for nonadoptees is .24. Björklund, Jäntti, and Solon [2007] explore more deeply this additive property. They find that a simple additive model explains the data quite well. Note that for adoptee earnings, BLP find that adoptive fathers are a more important source of transmission of earnings. For schooling, adoptive and biological fathers seem to matter about equally.

BLP also ask whether there are statistically significant effects from interacting biological and adoptive parent characteristics. They do not find strong evidence of interaction effects. This finding is not surprising given that we already noted above that within their data, the entire transmission coefficient for nonadoptees can be explained by the main effects of adoptive and biological parent characteristics.

So the bottom line from the BLP study appears to be that transmission of earnings and education works strongly through both biological channels and through environmental channels. To say a bit more about the relative importance of the two channels, I now turn to transmission coefficients found in other adoption studies.

One caveat to the BLP study might be potential selective placement of adoptees into Swedish families and that this might affect their findings on the sources of transmission. For example positive selection of healthier adoptees into high income families might cause BLP to overstate how much transmission comes from the nurturing parents. In Sacerdote [2007] I am able to provide transmission coefficients for a set of Korean American adoptees whose assignment to US families was effectively random. Holt used a queueing system to assign children to families and I provide evidence that this yields quasi-random assignment of children to families.

Table VII provides estimated transmission coefficients from 4 different adoption samples including the BLP study, the Holt study, the National Longitudinal Survey of Youth 1979, and the Wisconsin Longitudinal Study analyzed in Plug [2004]. I report figures for both the transmission of years of education and transmission of a dummy variable for having completed four or more years of college. The upper panel is for the nonadoptees and the lower panel is for the adoptees. These are coefficients for transmission from mothers to children.¹⁴

¹⁴ Switching to fathers would not affect the Holt numbers, but it would raise the transmission coefficients for adoptees in the BLP data.

For the nonadoptees, the Holt, BLP, NLSY samples deliver transmission coefficients that are roughly in the .25-.40 range. The NLSY numbers tend to be at the higher end of this range. It is possible that this stems from nonlinearities in transmission combined with the greater range of parental education in the NLSY data. The Wisconsin data deliver a large transmission coefficient of .54 for years of education, but the transmission coefficient for "college graduate" status in the WLS sample is .385. This latter number is in line with the results found in the other three samples.

The transmission coefficients from adoptive mothers to adoptees show a different pattern. Both the Holt and the BLP samples of adoptees have coefficients that are within one standard error of each other. Transmission of years of education is about .08 and transmission of college status is about .12. The other two samples yield significantly larger transmission from adoptive mothers to children. One natural explanation for this finding is that the two smaller samples (NLSY and WLS) have strong positive selection of adoptees into families in which the healthiest or most naturally able infants were more likely to be adopted by the higher education mothers. On the whole, comparing the transmission coefficients for the adoptees to those for nonadoptees gives the impression that adoptees receive from their adoptive mothers about 1/4 to maybe 1/2 of the transmission effects that nonadoptees receive. The transmission coefficients from adoptive mothers to adoptees are lower than the BLP results using adoptive fathers. Nonetheless both the biological parents and the nurturing parents matter a great deal. I cannot reject the hypothesis that the two sources of transmission influences are equal in size, though the point estimates of Table VII indicate that transmission to adoptees via nurture is less than half of total transmission to nonadoptees.

In Table VIII, I report the results on income transmission for the Holt and BLP samples and the Panel Study of Income Dynamics as analyzed by Liu and Zeng [2007]. In the case of BLP this is transmission of income from fathers to children and averages over multiple years of income for both. In the Holt sample, this is a single survey report in which respondents choose from among ten categories of income. Since I also had administrative data on income at the time of adoption, I instrumented for the survey

measure of family income with the administrative measure. In both samples, transmission for nonadoptees is about .24 and transmission for adoptees is about .18. This would indicate that the income transmission process is substantial even without a biological connection between parent and child. Since the measurement of income in the Holt sample is less than ideal, I do not want to lean too heavily on the Holt result in reaching this conclusion. Liu and Zeng [2007] find a larger transmission coefficient for the nonadoptees than do the other two studies and they attribute this fact partially to the fact that they are using earnings for older offspring.¹⁵

A related and interesting question is how the transmission process from parents to adoptees and nonadoptees differs when one looks across different outcomes. Figure I graphs transmission coefficients for nine different outcomes for adoptees and nonadoptees in the Holt sample. The vertical axis is for the transmission coefficient for nonadoptees and the horizontal axis is used for the transmission coefficient for the adoptees. Outcomes close the 45 degree line such as drinking and smoking are transmitted equally strongly from parents to adoptive and nonadoptive children. Not surprisingly height is very heavily transmitted to nonadoptees and not at all to adoptees. The pattern one notices in Figure I is that physical outcomes like obesity and height show very little transmission to adoptees while social outcomes like moderate drinking require no genetic connection for transmission.¹⁶ Education is somewhere in between.

As discussed in the preceding section, one of the advantages of using multiple regression in this context is that it allows one to regress adoptee outcomes on a host of factors and to potentially make inferences about which factors have the largest and most statistically significant influences on adoptees. I did this in Sacerdote [2007] for adoptee's years of education and a very clear pattern emerged. The two adoptive family characteristics that are statistically significant predictors of adoptee educational attainment are family size and mother's education. Each additional year of mother's education is associated with an extra .09 years of education for the adoptee. Each

¹⁵ Haider and Solon [2007] and Böhlmark and Lindquist [2006] address how the ages at which earnings are measured affects the measured transmission coefficients.

¹⁶ In contrast a large part of the transmission of alcoholism may be genetic (Cloninger et al [1981]).

additional child in the family is associated with a statistically significant decrease of .12 years. These facts remain true regardless of what additional controls are added.

The strong finding with regard to family size indicates that either family size is correlated with some important unobservables about the family (as suggested by the findings of Black Devereux and Salvanes [2005b]) or there are indeed direct effects from family size. In fact in later work, Black Devereux and Salvanes [2007] find that unexpected increases in family size do have significant negative effects on achievement. Family size in the adoption data covaries with other important family characteristics, and thus one cannot be certain that the effects I find are strictly causal effects from family size itself. However, the adoption results are certainly suggestive and push social scientists towards better understanding the mechanisms by which family environment affects outcomes.

Consistent with Blau [1999] and Mayer [1997], controlling for other family characteristics there is no direct impact from family income. This remains true regardless of how I employ the four measures of parental income in the data set. For the nonadoptees in the sample, the income measures generate transmission coefficients that resemble those in other data sets so this unlikely to be purely a story of measurement error.

In order to make some broad causal statements about the effects of family environment on adoptee outcomes, I then asked about the treatment effects on an adoptee from being assigned to a small, high education family. Here small means three or fewer children and high education means that both parents have college degrees. The measured treatment effects of family environment shifts on adoptee outcomes are quite large. For example, assignment to a small high education family leads to a 16 percentage point increase in the likelihood of graduating from college, relative to assignment to a large family where neither parent has a college degree. That effect is on a mean of about 58 percent of adoptees graduating from college.

VII. Putting It All Together: What Does It Mean?

A review of the behavioral genetics literature and the recent economics literature on nature and nurture effects yields several conclusions. First the BG estimates of the heritability of certain outcomes including IQ are incredibly robust. The canonical result is that adult IQ is about 50 percent heritable and that for adults, little of the remaining variation is attributable to family environment. The numbers are somewhat similar for decompositions of the variance of educational attainment. Behrman and Taubman [1989] and Teasdale and Owen [1984] find no role for family environment in determining years of education although Behrman Taubman and Wales [1977] found substantial effects from family environment on schooling. The finding of no role or only a small role for family environment in determining educational attainment and income also appears to be relatively robust within the BG framework. However as Björklund, Jäntti and Solon [2005], Jencks et al [1972], and Goldberger [1977] and others have noted, that fact need not have major implications for social scientists' investigations of the merits or treatment effects from changes in environment. For example, the variance decomposition may not incorporate the environmental shifts being contemplated.

Furthermore, a tremendous amount of work has recently been done to make the structural BG model more sophisticated. Dickens and Flynn [2001] model the potential endogeneity between genes and environment. Turkheimer et al [2003] deal with the nonlinear nature in which genes and environment translate into outcomes.

Implementing such decompositions and then applying the results out of sample is so challenging that economists have recently bypassed the problem of fully identifying nature and nurture effects. Instead we have calculated transmission coefficients from parents to children for adoptees and nonadoptees. This delivers an estimate of how much of the transmission of education, income or some other outcome takes place even in the absence of a genetic connection between parents and children. The resulting picture is one which appears to be quite plausible and to match what we know about the potency of environment from experimental interventions in school characteristics or neighborhood

characteristics (See Katz, Kling and Liebman [2001]). For example, Björklund Lindahl and Plug find that about half of transmission of education to adoptees works through biological parents and about half works through adoptive parents.

In some sense, the more we learn about the effects of environment on children's outcomes, the more we see a picture which fits the existing data and parents' intuition. Surely it would be difficult to deny that genetic effects matter. Just look at how much more biological siblings resemble each other on education and income than do adoptive siblings. At the same time, there are potent environmental effects observed from assigning an adoptee to one type of family versus another. Many social scientists have the intuition that differences in school quality and home environment can explain a lot of inequality of average outcomes that is observed. This intuition may be right. For example, the black white gap in college completion rates in the US is roughly 15.4 percentage points. Even within the family environment variation observed in the Holt sample, I observe similarly large gaps in Korean-American adoptee outcomes from the assignment to one family environment versus another. Overall, it appears that economists' work with adoptees will help create a consistent picture of what aspects of family environment matter and how much they matter.

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Table I
Correlations in IQ Between Siblings, Adoptive Siblings, and Identical Twins

The table reports results from four surveys of the IQ literature and incorporate hundreds of individual studies of twin and adoptee samples.

<u>Meta Study Authors</u>	Number of Studies Considered	Correlation for Siblings Raised Together (Nonadoptive, non identical twins)	Correlation for Adoptive Sibs	Correlation for Identical Twins	Correlation for Fraternal Twins
Devlin, Daniels and Roeder (1997)	212	0.44		0.85	
Bouchard and McGue (1981)	69	0.45	0.29	0.85	
Golberger (1977)	7	0.51	0.31	0.91	
Jencks at al (1972)	18	0.54	0.42	0.86	0.58

Data for Jencks at al are as summarized by Taylor[1980] p, 46.

Table II
IQ Results: Implied Variance Decomposition from the Behavioral Genetics Model

	Variance Attributable to Additive Genetic Effects	Variance Attributable to NonAdditive Genetic Effects	Total Genetic	Variance Attributable to Common Environment	Non-Shared Environment
<u>Meta Studies</u>					
Devlin, Daniels and Roeder (1997)	0.34	0.15	0.49	0.05	0.46
Golberger (1977)	0.47	0.11	0.58	0.22	0.20
Bouchard and McGue (1981) MZ vs DZ Twins*			0.54	0.04	0.42
Bouchard and McGue (1981) Adoptees*			0.32	0.29	0.39
<u>Individual Studies</u>					
Cherny and Cardon (1994) (For 9 year old Adoptees and Sibs)			0.60	0.16	0.24

* Bouchard and McGue do not calculate estimates of heritability from the sibling correlations they aggregate. Loehlin (1989) does this calculation using the Bouchard and McGue aggregates does not split environmental effects into common (family) and non-shared. I calculated these using the simple version of the BG model in equations (4) and (5).

Table III
Years of Education: Implied Variance Decomposition from the Behavioral Genetics Model

Authors and Sample	Variance Attributable to Additive Genetic Effects	Variance Attributable to NonAdditive Genetic Effects	Total Genetic	Variance Attributable to Common Environment	Non-Shared Environment
Behrman and Taubman (1989) 2,000 twins pairs and their relatives NAS-NRC sample	0.88 (.002)	-0.01 (.047)	0.88		
Scarr and Weinberg (1994) 59 adoptive sibling pairs and 105 nonadoptive sibling pairs			0.38	0.13	0.49
Teasdale and Owen (1984) 163 pairs of adoptees from Danish National Register	0.678		0.678	0.052	0.270
Behrman, Taubman, and Wales (1975) 2,478 MZ and DZ Twins in the NAS-NRC sample			0.36	0.41	0.23

Scarr and Weinberg (1994) report adoptive and biological sibling correlations. I used equations (4) and (5) to translate this into the decomposition implied by the simplest form of the BG model. Teasdale and Owen report their results in variance of years of education explained by additive genes, common environment and separate environment. I calculated the fractions explained by each factor. The NAS-NRC sample is a National Academy of Science - National Research Council survey of twins performed in 1974.

Table IV
Earnings: Implied Variance Decomposition from the Behavioral Genetics Model

Authors and Sample	Variance Attributable Genetic Effects	Variance Attributable to Common Environment	Variance Attributable to Non-Shared Environment
Björklund, Jäntti and Solon (2005) Model 1 Swedish Brothers Including Raised Apart, Together, Twins, Adoptees, Half Sibs	.281 (.080)	0.038 (0.037)	0.681
Björklund, Jäntti and Solon (2005) Model 1 Swedish Sisters Including Raised Apart, Together, Twins, Adoptees, Half Sibs	.245 (.080)	0.009 (0.037)	0.746
Björklund, Jäntti and Solon (2005) Model 4 Swedish Brothers Including Raised Apart, Together, Twins, Adoptees, Half Sibs	0.199 (0.157)	0.164 (0.158)	0.637
Behrman, Taubman, and Wales (1975)	0.45	0.13	-0.42

Björklund, Jäntti and Solon estimates the BG parameters to fit the nine sibling correlations in the data from nine sibling types (MZ raised together, MZ apart, DZ together, DZ apart, Full sibs together, full sibs apart, half sibs together, half sibs apart, adoptive sibs). The difference between models 1 and 4 is that model 4 adds parameters to allow for different degrees of environmental correlation among different types of sibling pairs.

Table V
Behavioral Genetics Results on Personality Traits
Meta Study of Correlations Between Parents and Children

<u>Dimension</u>	Parent Child Relationship		
	Biological and social	Social, not biological	Biological, not social
Extraversion	0.14 (117, .010)	0.03 (40, .011)	0.16 (15, .019)
Agreeableness	0.11 (65, .013)	0.01 (16, .021)	0.14 (3, .067)
Conscientiousness	0.09 (64, .013)	0.02 (26, .012)	0.11 (2, .110)
Neuroticism	0.13 (131, .010)	0.05 (40, .011)	0.11 (21, .022)
Openness	0.17 (24, .028)	0.07 (12, .031)	0.14 (1 -)

This is a summary of the literature on personality traits and is reprinted exactly from Loehlin (2005) Table 6.3. Number of correlations that were averaged and the implied standard errors are in parentheses.

Table VI**Transmission Coefficients from the Björklund, Lindahl, Plug [2006]**

This reproduces most of BLP [2006] Table II. Sample sizes are roughly 2,000 adoptees and 90,000 nonadoptees. Each transmission coefficient is from a separate regression of child's outcome on parents outcomes for years of schooling, a dummy for having 4 years of university, earnings and income. The latter two variables are averaged over multiple years. All data are from the Swedish National Registry.

	(1)	(2)	(3)	(4)	(5)	(6)
	Years of Schooling	Years of Schooling	University	University	Earnings	Income
<u>Non Adoptees</u>						
Biological father	.240** (0.002)		.339** (0.004)		.235** (0.005)	.241** (0.004)
Biological mother		.243** (0.002)		.337** (0.004)		
<u>Adoptees</u>						
Biological father	.113** (0.016)		.184** (0.036)		0.047 (0.034)	.059* (0.028)
Biological mother		.132** (0.017)		.261** (0.034)		
Adoptive father	.114** (0.013)		.165** (0.024)		.098** (0.038)	.172** (0.031)
Adoptive mother		.074** (0.014)		.145** (0.024)		
Sum of estimates for bio and adoptive fathers	.227** (0.019)		.349** (0.040)		.145** (0.049)	.231** (0.040)
Sum of estimates for bio and adoptive mothers		.207** (0.021)		.406** (0.039)		

Table VII
Transmission of Education in Five Samples of Adoptees and Nonadoptees

	Transmission of Years of Education (Mother- Child)	Transmission of 4+ Years College (Mother-Child)	N
Holt Non Adoptees	0.315 (0.038)**	0.302 (0.037)**	1,213
Swedish Non Adoptees	.243 (.002)**	.337 (.004)**	94,079
Swedish Non Adoptees (Holmlund et al)	.280 (.001)**		570,555
NLSY Non Adoptees	.401 (.011)**	.440 (.018)**	5,614
WLS Non Adoptees	.538 (.016)**	.385 (.015)**	15,871
Holt Adoptees	0.089 (0.029)**	0.102 (0.034)**	1,642
Swedish Adoptees	.074 (.014)**	.145 (.024)**	2,125
Swedish Adoptees (Holmlund et al)	.030 (.010)**		4,603
NLSY Adoptees	.277 (.060)**	.420 (.078)**	170
WLS Adoptees	.276 (.063)**	.178 (.063)**	610

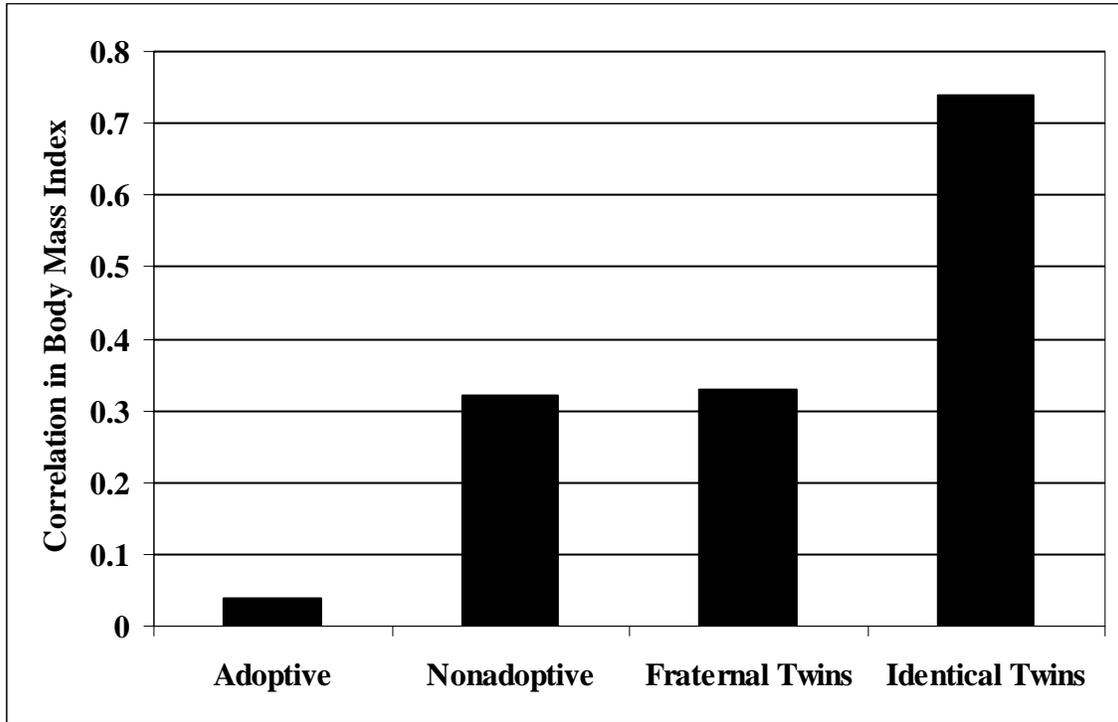
I report transmission coefficients for education and income in the Holt Sample, my calculations from the NLSY79, Björklund et al's [2006] for Sweden, Plug [2004] for the Wisconsin Longitudinal Survey (WLS). The adoptees in BLP's study are ages 35-37 in 1999. The adoptees in Plug's study are ages 23 and older. For the NLSY data I use adoptees ages 28-36 in 1993. Swedish international adoptions are analyzed by Holmlund, Lindahl and Plug [2005].

Table VIII
Transmission of Income in the Holt and Swedish Samples and the PSID

	Transmission of Log (Income)	N
Holt Nonadoptees	0.246 (0.080)**	1,196
Swedish Nonadoptees	0.241** (0.004)	91,932
Panel Study of Income Dynamic Nonadoptees	0.369** (0.018)	4,160
Holt Adoptees	0.186 (0.111)	1,209
Swedish Adoptees	.172** (0.031)	1,976
Panel Study of Income Dynamic Adoptees	0.096 (0.121)	120

I report transmission coefficients for education and income in the Holt Sample, Björklund et al's [2006] for Sweden and Liu and Zeng [2007] for the Panel Study of Income Dynamics (PSID).

**Figure I: Correlations in Body Mass Index
For Four Types of Sibling Pairs**



Data are from meta-study done by Grilo and Pogue-Geile [1991]. Numbers for adoptive siblings add results from Sacerdote [2007] since Grilo and Pogue Geile have only one study with BMI figures. All calculations include same and mixed gender pairs.

Figure I: Comparison of Coefficient of Transmission from Parent to Child

Reproduced from Sacerdote [2007]. Graph shows coefficient from a regression of child's outcome on mother's outcome for adoptees and nonadoptees in the sample.

