Modeling the Cultural and Biological Inheritance of Social and Political Behavior in Twins and Nuclear Families

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Abstract

There has been growing interest in the possibility that biological factors, including genes, might contribute to differences in political and social behavior. Over the last 40 years, human statistical and behavioral genetics have developed a variety of models that allow the effects of genetic and non-genetic inheritance to be estimated from human kinship data. Until quite recently, these methods were unknown to political scientists and there has been no systematic attempt to illustrate the general approach to modeling genetic and social influences on differences in complex human social traits in the political science literature. The current chapter seeks to provide political scientists with the elements of a conceptual and methodological toolkit for analyzing overall biological and social influences on socially significant political outcomes. We introduce approaches that may be used to analyze family resemblance and estimate the contributions of multiple genetic and environmental sources if individual differences. The approach is illustrated by analysis of the correlations between pairs of adult spouses (N=8,287 pairs), parents and offspring (N=25,018), siblings (N=18,697), monozygotic (N=4,623) and dizygotic (N=5120) twin pairs comprising an informative subset of relationships from studies of extended kinships of twins in Virginia and Australia. The two studies comprise self-reports from a total of approximately 50,000 relatives. Six illustrative physical and behavioral variables were chosen to reflect potentially different mechanisms of familial resemblance and transmission: stature, conservative-liberal orientation; neuroticism; church attendance; political affiliation; educational attainment. The effects of assortative mating (the tendency for non-random pairing of potential mates) were incorporated in the model for biological and cultural inheritance and tests conducted for sex differences in the effects of genes and environment. The results showed that the two samples gave comparable best fitting-models for each variable. Family resemblance in stature is explained almost entirely by the additive and dominant effects of genes. The same model accounted for differences in neuroticism, though the overall genetic contribution was far smaller. By contrast, the correlations between relatives for political affiliation were entirely due to environmental influences, including a large direct influence of parental political affiliation on their offspring. The other variables showed a mixture of genetic and non-genetic influences on family resemblance. The contribution of parents to children was largely genetic, but other environmental factors shared by siblings and twins made significant contributions to variation in these outcomes. The correlation between mates for personality (neuroticism) was extremely small, modest for stature, and marked for conservatism, educational attainment, church attendance and political affiliation. All spousal correlations are positive. The final model for political affiliation is consistent with a complex of long- and short-term social influences within and between families. The results for a major dimension of social attitudes (conservatism-liberalism) suggest a mixed model that implicates both social and biological influences. Data on the spouses of twins and siblings are used to test a variety of assumptions about the processes underlying the correlations between mates. The analyses strongly support the view that mate selection depends mainly on the direct choice of spouses for the measured traits ("phenotypic assortative mating") rather than selection for family background ("social homogamy") or mutual reinforcement as a result of spousal interaction. Longitudinal data are presented on the development of social attitudes in adolescent that reveal a stark contrast between the causes of individual differences among juveniles, which are entirely due to the effects of the shared environment, and those in adults in which the effects of genetic differences are much more marked. Implications and limitations of the model, design and statistical method are noted. Proposals for future study are considered.

Introduction

The dominant paradigm in the social and political sciences embodies the theory that the origin and transmission of behavioral differences is social. Indeed, Emil Durkheim's (1895) view that social behaviors could only be explained by social indicators has only recently been challenged. Lumsden and Wilson (2005) refer to this as the theory of the "Promethean genotype", i.e. that human evolutionary history has emancipated humans entirely from the influence of their genes. Thus, the effects of genetic inheritance on social and political behavior can safely be ignored and the social sciences can proceed with little reference to biology. Notwithstanding several publications in the life and behavioral sciences that have explored the role of genetic differences in normal and abnormal human behavior, including social, religious and political attitudes and behavior (Eaves and Eysenck 1974; Eaves et al. 1978; Martin and Jardine 1986; Martin et al. 1986; Eaves et al. 1989; Truett et al. 1994; Tesser 1993; Eaves et al. 1999ab; Bouchard et al. 1990; Bouchard and McGue 2003; Eaves et al. 2008), political science had remained largely unaware of or immune to such work. Recent exceptions to this trend (Alford, Funk and Hibbing 2005; Dawes and Fowler 2009; Fowler, Baker and Dawes 2008; Fowler and Dawes 2008; Hatemi et al 2007; Hatemi, Medland and Eaves 2009; Hatemi et al 2009a; 2009b) suggested that some political scientists were beginning to consider the implications of an alternative paradigm, grounded in the theory that, even in the domain of their primary interest, political science cannot entirely ignore the effects of genetic influences.

The current chapter is intended to provide a theoretical and empirical framework for evaluating the roles of biological and cultural inheritance in the transmission of human social and political behavior. Theoretically we take the position echoed by the life and social sciences that there can be no organism without both genes and environment and that the study of *individual* differences, which includes the roles of genes and environments, rather than differences between larger geographical, ethnic or cultural groups may give greater insight about the ontogeny of human differences. In any specific case, "social" and "genetic" theories may be treated initially as competing models that should be evaluated in terms of their ability to account for data on patterns of variation and correlation between relatives. Ultimately, however, it is likely that elements of both models are required in most circumstances. Our methodological perspective relies on the ability of empirical observations about the transmission of individual differences in human families to determine which model is to be preferred with what quantitative parameter values. We focus on three main themes: analyzing the effects of genes and environment on the correlations between relatives; exploring alternative explanations of the correlations between mates; the changing roles of genes and environment in attitude development. Thus:

1. We apply representative models of cultural and biological inheritance to a selected range of human physical, behavioral and socially significant variables. The data comprise the correlations between relatives derived for two very large studies of twins and their relatives in the United States and Australia (N=c. 50,000 adult subjects). We explain how theories of biological and cultural inheritance can be formulated and tested in different constellations of family relationships. We show that no single model (biological or social) is adequate to explain the pattern of inheritance for all of the traits selected to illustrate the approach. Rather, different outcomes, for different traits, illustrate different combinations of genetic and social influences with the implication that a far more nuanced understanding of both will be needed for a comprehensive understanding of human political behavior.

2. We conduct a preliminary analysis of some possible mechanisms of mate selection in the attempt to determine how far similarity between spouses is due to their own choice of partners like themselves for the behavior measured ("phenotypic assortative mating"), or selection based on a correlated variable, such as family and social background ("social homogamy") or simply a function of social interaction between partners after they establish their relationship ("spouse interaction"). Again, we do not pretend to offer an exhaustive treatment of all the subtleties and models of mate selection but we hope we present enough data to illustrate how data on marriages of family members can be used

to resolve some of the principal mechanisms. Although the data are not definitive, the analyses we present suggest that much of the data are consistent with phenotypic assortative mating (as assumed in "1" above) rather than social homogamy or spousal interaction.

3. We examine the developmental trajectory of genetic and environmental influences on the development of liberal/conservative social attitudes across the life span. The analysis demonstrates how the contributions of genes and environment change with age, most markedly at about the time when most children leave home, and illustrates the cumulative longitudinal influence of the social environment on the development of social attitudes during adolescence. The analysis shows that a dynamic understanding of the interplay between individuals and their social milieu is essential for a theory of social behavior.

Although our final goal is the substantive scientific understanding of significant features of human variation, our approach is expository in the hope that it will introduce political scientists to a significant and long-standing corpus of method and theory that may be helpful in their own work. After a brief review of the principal contours of the history of modeling family resemblance, we outline some of the questions that are being asked and then describe the model data set and statistics that will be used in the attempt to answer them. Next, we show how formal models can be developed for the contributions of genetic and social influences to human differences and initially apply the approaches separately to two types of data, nuclear families and twins and then attempt an integrated analysis of both sets of data in order to develop a more nuanced and general model.

Within this introductory scope a wide range of themes relevant to the study of genetic and environmental influences on human behavior will remain unexplored. Our goal is to provide a basic framework and ideas that, we hope, will engage a new generation of scholars in a way of thinking that may be unfamiliar but worth exploring. However, in this chapter, we do not consider how genes and environment contribute to the patterns of covariation between multiple outcomes ("the genetic analysis of covariance structure", Martin and Eaves 1977; Behrman, Taubman and Wales 1977, Neale and Cardon 1992). Many of the more challenging questions involve attempts to characterize the multiple facets of the environment to identify the multiple biological and social pathways from DNA to social behavior. Such studies are inherently multivariate and we hope that the motivated reader will use this chapter as a solid foundation from which to explore models involving the effects of genes and environments on multiple variables. All the models we consider do not model the effects of genotype x environment interaction. That is, the models assume that the behavior ("phenotype") of an individual can be approximated by the sum of his/her individual genetic and environmental deviations from their corresponding population means. Studies of experimental plants, humans, animals and microorganisms demonstrate that this is an approximation. Some genes affect the phenotype by increasing or reducing an individual's sensitivity to their environment. Some environments may restrict or facilitate the expression of genetic differences. The more our studies address complex behaviors that rely on the human capacity to evaluate and respond to the world cognitively and emotionally, the more plausible it becomes that such genotype x environment interactions ("GxE") are a significant feature of the genetic architecture of human behavior. Mather and Jinks (1982) provide a basic conceptual framework for the detection, analysis and significance GxE in experimental organisms. There are several approaches to the analysis of GxE in humans (see e.g. Jinks and Fulker, 1970; Kendler and Eaves 1986; Caspi et al., 2002; Rutter and Silberg 2002; Turkheimer et al. 2003; Eaves et al. 2003) that have met with varying degrees of success in practice. This aspect of the field is still fluid and time will tell how important such interactions are, relative to the main effects of genes and environment, as influences in the development of human behavior. A theme related to GxE that is especially relevant to political behavior and values is the interaction between the expression of genetic differences and secular change. It is quite conceivable that different epochs facilitate or suppress the expression of genetic differences. Boomsma et al. (1999) offer an intriguing example of the differential expression of genetic differences in juvenile behavior as a function of the difference between a liberal

secular upbringing compared with a more conservative religious one. The role of such influences in the social and political arena is extremely plausible but little explored at this point.

We have resisted the temptation to address methods for identifying the effects of specific genes on political values and behavior. This is partly a matter of space and partly because the field is still in such a state of flux that it would be premature to say too much. Thirty years of dedicated work, with ever more refined laboratory methods, increasingly large samples, and statistical approaches designed to minimize the effects of data-grubbing in the analysis of horrendously large number of potential predictors are only now reaching a point at which there is an emerging consensus that the number of genes underlying differences in most human traits is very large and their effects, though large in aggregate, appear individually small. There are, of course, exceptions and we never know in advance what those might be, but it seems that for the most part Fisher's original conjecture (1918), borne out by decades of careful study in experimental organisms (see e.g. Mather and Jinks, 1982), is no less true of differences in human behavior, i.e., that the number of genes is large and their individual effects are small (see e.g. Visscher 2008; International Schizophrenia Genetics Consortium 2009).

The current treatment is designed to help the newcomer gain some sense of a way of thinking that may be unfamiliar and constitutes an invitation to deeper study. We are conscious of steering a difficult course between appeal to intuition and rigor. The best way to become engaged in a new discipline is to think about real data. We, thus, devote a lot of space to "looking at the numbers" in preparation to developing and testing more formal models. In many places, we have chosen to sacrifice some statistical purity in our methods of estimation and model-testing in the interests of making the connection between the data and results more transparent. Methodologically-minded readers who have some familiarity with linear modeling and the elements of path analysis should have no problem tracing the details of the argument. Serious students who are coming to genetic ideas and models for the first time may need to get some practice in the basic concepts beyond that offered in the current chapter. We recommend reading some background material to help make the transition to an approach that might seem unfamiliar at first. Some of the more basic principles of modeling from a slightly different perspective are presented in the chapter by Eaves et al. (2005) in the introductory text edited by Kendler and Eaves (2005). The classic introduction to path analysis by Otis Dudley Duncan (1966) is helpful in giving some foundation to applications in territory more familiar to social scientists. David Fulker's (1979) chapters in Eysenck's volume on intelligence are a didactic masterpiece that translates easily from the analysis of intelligence and socio-economic variables to studies in political science. Greg Carey (2003) has an excellent broader introduction specifically written for students in the social sciences. John Loehlin (2003) offers a characteristically thoughtful and lucid exposition of linear structural modeling. The examples presented in Neale et al's. manual (2002) for the "Mx" program for structural modeling are well tried and tested. These examples, and many others, are available online and have evolved over some 20 years of experience in teaching the elements of genetic modeling to behavioral and social scientists in a series of International Twin Methodology Workshops. Readers are encouraged to use these resources as best suit their needs.

Modeling Genetic and Environmental Contributions to Family Resemblance.

The mid-nineteenth century saw the emergence of two significant, but virtually independent biological theories of heredity and variation. Gregor Mendel's careful "experiments in plant hybridization" (1865) formulated and tested the classical theory of particulate inheritance that now bears his name. Mendel deliberately chose his experimental material and the traits of interest with great care and was able to capture with mathematical precision the properties of inheritance in a model that was vindicated in the next century by actual biological observations on the behavior of chromosomes (e.g. Sutton, 1903), the inheritance of inborn errors of metabolism (Garod, 1923) and the molecular basis of heredity (Watson and Crick, 1953). About the same time that Mendel was studying particulate inheritance of differences in families of peas, Francis Galton (e.g. 1869) began the search for comparable "laws" for the inheritance of

differences in human families, culminating in the classic assembly of data on the correlation between relatives for physical traits such as stature by Karl Pearson and Alice Lee (1903). At the turn of the 19th century, a debate raged, sometimes acrimoniously, between those who were viewed as squeezing all human differences into the procrustean bed of Mendelism and those, notably Karl Pearson, who sought to explain differences, even those discrete categories such as eye-color, in terms of Galtonian inheritance. The abiding genius of Ronald Fisher (1918) provided the synthesis that remains virtually unchallenged almost a century later. He showed that the correlations between relatives for continuous human traits amassed by Galton and his student Karl Pearson could be explained by reference to Mendelian principles with only a few simple modifications of Mendel's original theory. In particular, Fisher considered that variation in continuous traits resulted from the accumulation of the very small effects of differences at a very large number of individual genes (hence "polygenes") each behaving in strict accordance with Mendel's earlier laws. In making this generalization, Fisher realized that three other modifications might be needed to account for the data. First, he noted that the genes underlying continuous variation did not necessarily adhere strictly to the concept of "dominance" (see Appendix 1) as it was originally conceived by Mendel. Rather, different genes may show intermediate levels of dominance or even no dominance at all ("genetic additivity"). Secondly, Fisher realized that humans did not mate at random, but that like tended to marry like to a greater or lesser extent ("assortative mating") with the result that genetic variance and the genetic resemblance between relatives might be inflated by the accumulation of correlation between increasing and decreasing effects at otherwise independent genetic loci. That is, the assumption that purely genetic traits in offspring will show a correlation of ½ with parents depends on the assumption that their biological parents are not correlated for their own genetic predisposition for the same traits. In short, random mating for a trait ensures that the genetic liabilities of spouses are also uncorrelated for the same trait. This assumption is most certainly violated if mate choice itself is based on the trait of interest, or on variables that are correlated with the trait. If parents tend to be correlated for their genetic liabilities as a result of mate selection, then they tend also to transmit to their offspring genetic liabilities that are more similar that would be expected if parents mated at random, thus increasing the correlation between relatives. The *genetic* consequences of assortative mating perceived by Fisher, of course, extend to the *environmental* similarity between relatives for traits that are transmitted socially within families. Finally, Fisher noted that the observed characteristics of the individual (the "phenotype") correlated only imperfectly with the underlying genetic liability ("genotype") because of the intervening influences of the environment. Fisher adopted a very simple and, from the standpoint of the social sciences, unrealistic model of environmental influences by choosing to consider only those random effects that did not correlate between family members. It is remarkable that Fisher's polygenic theory of continuous variation is only now being vindicated at the molecular level by the demonstration that variation in a very large number of individual genes appear to affect differences in complex variables such as human stature (Visscher, 2008).

Francis Galton's own inquiry into "hereditary genius" (1869) led him to recognize (1883) the dangers of too facile generalization from family resemblance to biological theories of inheritance of complex behavioral traits and to conceive of the potential value of twins, especially monozygotic (MZ) twins to reveal effects of the environment that were otherwise confounded with genetic inheritance. Galton's own twin data (1883), amassed by anecdotal self-reports, were instructive qualitatively but fell far short of a quality and quantity needed to survive rigorous scrutiny. Newman, Freeman and Holzinger (1937) published an early study of twins that included identical twins separated at birth but did not include assessment of social and political behavior. Bouchard and his colleagues (Bouchard et al., 1990; Bouchard and McGue, 2003) examined the correlations in social attitudes for a more recent sample of twins separated at birth, showing that the results for the attitudes of separated twins bore out earlier conclusions based on the much larger studies of twins reared together (see e.g. studies described in Eaves et al. 1989) and other relationships that genetic factors may play a significant role in creating individual differences in these aspects of human behavior.

The mathematical elegance of Fisher's classical model of polygenic inheritance remained largely unmatched by theories of non-genetic inheritance until the early 1970's when Cavalli-Sforza and Feldman (e.g. 1973, 1981) generalized the mathematical theories of classical population genetics to reflect the non-genetic transmission of information from parent to child through social learning from the parental phenotype. They subsequently applied their models to attitudes data obtained from (non-twin) kinship data (Cavalli-Sforza and Feldman, 1982). A practical limitation of this seminal work was the fact that it considered the direct transmission of discrete cultural differences. Newton Morton (1973), aided subsequently by several others (e.g. Rao et al., 1974) reintroduced the principles of Sewall Wright's (1921) method of path coefficients to modeling both genetic and non-genetic correlation between relatives that culminated in 1979 with the publication of a generalization of Fisher's model for polygenic inheritance to include the simultaneous non-genetic transmission from parent to child (Cloninger et al., 1979). Path analysis forms a helpful methodological point of contact between genetics and the social sciences through the comparable influence of Otis Dudley Duncan's classical sociological application of the approach to the study of the causal relationships between socio-economic outcomes (1966). Christopher Jencks's study of the familial transmission of differences in IQ (Jencks et al., 1972) provides an early application of this approach in the social sciences and that of income and occupational status in twins (Behrman et al., 1977) illustrates a seminal application in econometrics. Among the many criticisms of such work, by far the most penetrating were those of Arthur Goldberger (e.g. 1978). Many of these early criticisms translate, *mutatis mutandis*, from genetic studies of differences in intelligence to those of social, religious and political attitudes and behavior.

This brief historical portrait serves merely to sketch the crude contours of the approach that informs the current study. There are many nuances of genetic and environmental influences that have featured in theoretical and empirical investigations in the last three decades. These include: analysis of environmental effects of maternal genes (e.g. Nance and Corey, 1976); sex differences in the expression of genetic and environmental influences (e.g. Eaves, 1977) ; social interaction between siblings (e.g. Carey, 1986); alternative mechanisms of mate selection (e.g. Heath et al. 1985; Heath 1987); genotype-environment interaction and correlation (see references above); developmental change in the expression of genetic and environmental effects (e.g. Eaves et al., 1986); and multivariate genetic analysis (e.g. Neale and Cardon, 2003) to name a few. Some of this work includes application to variables of potential significance to the social and political sciences (see Eaves and Hatemi 2008; Hatemi et al 2007).

Family Clusters and Individual Differences

Behavior genetic and epidemiological techniques have developed in an attempt to understand why individuals in a population differ from one another. Focusing on individual differences allows us to identify a range of factors that are otherwise hard, even impossible, to resolve in studies of differences between groups that almost inevitably differ biologically and socially. Thus, by *focusing on individual differences rather than group differences* we may begin to explore to what extent differences between individuals express their genetic or environmental individuality and/or the clustering of these effects by social group of which, in many if not most communities, the family is the primary group around which many other effects are clustered. Although *group* characteristics comprise a convenient device for summarizing population data and trends, and even for developing more cost-effective strategies for targeted manipulation and intervention, it is ultimately the *individual* who is the locus of action and decision, so it the individual who comprises the ultimate unit of analysis even when he or she is subject to social influences as part of a family or larger social group.

Family clusters differ from one another genetically and socially. Thus there are *between family* and *within-family* genetic and environmental differences (see e.g. Cattell, 1960 and critique by Loehlin, 1965). Furthermore, as Cattell pointed out, the effects of genes and environment may correlate within and between families. The environments of individuals may be created by or arise in response to their own genetic differences. They might reflect the genetically-influenced behavior of their relatives (e.g.

parents and siblings). Our treatment focuses only on that genotype-environment correlation that arises because parents may exercise an environmental influence on their offspring. In so far as parents are sources of both genetic and social advantage (or disadvantage) Jencks et al. (1972) refer to this type of genotype-environment correlation as a "double advantage" phenomenon.

A problem with these early formulations, such as Cattell's (1965) Multiple Abstract Variance Analysis (MAVA), is that they lacked parsimony. Parameters metastasized at will and there were often more parameters to be estimated than there were data points to solve for them. A classic paper by Jinks and Fulker (1970) pointed out that the underlying simplicity of Mendelian genetics, following Fisher, allowed for many of Cattell's genetic parameters to be redefined in terms that required far fewer parameters that reflected how clusters of genes (polygenes) affected the behavioral phenotype. They further showed that the model-fitting methods already established in statistical genetics at that time (e.g. by Nelder, 1960) made it possible to estimate model parameters reliably and provide a basis for the statistical comparison of alternative models for the same data.

Thus were born the elements of a decomposition of the variance between individuals into a relatively small number of parameters: genetic differences between and within families of differing genetic relationship could be expressed in terms of the additive and non-additive effects of genes. Those due to the environment partitioned into those due to differences *between* family clusters (also referred to as the "shared" or "common" environment) and those due to differences between individuals *within* families (also called the "unique", "non-shared", or "specific" environment). The reader of this literature from the 1970's encounters a frustrating series of different notations and coefficients that largely reflected the academic allegiance of the authors. Now the notation for the variance components has largely been standardized, especially for the study of twins and randomly mating populations (see e.g. Neale and Cardon, 2002). These days, it is typical to refer to the components of variance thus: A=additive genetic component; D=dominance genetic component; C=shared ("common") family environment; E=unique (within-family) environment. In much of the literature, especially that following the school established by Douglas Falconer (Facloner and MacKay, 1995) these parameters are referred to as V_A, V_D, V_{EC} and V_E respectively. These are merely matters of naming convention and imply no substantive difference. In the absence of genotype-environment interaction and correlation the total variance in the population may be expressed as the simple sum of the variance components V_P=A+D+C+E. Further discussion of this model (with notational differences and limitations) may be found in Jinks and Fulker (1970), Neale and Cardon (2002) and standard texts in quantitative genetics (e.g. Mather and Jinks, 1982; Falconer and MacKay , 1995). Potential sources of the shared environment include variables such as socioeconomic status, parental influence, religion, and access to education. The distinction between additive ($A=V_A$) and dominance ($D=V_D$) genetic components of genetic variance was first clarified by Fisher and is a function of the relationship between genotypes and their expression in the phenotypes. Each represents the sum of the individual contributions of separate genetic loci to the phenotype and can be expressed as a function of the frequencies and effects of the different forms ("alleles") of each gene in the homozygous and heterozygous forms (see Appendix for more detail). Neale and Cardon (2002, Ch. 3) provide a simple derivation and definition of the additive and dominance variance components. Our treatment, and that of most investigators, ignore the effects of higher order interactions between genes, known collectively as "epistasis" or "non-allelic interactions". Although variance components may be defined that represent such effects (see e.g. Mather, 1974) their effects are often largely confounded with those of dominance in most practical applications and will not be considered further here.

The basic components of variance model is used widely in univariate analysis, is easy to understand and extends conveniently to the multivariate analysis of twins and sibship data. However, it does not generalize very well to complex kinship structures, especially when there is both social and genetic inheritance and mating is not random. Thus, while we do not lose sight of the goal of partitioning the total variance into its multiple genetic and environmental components and refer to these often, our actual

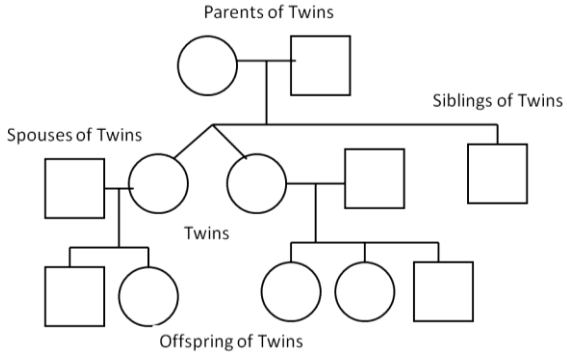
modeling is conducted in terms of path coefficients that may then be translated into components of variance where needed.

Data used in the current study.

The investigation that forms the core of this paper was designed in the early 1980's as the culmination of a decade of theoretical analysis and computer-aided design with the goal of identifying constellations of kinships that had a structure, given large enough samples, to test competing theories of biological and cultural inheritance in the presence of assortative mating and to provide estimates of the critical parameters (path coefficients) of such models. Figure 1 summarizes the pedigrees that form the core of the data set exploited in our illustrative analyses.

We refer to such pedigrees as "extended twin kinships" (ET) because they are constructed around pairs of twins. There are five types of twins featured in the ET kinships: monozygotic (MZ) male and female pairs; dizygotic (DZ) male, female and unlike-sex pairs. Twins form the core of our ability to identify critical genetic effects. However, the ET study is extended to include other constellations of relationships including the parents, spouses, siblings and (adult) children of the original twins (see Eaves et al 1999). If we allow for the five types of twins, and distinguish the various compositions of relative pairs by sex (e.g. mother-daughter, father-son etc.) the ET kinships comprise some 80 unique biological and social relationships (ignoring relationships that span more than two generations since there are relatively few kinships in which we can obtain direct measures on all three generations of adults at the same time, see Keller et al 2009). Pedigrees can be extended further (and sample sizes increased) by using reports provided by relatives about others in the family but the number and detail of measures that can be obtained this way is limited and such data raise additional analytical problems of reporting bias so will not be considered here.

Figure 1. Idealized kinship structure in Virginia 30,000 and Australian 20,000.



Note: Circles represent women. Squares represent men. The figure illustrates the idealized pedigree constructed around female monozygotic twins. Similar pedigrees are constructed around male (MZ) twins, male, female and male-female dizygotic (DZ) pairs.

The richness of the ET kinships can be appreciated by a brief consideration of the implications of some potential comparisons. For example, the spouses of MZ and DZ twins (and of sibling pairs) provide an unparalleled opportunity to resolve subtleties of mate selection including the roles of measured phenotype, interaction between spouses and latent genetic and social differences in mate selection (e.g. Eaves, 1980; Eaves and Heath, 1981; Heath et al., 1985; Heath, 1987; Grant et al.,2007; Keller et al., 2008). The offspring of male and female twins provide a powerful opportunity to explore the environmental effects of genetic differences between parents (e.g. Nance and Corey, 1976; Haley et al., 1983; Heath et al., 1985; D'Onofrio et al. 2003, 2007; Eaves et al., 2005; Silberg and Eaves, 2004; Silberg et al., 2009; Keller et al., 2008). The monozygotic cotwin of a parent is, genetically related to his/her nephew niece as a biological parent, but is socially an uncle/aunt. Such relationships illustrate the

unique opportunity that these kinships offer to resolve genetic and environmental effects that are typically confounded in studies of ordinary kinships. For the purposes of this analysis we confine ourselves to data that comprise the relationships in nuclear families (spouses, parent-offspring and siblings) and the five types of twin pairs.

The families that comprise our illustrative were obtained as part of two parallel studies, conducted in the greater Virginia USA area and from Queensland Australia. The studies are referred to as the Virginia 30,000 (VA30K) and the Australian 20,000 (OZ20K) respectively in this paper. Ascertainment of the samples and zygosity determination for the twins is described elsewhere (see Lake et al 2000) The structure of the sample is summarized for individual twins and their relatives in Table 1. The structure of the sample with respect to the relationships chosen for the present analysis is summarized in Table 2. The Ns in Table 2 reflect the number of pairs that can be reconstructed from the sample to yield estimates of the correlation. The same individual may enter into several correlations, so the correlations are not independent and their sampling errors may be inflated with respect to the values expected if the pairs had been independent. Typically , the lack of independence inflates estimates of the sampling errors of model parameters but does not lead to significant bias (McGue et al., 1984). Numerous comparisons of the Australian samples to the general public have shown the sample to be remarkably similar in attitudes, personality, and somewhat similar in income, educational attainment, religiosity. The Virginia sample has higher levels of income and education than the general public but is quite similar in attitudes, personality and religiosity in comparison to the general public in the region.

Table 1: Sample sizes for Virginia 30,000 and Australia 20,000.

Sample	*Australia*			*Virginia*		
Relationship	Male	Female	Total	Male	Female	Total
Twins	3,459	6,098	9,557	5,325	9,436	14,761
Parents of Twins	1,418	1,956	3,374	913	1,447	2,360
Spouses of Twins	1,547	823	2,370	2,515	1,876	4,391
Children of Twins	925	668	1,593	1,890	2,910	4,800
Siblings of Twins	1,554	2,032	3,586	1,260	1,924	3,184
Total	8,646	11,834	20,480	11,903	17,593	29,496

To illustrate the range of different mechanisms of biological and cultural inheritance, we chose six variables from each sample: self-report stature; scale scores for a general factor of liberalism/conservatism derived from US and Australian modifications of the Wilson-Patterson Conservatism inventory (Wilson and Patterson 1970); arcsin transformed scale scores from 12 neuroticism items selected from the adult form of the longer Eysenck Personality Questionnaire (EPQ, Eysenck 1975) with the aid of Dr. Sybil Eysenck; church attendance; political affiliation; and educational attainment. The ordinal values of church attendance, political affiliation and educational attainment were coded differently in the US and Australian samples to reflect local practice. Thus, in the US, political affiliation was assessed by a five point scale rating strength of preference for Republicans compared to Democrats. In the Australian sample the scale comprised self-reported voting preference for the three principal parties in their parliamentary system, scored as follows: (0) Conservative (Labor + National coalition) (1) Labor. In the VA30K, educational attainment was coded ordinally using the following categories: (0) 0-7 yrs, (1) 8-yrs, (2) 1-3 yrs High School, (3) High School degree or 4 yrs of High School (4) 1-3 yrs College (5) 4+ yrs college. Educational attainment in the OZ20K was coded: (0) < 7 yrs, (2) 8-10 yrs (3) 11-12 yrs, (4) apprenticeship, diploma, certificate etc. (5) technical or teachers college (6) 4 yr degree (7) post graduate.

Family resemblance for each of the variables was summarized by computation of the correlations between relatives for each of the variables using every possible pair that could be derived for each relationship in each sample. Product moment correlations were used to summarize data for stature, conservatism and neuroticism. Polychoric correlations were generated for church attendance, political affiliation and educational attainment. The correlations are given in Table 3. Each was stored for analysis along with the number of pairs on which it was based for each variable. These are not reported individually, but summarized in Table 2.

Table 2: Sample Sizes (Number of Pairs of Relatives) for Participating Families.

	Sample Sizes (N pairs)						
	Virginia			Australia			Total
Relationship	Min	Max	Median	Min	Max	Median	Median
Spouses	4525	4930	**4865**	2569	3474	**3422**	**8287**
Mother-Daughter	3994	4667	4549	2882	4291	4208	8875
Mother-Son	2724	3138	3045	2001	2948	2861	5906
Father-Daughter	2675	3095	3010	2009	3005	2947	5957
Father-Son	1962	2247	2174	1552	2859	2224	4398
Total Parent-Offspring			**12778**			**12240**	**25018**
Male siblings	1368	1551	1523	1105	1586	1540	3063
Female siblings	3203	3645	3588	2139	3288	3228	6816
Male-Female siblings	3858	4395	4331	3131	4562	4487	8818
Total Siblings			**9442**			**9255**	**18697**
Male DZ twins	505	583	575	285	476	380	955
Female DZ twins	1022	1183	1151	624	955	826	1977
Male-Female DZ twins	1147	1334	1310	629	1070	878	2188
Total DZ Twins			**3036**			**2084**	**5120**
Male MZ twins	721	790	774	482	723	632	1406
Female MZ twins	1657	1885	1843	1032	1469	1374	3217
Total MZ Twins			**2617**			**2006**	**4623**

Note: variation occurs in the number of pairs for different variables because of patterns of missing variables. For the purposes of this analysis values are assumed to be missing at random.

Application to Twins and Nuclear Families.

An ideal analysis would focus on the entire set of 80 correlations for each variable including those for more remote relationships (see e.g. Eaves et al., 1999; Keller et al., 2009; Maes et al., 2008). However, it is informative to conduct the analysis in stages that reveal the kinds of model and information that can be obtained from different kinds of data.

With this in mind we conducted three separate analyses using: (1) correlations between members of nuclear families only (spouses, parents and offspring and siblings); (2) MZ and DZ twins; (3) twins and nuclear families jointly. Each analysis illustrates different elements of the modeling process and illuminates the strengths and weaknesses of different constellations of relatives. The analysis of twin data (2) was conducted with and without the correlation between mates in order to exemplify the effect of assortative mating on conclusions from the analysis of twin data. The full data set comprise 13 unique correlations for each variable (c.f. Table 3).

Nuclear Family Resemblance: The Data.

The data on nuclear family resemblance comprise eight unique correlations between relatives: spouses; mother-daughter; mother son; father-daughter; father-son; male siblings; female siblings; unlike-sex siblings (e.g., see Eaves and Hatemi 2008). The data comprise a total of approximately 8,000 spouse

pairs, 25,000 parent-offspring pairs and 18,000 sibling pairs (c.f. Table 2). The raw correlations (Table 3) show a variety of different patterns for different variables even within nuclear families. The pattern of correlation between mates differs markedly across variables in both countries. All the correlations between spouses are positive, confirming a repeated finding that spousal correlations are frequently zero but seldom, if ever, negative. That being said, the correlation between mates for neuroticism in very close to zero in both samples. This pattern is typical for the principal personality dimensions (see Eaves et al., 1990; 1999) and implies that services which seek to match partners for personality may be capitalizing on information that most potential mates regard as relatively unimportant in choosing their partners in the real world. Similarly, the correlation for stature is also small, though significant. Neither personality nor stature play such a marked role in the choice of mate as the more "social" and "political" dimensions and are expected to have less impact on the transmission of differences from one generation to the next. The correlations between mates are much higher for conservatism, church attendance, political affiliation and educational attainment, especially so for church attendance and political affiliation in Australia. All these correlations are among the highest documented for human traits and are expected to have a major impact on individual differences and similarity between relatives. By itself, a spousal correlation may be due to one of several processes individually or jointly including convergence due to interaction between mates ("spousal interaction"); matching on the measured phenotype ("phenotypic assortment"); and matching for correlated features of family and social background ("social homogamy"). We assume initially that assortment is based directly on the measured traits (i.e. phenotypic assortment. This assumption is explored further below.

The correlations between biological relatives in nuclear families – parent-offspring and siblings – reveal a heterogeneous picture of family resemblance. The pattern for stature is very close to that expected from a highly heritable polygenic trait, with modest assortative mating with additive and cumulative genetic effects. These correlations are very similar in both the US and Australia and reminiscent of those published by Pearson and Lee more than a century ago that comprised the foundation for Ronald Fisher's (1918) analysis of family resemblance. In the absence of assortative mating, a completely heritable additive polygenic trait produces correlations of 0.5 between first degree relatives. Assortative mating and vertical cultural transmission tend to inflate parent-offspring correlations relative to those for siblings. This is clearly not the case for stature. Thus the pattern of correlations for nuclear families, including that between spouses, is consistent with a model of substantial genetic influence with little genetic consequence of assortative mating or large additional impact of non-genetic inheritance from parent to child. Genetic "dominance" arises when individuals who carry only one variant copy of a form ("alleleomorph") of a gene resemble those who carry two copies. Genetic dominance was characteristic of the traits analyzed by Mendel in *Pisum sativum.* One of Fisher's critical insights lay in the recognition that not all genes behaving like Mendel's at the level of gamete formation and transmission behaved like Mendel's examples at the level of expression. That is "dominance is a characteristic of the phenotype, not of the genotype." In nuclear family data, large amounts of genetic dominance tend to inflate the correlation between siblings relative to those between parents and offspring. The same is also true of additional environmental factors shared by siblings that do not depend directly on the measured parental phenotype. The US and Australian correlations for stature give little *prima facie* hint of dominance, though we add the qualification that a modest amount of assortative mating may offset the apparent effects of dominance on the correlations between relatives, reinforcing the overall pattern of polygenic additivity. The fact that the correlations between first degree relatives for stature hover around 0.4 rather than 0.5 implies that other random, non-genetic influences create differences between individuals within families. These may include random developmental effects, or specific developmental effects, such as nutritional differences during development and errors of measurement.

Table 3: Correlations between relatives for illustrative variables in United States and Australia

Country	Variable	Spouse	Mo-Da	Mo-So	Fa-Da	Fa-So	Sib M	Sib F	Sib MF	DZM	DZF	DZMF	MZM	MZF
USA	Stature	0.223	0.430	0.446	0.411	0.439	0.432	0.429	0.411	0.483	0.502	0.432	0.850	0.855
Australia	Stature	0.208	0.455	0.424	0.399	0.424	0.391	0.421	0.371	0.415	0.501	0.441	0.872	0.827
USA	Conservatism	0.619	0.456	0.369	0.396	0.410	0.341	0.405	0.328	0.379	0.432	0.310	0.593	0.637
Australia	Conservatism	0.683	0.469	0.409	0.437	0.443	0.423	0.488	0.408	0.513	0.562	0.464	0.612	0.691
USA	Neuroticism	0.092	0.157	0.148	0.127	0.134	0.109	0.172	0.137	0.178	0.224	0.097	0.353	0.410
Australia	Neuroticism	0.059	0.150	0.111	0.139	0.162	0.145	0.137	0.111	0.108	0.197	0.115	0.360	0.429
USA	Church Attendance	0.819	0.566	0.565	0.574	0.585	0.496	0.531	0.456	0.608	0.603	0.488	0.722	0.714
Australia	Church Attendance	0.754	0.434	0.412	0.383	0.379	0.344	0.397	0.372	0.437	0.459	0.352	0.536	0.653
USA	Political Affiliation	0.642	0.434	0.378	0.382	0.400	0.377	0.316	0.329	0.444	0.491	0.404	0.509	0.563
Australia	Political Affiliation	0.831	0.742	0.716	0.686	0.739	0.605	0.532	0.561	0.767	0.643	0.611	0.790	0.753
USA	Educational Attainment	0.568	0.472	0.427	0.449	0.502	0.539	0.573	0.534	0.623	0.673	0.549	0.880	0.854
Australia	Educational Attainment	0.508	0.294	0.234	0.303	0.294	0.385	0.469	0.340	0.490	0.543	0.443	0.706	0.746

Notes: Correlations are based on all possible pairs for each relationship so the same person may contribute several times to one correlation or to several correlations. Product-moment correlations are used for stature, conservatism and neuroticism. Polychoric correlations were computed for church attendance, political affiliation and educational attainment.

We devote more space to the discussion of stature because it is a benchmark of a trait that appears to be influenced substantially by genetic factors and offers a point of departure for the comparison of other traits of greater interest to social and political scientists. Indeed, even the most ardent supporters of environment only models do not refute the importance of heritability for height. At the other end of the spectrum of interest lie the correlations for neuroticism. Along with extraversion (see e.g. Eysenck 1967), and more recently, "openness to experience", neuroticism ("N") is perhaps the most studied of personality traits largely because of the ease of measurement by self report and broad validation against clinical and epidemiological data on depression and anxiety disorders. Our correlations for N in Virginia and Australia, based on very large samples, are typical of those reported by other investigators over a long period in numerous western contexts (see Loehlin and Nichols, 1976; Floderus-Myrhed et al., 1980; Martin and Jardine, 1986, Eaves et al, 1989; 1999; Lake et al., 2000). The correlations between spouses, though statistically significant in these large samples are substantively so small as to be of little account in describing the social process of mate selection on family resemblance. The correlations between first degree biological relatives are all very small in both samples, implying that a large part of the variation between individuals for this aspect of personality is due to random environmental influences unique to individuals, not shared with family members. These effects may include the long-term consequences of individual experiences and the shorter term fluctuations of mood that characterize individual day-to-day changes. The pattern for N is shared by several other related traits, including liability to depression and anxiety disorders and other measured dimensions of personality, including extraversion.

The results for stature and neuroticism serve to highlight the marked differences between these relatively simple traits and those of greater interest to social and political scientists. The parent-offspring and sibling correlations for conservatism, church attendance, political affiliation and educational attainment are all large and resemble those for stature more than neuroticism. For all these outcomes, there is considerable, even extreme resemblance between mates that is far in excess of that seen here for stature, or for most other psychometric traits such as IQ (Vandenberg, 1972; Eaves, 1973; Rao et al., 1975; Mascie-Taylor and Vandenberg, 1988). Whether correlations between spouses reflect mate selection, spousal interaction or a mixture of both it is quite clear that partners who care little about physique and personality invest heavily in partners resembling themselves for religious behavior, social attitudes, educational attainment and political commitment. The same is clearly true for intergenerational transmission. Whether its cause be genetic or social or some function of both, differences between pairs of parents are transmitted to their offspring with considerable fidelity such that the new generation is not *creation ex nihilo* but heavily dependent on influences from the previous generation. No more extreme example of this process may be seen than that for voting preference in Australia for which the correlations between first degree relatives exceed even those for stature! (see Table 3). The excess of the parent-offspring correlation over that for siblings is consistent with the intergenerational transmission of differences between families established by the very high degree of assortative mating for political commitment. In theory, this effect will be apparent whether intergenerational transmission is genetic, cultural or both. Further analysis (see below) allows us to tease apart these theoretically significant alternatives. Whatever the cause, marked resemblance between spouses has more than passing significance for the transmission of political attitudes.

Although the small fluctuations in the correlations as a function of the sexes of the relatives are highly significant statistically with these very large samples, the broad trend of the correlations challenges many popular psychological myths about the effects of parents on their children. The correlations between unlike-sex parents and children are generally little smaller than those for like-sex pairs. This effectively excludes notions of children modeling their behavior more on that of their like-sex parent. Likewise, there is no consistent evidence that mother-offspring correlations exceed father-offspring correlations. Indeed, neither mothers nor fathers appear to play a predominant role in the transmission of these aspects of behavior to the next generation. A variety of biological and social mechanisms are thus excluded as vital features of the transmission of behavior. From a genetic perspective, the data exclude large

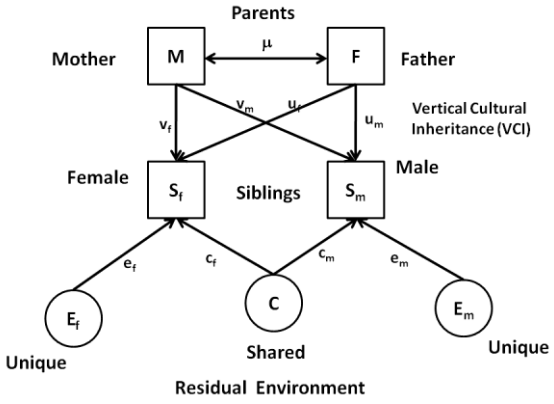
contributions of sex-dependent transmission such as sex-linkage (which predicts much-reduced father-son resemblance, e.g. Mather and Jinks, 1983), environmental effects of the maternal genotype (predicting larger mother-offspring correlations, *ibid*. 1983) or sex-dependent gene expression such as sex-limitation (which predict greater correlations between like-sex pairs than unlike-sex pairs, Eaves, 1977). Although there are some statistically significant differences between sibling correlations as a function of sex, these tend to have no consistent pattern and confirm that, smaller effects notwithstanding, the same basic principles of family resemblance whether genetic or social, apply to both sexes and that there is no overall tendency for men and women raised in the same home to share different, etiologically salient, familial effects. At least as far as shared genetic and environmental influences are concerned, the broad first impression is that sons and daughters experience and respond in like way to common features of their family background in the same way.

Nuclear Family Resemblance: Models for Cultural and Biological Inheritance.
The discussion so far has concentrated on purely visual inspection of the data to describe the principal contours of family resemblance for the chosen variables in these two contexts. We now turn to a more rigorous mathematical treatment by developing and testing elementary models for genetic and non-genetic inheritance. There is, of course, no one model or set of models. Those we describe have some heuristic value and capture many essential features of the processes and theories we seek to evaluate. However, other investigators may and should develop their own models to test more specific alternatives, dependent upon the trait and theoretical foundation they seek to explore. The key to any model is ultimately not merely its ability to characterize a current given data set, but also to generate predictions that may be tested with other kinds of data and in other contexts.

We present two initial path models for nuclear family resemblance. The first (Figure 2) is a model for non-genetic transmission. The second (Figure 3) is a simple model for additive genetic transmission. The models are illustrative and neither exhaustive or mutually exclusive. Both models are well-established in the genetic literature on human family resemblance (see e.g. Cloninger et al., 1979; Neale and Cardon, 2002). The conventions in the figures are similar to those originally developed by Sewall Wright (1921) and expounded for social scientists by Duncan (1966). Single-headed arrows represent hypothetical directions of causation. The variables u_m, v_f etc. are the path coefficients, standardized regressions of the consequences on their assumed causes. Double-headed arrows (such as that labeled μ in the path diagram) reflect correlations for which now direction of cause can be attributed. Most models for family resemblance involve manifest variables such as the measured trait values of parents (M and F) and siblings (S_f and S_m) and latent variables that are not measured explicitly (such as residual shared, C, and specific environmental influences on males and females, E_m and E_f). The task of modeling consists primarily in: (1) estimating the paths and correlations between manifest and latent variables for a given model and set of correlations and; (2) assessing the goodness of fit, i.e. addressing the ability of any model to account for the data.

The non-genetic model assumes assortative mating is based on the measured phenotypes of mothers and fathers (M and F) and that the marital correlation is μ. Parent-offspring transmission occurs by the direct effect of the measured phenotypes of mothers and fathers on the phenotypes of their children. The paths u_m and u_f reflect the environmental impact of fathers on their male and female children, respectively. Similarly paths v_m and v_f reflect the environmental impact of mothers on their sons and daughters. The model also incorporates two non-genetic residual sources of variation. We postulate that siblings also share environmental influences, C, that do not depend directly on the manifest variables of their parents. These may have a different influence on male (c_m) and female (c_f) offspring.

Figure 2: Elementary model for non-genetic inheritance in nuclear families.



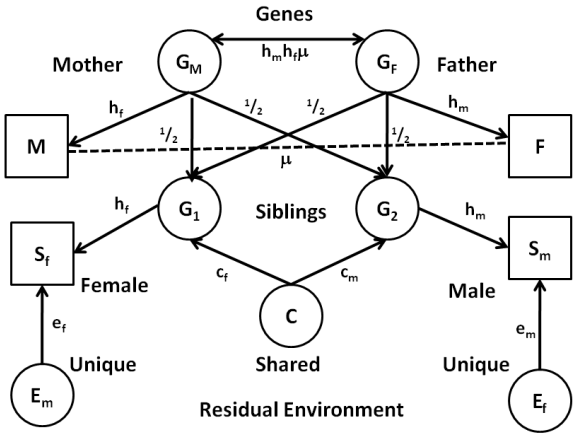
In addition, we anticipate that residual non-genetic influences are specific to individual offspring that are not shared with their siblings so are uncorrelated between siblings. These are denoted in the diagram by the latent variables E_m and E_f for men and women respectively. The paths from the unique, specific environments of siblings are e_m and e_f. Since we are working with correlations and there is no theory of differences in variance in this application, we assume that all latent and manifest variables are standardized to unit variance.

The basic genetic model (Figure 3) has structural similarities with the model for vertical cultural inheritance (Figure 2). Both models allow for residual non-genetic effects of the shared and non-shared environment (C and E). Both also allow for phenotypic assortative mating, represented by μ in the diagram. However, the mechanism of transmission is assumed to be different. In the genetic model, intergenerational transmission depends on latent genetic variables, G, rather than on the manifest phenotypes. Fisher's exposition of polygenic Mendelian inheritance shows that the intergenerational paths between the latent genetic components are all the same and have an explicit prior value (½) that follows from Mendel's first law of segregation regardless of the sex of the parent or recipient. The same may not be said, however, for the effects of the genes on the phenotype. These may depend on sex. Thus the model allows for different paths, h_m and h_f, from latent genetic effects to manifest outcomes in males and females respectively.

Although the "genetic" model assumes that assortment is based primarily on the phenotype of the spouses, the fact that the phenotypes are only an unreliable indicator of the latent genotype means that the correlation between the genetic effects of spouses will be attenuated by non-genetic effects. Following Fisher (1918), we note that primary phenotypic assortment induces a correlation between spouses' genetic effects that depends on the marital correlation, μ, and the paths, h_m and h_f, from additive genetic effect to phenotype.

Fisher considers other possible mechanisms of assortative mating, including assortment for a (genetically) correlated variable and assortment based primarily on genotype rather than phenotype. These mechanisms are considered below. Both the genetic and non-genetic models assume that the effects of genes and environment are not modulated by age. This assumption appears to be approximately true for adults in so far as it has been tested. Hatemi et al. (2009) consider application of models for age-dependent expression of genetic and environmental factors in the development of conservative-liberal attitudes. There analysis is summarized below. The current adult sample comprises individuals who are all older than 18 years.

Figure 3: Elementary model for genetic inheritance in nuclear families.



Note: Model assumes no cultural transmission from parent to offspring. Assortative mating is assumed to be based primarily on parental phenotype.

The rules of linear algebra, as explained in a range of publications in the life and behavioral sciences (e.g. Wright, 1921; Duncan, 1966; Morton, 1973; Cloninger et al., 1979; Heath et al., 1985; Truett et al., 1994; Li, 1975; Loehlin, 2003; Neale et al., 2002; Eaves et al., 2005) allow derivation of algebraic expectations of the correlations in terms of the model parameters. The expectations for the correlations between nuclear families are summarized for the two basic models in Table 4.

Table 4: Expected correlations between members of nuclear families under models for phenotypic (Figure 2) and genetic (Figure 2) inheritance.

	Transmission model	
Correlation	Phenotypic (Cultural)	Genetic
Spouses	μ	μ
Mother-daughter	$v_f(1+u_f)$	$\frac{1}{2}\, h_f(h_f+ h_m\mu)$
Mother-son	$v_m(1+u_m)$	$\frac{1}{2}\, h_m h_f(1+\mu)$
Father-daughter	$u_f(1+v_f)$	$\frac{1}{2}\, h_m h_f(1+\mu)$
Father-son	$u_f(1+v_m)$	$\frac{1}{2} h_m(h_m+ h_f\mu)$
Male sibling	$u_m^2+ v_m^2 + 2\mu\, u_m\, v_m + c_m^2$	$\gamma\, h_m^2 + c_m^2$
Female sibling	$u_f^2+ v_f^2 + 2\mu\, u_f\, v_f + c_f^2$	$\gamma\, h_m\, h_f + c_m\, c_f$
Male-female sibling	$u_m\, u_f + v_m\, v_f + \mu\, (u_m\, v_f + u_f\, v_m\,) + c_m\, c_f$	$\gamma\, h_f^2 + c_f^2$
Total variance (male)	$u_m^2+ v_m^2 + 2\mu\, u_m\, v_m + c_m^2 + e_m^2 = 1$	$h_m^2 + c_m^2+ e_m^2= 1$
Total variance (female)	$u_f^2+ v_f^2 + 2\mu\, u_f\, v_f + c_f^2 + e_f^2= 1$	$h_f^2 + c_f^2+ e_f^2= 1$

Note: γ is the genetic correlation between siblings. $\gamma = \frac{1}{2}\,(1+h_m h_f \mu) = \frac{1}{2}$ when mating is random (μ=0).

Fitting the Model: Computational Method

The nuclear family data comprise eight unique correlations between relatives. The "non-genetic" model involves seven free parameters: the correlation between mates, μ; four parameters for the environmental effects of parents on children, u_f, v_f, u_m, v_f; and the two paths from the residual shared environment, C, to the phenotypes of male and female offspring, c_m and c_f. The remaining paths, e_m and e_f, are fixed when the others are known by the constraint that the total variance is standardized to unity in males and female. Similarly, the full genetic model has five free parameters: m, h_f, h_f, c_m and c_f. In principle, estimates of the parameters of both models might now be derived by solving the eight simultaneous equations generated by equating the eight observed nuclear family correlations for each of the variables in Table 3 to their

algebraic expectations under each model in Table 4. However, this may not be as easy as it seems, especially as the models get more complex. First, there are more equations than unknown parameters, so there is no unique solution for each parameter. Second, the equations are non-linear, sometimes extremely so making the algebra very tedious. Third, some models (see below) require the imposition of (non-linear) constraints on the parameters. In addition, it is preferable that any approach to estimation makes optimal use of the data and allows some statistical assessment of the adequacy of any model and relative predictive value of alternative explanations of the empirical data.

Ideally, we would employ the method of maximum likelihood (ML) which has been used extensively in the analysis of kinship data (see e.g. Lange et al., 1976; Neale et al., 2004) including the extended kinships of twins (e.g. Maes et al., 1997; 2009, Silberg et al, 2009). However, this approach requires that the expectations for all the kinship relationships be specified, which are far more numerous than the eight chosen for this application. Although ML makes optimal use of the raw data, in the sense that it yields the most precise estimates attainable with a given data set, and allows likelihood ratio chi-square tests of different sub-hypotheses, it is less transparent for the purposes of model description because it is applied to the raw data from the extended pedigrees and is harder to use for the partly didactic purposes of this chapter.

For the purpose of these analyses we employed a close relative of ML namely non-linear weighted least squares (WLS, see Nelder 1960; Rao et al. 1974).[15] Both ML and WLS can be implemented in well-tried and readily available software for structural modeling. We used Dr. Michael Neale's Mx package (Neale et al. 2002) that has been widely used for fitting models of biological and cultural inheritance to complex pedigrees. Translation of models into code is relatively easy, and especially for WLS, processing is rapid allowing the comparison of series of models for numerous variables to be accomplished relatively quickly. The package uses Gill and Murray's program NPSOL for efficient non-linear optimization subject to boundary, linear and non-linear constraints (Gill et al., 1998).

The numerical analysis requires minimization of the loss-function:

$$S^2 = \Sigma\delta^2 = \sum_{i=1}^{i=k} N_i[r_i - E(r_i)]^2 \quad .....(1)$$

with respect to differences in the p model parameters, θ. Summation is applied over all $i=1...k$ observed correlations, r_i, each based on N_i pairs. The $E(r_i)$ are the corresponding expected correlations obtained by substituting current values for the model parameters, μ, u_m, u_f etc in the algebraic expectations of Table 4. The weights, N_i, ensure that observations known more precisely (because they are based on larger sample sizes) have proportionally more influence on the final solution. The minimum weighted sum of squared residuals, $\Sigma\delta^2$ is expected to be smaller for models that fit better than those that fit to zero. $\Sigma\delta^2$ has k-p d.f. when a model with p free parameters is fitted to k observed correlations.

Model-Fitting Results: Non-genetic Models

The full model for non-genetic transmission (Figure 3) was fitted initially to each of the variables by WLS using code written in *Mx (see Appendix 2*). Subsequently, reduced models were fitted by setting specific sets of parameters to zero, or imposing constraints on parameter values. The model-fitting results are summarized for the non-genetic model in Table 5. For most of the variables, the full non-genetic model fits quite well. As a "rule-of-thumb" we may, as an approximation, compare the weighted residual sum-of-squares to the chi-square distribution for the same d.f. By this criterion, the largest $\Sigma\delta^2_{(1)}$ for the full model is a highly significant value of 13.92 for educational attainment in the Australian

[15] In comparing WLS to ML in extended kinship design for political attitudes, Hatemi et al 2009 (ML) found no differences with Eaves and Hatemi (2008) (WLS).

sample. The only other variable to which the model gives a poor fit is church attendance in the US ($\Sigma\delta^2_{(1)}$ = 5.62). The table also gives the changes, $\Delta\delta^2_{(d)}$, in $\Sigma\delta^2$ for a series of reduced models. The d.f., *d*, of $\Delta\delta^2$ is equal to the number of additional constraints imposed in the reduced model. Thus, for a model that assumes random mating, one parameter, μ, is fixed at zero so the corresponding $\Delta\delta^2$ has 1 d.f. A model that equates all parameters across sexes, i.e. $u_m = u_f = v_m = v_f$ and $c_m = c_f$, requires 4 fewer parameters than the full model so $\Delta\delta^2$ has 4 d.f. (c.f. Table 5, "No Sex" model).

In general, the full model is quite resistant to reduction for most of the variables. In no case, is it possible to ignore the effects of assortative mating (i.e. set m=0). This is true even for neuroticism, for which the marital correlation is very small and reflects the considerable precision resulting from the relatively large samples of spouse pairs in the sample. Similarly, it is not possible to discount parent-offspring transmission for any of the variable because the $\Delta\delta^2$ for testing the effects of vertical cultural inheritance ("No VCI" in Table 5) are all very large and highly significant. Nor is parent-offspring transmission, though highly significant for all variables, sufficient to explain the correlations between siblings. Thus, estimates of the residual shared environment of siblings are significant ("No C" in Table 5) for all variables except for political affiliation in Australian families. With this exception, the data show that the causes of family resemblance in complex behaviors cannot be explained simply by reference to the environmental influence of the parental phenotypes but also reflect other environmental factors that cannot be attributed directly to the corresponding variables in parents. Such effects may include other sources of environmental similarity, including other aspects of parental behavior and the social contexts shared by siblings independently of their parents.

The statistics in Table 5 suggest that the only acceptable simplifications to the most general model involve removing sex differences in the model parameters ("No Sex", $\Delta\delta^2_{(4)}$ in Table 5). In neither Virginia nor in Australia is there compelling evidence that the causes of family resemblance depend on sex for stature, neuroticism or political affiliation. The effects of mothers and fathers on their male and female offspring are all similar in magnitude. There is no support for the view that mothers are more influential than fathers, or vice versa, or that sons or daughters are more or less susceptible to parental influence on these variables. This being said, the data provide compelling evidence for sex effects on patterns of family resemblance in conservatism in both samples and, in Australia but not Virginia, for church attendance and educational attainment. Table 6 summarizes the relative contributions of the various sources of hypothesized environmental influences in nuclear families for the variables chosen for study.

Table 5: Parameters of phenotypic transmission in nuclear families.

		Parameter (c.f. Figure 2)										Model Comparison Statistics			
		Mating	Father-Offspring transmission		Mother-Offspring transmission		Residual Shared		Residual Unique		Full	No Sex	No C	No VT	No μ
Trait	Country	μ	u_m	u_f	v_m	v_f	c_m	c_f	e_m	e_f	$\Sigma\delta^2_{(1)}$	$\Delta\delta^2_{(4)}$	$\Delta\delta^2_{(2)}$	$\Delta\delta^2_{(4)}$	$\Delta\delta^2_{(1)}$
Stature	US	0.223	0.359	0.331	0.367	0.356	0.310	0.369	0.582	0.575	0.65	2.20	47.48	2349.62	235.97
	Au	0.208	0.354	0.316	0.352	0.366	0.252	0.335	0.635	0.586	2.12	8.11	27.85	2361.33	146.30
Conservatism	US	0.619	0.307	0.177	0.176	0.348	0.350	0.410	0.686	0.603	2.68	25.37	109.26	2262.05	1882.05
	Au	0.683	0.323	0.209	0.185	0.328	0.414	0.482	0.608	0.523	3.68	15.37	184.39	2449.18	1617.75
Neuroticism	US	0.092	0.121	0.114	0.137	0.147	0.271	0.367	0.890	0.828	0.00	6.74	91.10	267.16	40.78
	Au	0.059	0.157	0.130	0.101	0.143	0.297	0.296	0.875	0.873	1.84	4.16	56.55	250.31	12.10
Church	US	0.819	0.404	0.320	0.236	0.304	0.278	0.411	0.548	0.478	5.62	6.25	62.07	4174.16	3361.87
attendance	Au	0.754	0.158	0.129	0.293	0.336	0.407	0.450	0.654	0.602	0.02	11.85	138.76	2032.32	1952.30
Political	US	0.642	0.269	0.175	0.204	0.323	0.434	0.325	0.627	0.687	0.11	9.51	87.63	1846.18	1865.69
affiliation	Au	0.834	0.464	0.233	0.331	0.535	0.157	0.015	0.395	0.451	1.20	9.16	0.36	4404.42	2012.25
Educational	US	0.568	0.387	0.264	0.206	0.322	0.495	0.546	0.472	0.431	0.48	7.93	275.65	2730.66	1590.63
attainment	Au	0.508	0.250	0.199	0.103	0.195	0.471	0.575	0.679	0.552	13.92	26.22	389.24	966.39	878.04

Note: Significance levels of χ^2

	P (%)				
d.f.	10	5	1	0.5	0.1
1	2.70	3.84	6.63	7.88	10.83
2	4.61	5.99	9.21	10.60	13.82
3	6.25	7.81	11.34	12.84	16.27
4	7.78	9.49	13.28	14.86	18.47

Table 6: Relative contributions (% total variation explained) of phenotypic familial influences outcome measures.

	Sex	Males			Females		
Trait	Source	Parents	Shared	Unique	Parents	Shared	Unique
Stature	US	32.2	9.6	58.2	28.8	13.6	57.5
	Au	30.1	5.4	63.5	30.2	11.2	58.6
Conservatism	US	19.2	12.2	68.6	22.9	16.8	60.3
	Au	22.0	17.2	60.8	24.5	23.2	52.3
Neuroticism	US	3.6	7.4	89.0	3.7	13.5	82.8
	Au	3.7	8.8	87.5	4.0	8.8	87.3
Church	US	37.4	7.7	54.8	35.3	16.9	47.8
attendance	Au	18.1	16.6	56.4	19.6	20.2	60.2
Political	US	18.5	18.8	62.7	20.7	10.6	68.7
affiliation	Au	58.1	2.5	39.5	54.9	0.0	45.1
Educational	US	28.3	24.5	47.2	27.1	29.9	43.1
attainment	Au	9.9	22.2	67.9	11.7	33.1	55.2

The table shows that by far the largest contributors to variation in outcome are influences that cannot be predicted by family membership, i.e. the influence of parental phenotypes or other, residual contextual effects shared by siblings. In the most extreme case, neuroticism, 80-90% of the total variation cannot be explained by reference to parents or the shared environmental influences on siblings. For the other variables, unique non-familial influences account for less of the variance, but seldom less than 45% of the total. The data reveal two striking differences in transmission between Virginia and Australia. In Virginia, parental influences account for about twice as much of the variation in church attendance compared with Australia. By contrast, in Australia, the impact of parents on the political preference of their offspring explains more than 50% of individual variation, almost twice as much as in the Virginia sample. It appears that parents care more about how their children vote in Australia and they care more about whether or not they go to church in Virginia. The fact that the contributions of parents to the religious and political behavior of their sons and daughters is so labile across cultures perhaps argues for a greater impact of the cultural environment on these variables than for others, such as stature, for which the relative contributions of the sources of variation are more uniform between the two contexts.

Model-Fitting Results: Genetic Models.

Table 7 presents parameter estimates and model comparison statistics for the model that assumes inter-generational transmission is purely genetic. Results are not presented for tests of random mating because they depend almost exclusively on the correlation between mates and differ very little from the corresponding values for the non-genetic model in Table 5. Most of the features in Table 7 resemble closely the essentials of their counterparts in Table 5. Thus, just as it is impossible to delete parent-offspring transmission for the non-genetic model, so it is impossible to remove genetic effects from the genetic model. With few exceptions, the genetic model fits no better or worse ($\Sigma\delta^2_{(3)}$) than its cultural counterpart ("No VT" in Table 7 $\Sigma\delta^2_{(1)}$), although the genetic model requires two fewer parameters.

Thus, in general, there is little to choose empirically between genetic and non-genetic explanations of nuclear family data: a fact appreciated more than a century ago by Francis Galton (1883) and which led to his recognition of the possible significance of twin studies for the resolution of cultural and biological inheritance. By contrast, Karl Pearson (1903) had no compunction in generalizing his conclusions about the inheritance of physical human and animal traits to "the mental and moral characteristics of man" (sic) using data on the ratings of sibling pairs by their teachers.

There are some exceptions to the conclusion that nuclear family data are inherently weak for resolving genetic and social models for transmission. There is little doubt that a purely genetic model cannot

explain the transmission of political affiliation in Australia. The residual sum of squares for the cultural model is only 1.20 compared with a much larger value of 30.08 for the genetic model. A non-genetic model is also marginally better supported than its genetic counterpart for conservatism in Virginia and educational attainment in Australia but the difference is not great. Table 8 presents the estimated contribution of genetic and environmental factors to differences in the outcomes based on the nuclear family data. These values should be compared with the estimated contributions under the purely "non-genetic" model in Table 6.

A noteworthy feature of the genetic model is an inherent reduction in estimates of the relative contribution of the unique environment (E). This result is an artifact of the fact that model for genetic effects recognizes that genetic influences that contribute to parent-offspring transmission and to differences *between* family clusters also contribute to variation *within* sibships as a result of the separation of genetic variants into different gametes at meiosis (see glossary). These effects are implicitly estimated from parent-offspring transmission and a component subtracted from the variance within sibships, assigning any further residual effects to the unique environment (and/or the effects of genetic dominance).

Table 7: Parameters of genetic transmission in nuclear families.

		Parameter estimates (c.f. Figure 3)							Model comparison statistics				
					Environment								
Country	Trait	Mating	Genes		Shared		Unique		Full	No Sex	No C	No Genes	VT[1]
		μ	h_m	h_f	c_m	c_f	e_m	e_f	$\Sigma\delta^2_{(3)}$	$\Delta\delta^2_{(2)}$	$\Delta\delta^2_{(2)}$	$\Delta\delta^2_{(2)}$	$\Sigma\delta^2_{(1)}$
Stature	US	0.223	0.854	0.831	0.001	0.172	0.520	0.530	1.91	0.94	1.77	2348.36	0.65
	Au	0.211	0.808	0.854	0.131	0.000	0.580	0.528	7.55	3.83	0.33	2355.90	2.12
Conservatism	US	0.617	0.657	0.768	0.241	0.000	0.714	0.640	10.12	17.93	3.89	2254.60	2.68
	Au	0.683	0.703	0.744	0.221	0.322	0.676	0.587	7.46	11.59	32.68	2445.40	3.68
Neuroticism	US	0.092	0.479	0.535	0.029	0.159	0.877	0.829	1.16	5.57	1.38	266.00	0.00
	Au	0.061	0.495	0.512	0.143	0.000	0.857	0.859	5.95	0.47	0.47	246.25	1.84
Church	US	0.820	0.797	0.783	0.000	0.260	0.603	0.565	2.33	9.54	9.04	4177.45	5.62
attendance	Au	0.754	0.662	0.697	0.226	0.265	0.715	0.666	5.25	4.40	20.23	2027.09	0.02
Political	US	0.643	0.681	0.710	0.272	0.033	0.680	0.703	5.66	4.70	5.14	1840.63	0.11
affiliation	Au	0.850	0.878	0.844	0.000	0.000	0.479	0.535	30.08	3.10	0.00	4375.54	1.20
Educational	US	0.568	0.768	0.766	0.359	0.421	0.529	0.487	9.08	2.33	122.00	2722.02	0.48
attainment	Au	0.508	0.597	0.625	0.322	0.470	0.735	0.624	20.47	19.67	156.73	959.84	13.92

Notes: [1] Goodness-of-fit test for phenotypic transmission model (see Table 6)

Table 8: Relative contributions (% total variation explained) of genetic transmission on outcome measures (nuclear family data).

	Sex	*Males*			*Females*		
Trait	Source	Genes	Shared E	Unique E	Genes	Shared E	Unique E
Stature	US	73.0	0.0	27.0	69.0	2.9	28.1
	Au	65.2	1.7	33.0	73.0	0.0	27.0
Conservatism	US	43.1	5.8	51.0	59.0	0.0	41.0
	Au	49.4	4.9	45.7	55.3	10.4	34.3
Neuroticism	US	22.9	0.1	77.0	28.7	2.5	68.8
	Au	24.5	2.1	73.4	26.2	0.0	73.8
Church	US	63.6	0.0	36.4	61.3	6.8	31.9
attendance	Au	43.8	5.1	51.1	48.6	7.0	44.4
Political	US	46.3	7.4	46.3	50.4	0.1	49.5
affiliation	Au	77.1	0.0	22.9	71.3	0.0	28.7
Educational	US	59.1	12.9	28.0	58.6	17.7	23.7
attainment	Au	35.6	10.4	54.0	39.0	22.1	38.9

The general conclusion of model-fitting to correlations for nuclear families is that parents affect the behavior of their children but, with few exceptions, nuclear family data provide little hard evidence for or against either theory of human differences.

Models for Twin Resemblance.

As Galton pointed out (1883), twins provide a natural experiment that may facilitate the resolution of biological and environmental influences on human variation. He noted that there were two kinds of twins. Identical twins, Galton theorized, arose by the division of a single fertilized egg into two genetically identical individuals and, hence were termed "monozygotic" (MZ). By contrast non-identical twins were assumed to arise because two separate ova, released at the same time, were fertilized by separate sperm from the same father. Such twins were thus "fraternal" because they were merely siblings who happened to have undergone gestation and birth at the same time or "dizygotic" because they represent zygoytes arising from completely independent events of fertilization. It is intuitively apparent that, *ceteris paribus*, variation within MZ pairs reflects only environmental effects, whereas intra-pair differences for DZ twins reflect both environmental dissimilarity and the effects of genetic segregation[16].

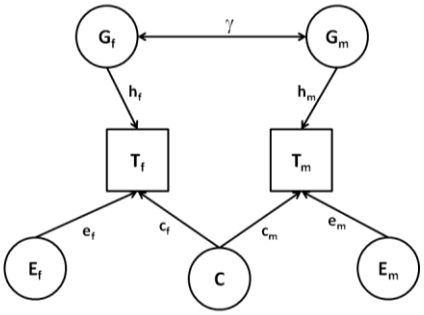
Figure 4 presents the elements of a simple model for the resemblance of twins. As for our model for nuclear family data, we recognize that the effects of genes and environment may vary across sexes. Thus, the figure represents the model for unlike-sex DZ twins. There are virtually no unlike-sex MZ pairs. The diagrams for like-sex pairs are the same, with the appropriate substitution of the genetic and environmental paths for males and females, *mutatis mutandis.* The diagram embodies a series of assumptions, some of which have been considered in the context of nuclear families above. Gene action is assumed to be additive initially and, to a first approximation we assume that the same genes and shared environmental effects contribute to individual differences within the sexes. However, we do not assume that the genetic and environmental paths are necessarily the same in males and females. The parameter γ

[16] An unspoken assumption of the twin method implies that MZ and DZ twins are equally correlated for equally variable environmental influences. The validity of this "equal environments assumption (EEA)" has been widely questioned and tested with a range of results. Frequently, the assumption is violated by measured aspects of the environment that turn out to show little or no correlation with the measured phenotype (e.g. Loehlin and Nichols, 1976). Even in the rare instance of when there is a *prima facie* case for violation of the EEA, the direction of causation is ambiguous. This issue is discussed further below.

is the genetic correlation between twins. Since MZ twins are genetically identical, $\gamma = 1$ for MZ twins. Under random mating, $\gamma = ½$ for DZ twins. For traits in which assortative mating increases the genetic resemblance between mates (e.g. phenotypic assortment) $\gamma > ½$. Typically, twin studies do not incorporate data on parents so mating is usually assumed to be random and the genetic correlation between DZ twins fixed at ½ *ex hypothesi*. If there is significant assortative mating and its effects on the genetic similarity of DZ twins (or siblings) are ignored, the genetic consequences of assortative mating inflate estimates of shared environmental effects derived from twin data. This tendency will be illustrated below by analyzing twin data with and without including estimates of the effect of assortative mating.

A further major assumption of the twin model is that the effects of genes and the shared environment are independent. In the event that genetic differences between parents have a direct or indirect effect on the environment of their offspring this will no longer be the case but the effects of genes and shared environment will be correlated. This possibility will be explored explicitly when we attempt to integrate the analysis of twins and nuclear families in a unified model. If the analysis of twins alone, however, such "passive genotype-environment covariance" will be confounded with estimates of the shared environment.

Figure 4: Model for resemblance between twins



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Note: The correlation between genotypes of twins (γ) is unity for MZ twins and ½ in DZ twins when mating is random and gene action is additive. In the presence of positive assortative mating, γ is increased (see text).

An implication of the "twin" model is that any genetic effects of assortative mating, and any environmental effects of the parental genotype contribute to estimates of the shared environment. Thus, in the analysis of twin data, estimates of the shared environment subsume a wide range of ways in which parental phenotypes influence those of their children. In the model for nuclear family data, estimate of the residual shared environment in siblings reflect effects that cannot be predicted directly from the parental phenotypes. Wright's rules for deriving expected correlations from path diagrams generate the expected correlations between twins shown in Table 9.

Table 9: Expected correlations between twins (c.f. Figure 4).

Correlation	*Expectation*
Spouses	μ
MZ male	$h_m^2 + c_m^2$
MZ female	$h_f^2 + c_f^2$
DZ Male	$\gamma\, h_m^2 + c_m^2$
DZ Male-Female	$\gamma\, h_m h_f + c_m c_f$
DZ Female	$\gamma\, h_f^2 + c_f^2$

Note: If spouses are not available it is assumed that $\gamma = ½$ (i.e. that mating is random). If $\mu > 0$ but spouse data are not available, the genetic consequences of assortative mating will inflate estimates of the shared environment (c).

If mating is random and genetic effects are additive, so that the genetic correlation between siblings is ½, the correlation between MZ twins is expected to be exactly twice that for DZ twins. The effects of the shared environment and positive assortative mating tend to inflate the DZ correlation relative to that for MZ's, so that $2r_{DZ} - r_{MZ}>0$ (see e.g. Eaves, 1982). Non-additive genetic effects, including genetic dominance tend to reduce the DZ correlation relative to MZ and result in $2r_{DZ} - r_{MZ}<0$. Generally, the power for the test of genetic dominance in twin studies is low (see e.g. Martin et al., 1978) and very large samples are required to test for it.

Preliminary inspection of the correlations for MZ twins and like-sex DZ twins in Table 3 suggest that the pattern for stature is close to what is expected under additive gene action in the absence of dominance and the shared environment. The DZ correlations for neuroticism are somewhat less than half those for MZs suggesting that some genetic effects may be non-additive. Most of the other variables in the study have DZ twin correlations that exceed half their corresponding MZ correlations suggesting that the effects either of the shared environment or assortative mating or both are contributing to twin resemblance. The model given in the table was initially fitted to the five twin correlations for each variable in each sample in Table 3 excluding the correlation between spouses and thus, ignoring the effects of assortative mating. Thus, the estimates of the shared environment will be inflated by any genetic consequences of assortative mating.

Table 10: Parameters of Model for Twin Resemblance (Excluding Spouse Pairs).

		Parameter Estimates (c.f. Table 4)						*Model Comparison Statistics*			
		Genes		Environment: Shared		Environment: Unique		Full	No Sex	No C	No Genes
Trait	*Country*	h_m	h_f	c_m	c_f	e_m	e_f	$\Sigma\delta^2_{(1)}$	$\Delta\delta^2_{(2)}$	$\Delta\delta^2_{(2)}$	$\Delta\delta^2_{(2)}$
Stature	US	0.917	0.836	0.144	0.393	0.371	0.383	1.20	1.98	6.03	204.20
	Au	0.917	0.811	0.154	0.414	0.367	0.414	0.38	2.90	5.26	146.22
Conservatism	US	0.765	0.630	0.163	0.486	0.624	0.605	2.52	7.71	13.69	88.42
	Au	0.570	0.523	0.541	0.644	0.618	0.559	2.39	4.45	55.35	22.27
Neuroticism	US	0.395	0.642	0.396	0.000	0.829	0.767	4.56	10.18	5.95	69.76
	Au	0.515	0.647	0.224	0.000	0.828	0.762	6.18	5.14	0.37	60.38
Church	US	0.681	0.444	0.531	0.711	0.504	0.555	8.05	2.31	93.79	38.35
attendance	Au	0.000	0.633	0.705	0.504	0.709	0.588	2.35	8.70	20.67	41.64
Political	US	0.498	0.379	0.520	0.644	0.694	0.664	2.09	3.47	64.23	11.59
affiliation	Au	0.000	0.509	0.882	0.706	0.471	0.493	0.35	4.98	91.86	13.28
Educational	US	0.823	0.589	0.468	0.708	0.321	0.390	3.54	6.49	81.80	92.46
attainment	Au	0.751	0.634	0.391	0.583	0.532	0.508	2.12	3.70	32.57	66.80

Weighted least squares estimates of model parameters and model comparison statistics are given in Table 10 using only the five twin correlations for each variable in each sample. The model thus assumes random mating. The fit of the four parameter model to the five correlations is good in most cases. The main exceptions are neuroticism where the explanation probably lies with the relatively low correlations for unlike-sex DZ twins compared with those for like-sex pairs. One possible explanation of this might be that different genetic or shared environmental effects contribute to variation in males and females. For example, genes with sex-limited effects (i.e. that influence one sex but not the other) might lead to a relatively lower correlation between relatives of opposite sexes. This being said, the fit of the model does not generally get worse when the effects of genes and environment are constrained to be the same in males and females ("No sex" in Table 10). In the US sample there is some evidence supporting heterogeneity across the sexes for conservatism, neuroticism and educational attainment. In Australia, the sexes appear to be significantly different with respect to the effects of genes and environment on church attendance. The issue of sex-dependent genetic effects will be considered further below.

With the exceptions of stature and neuroticism, the twin data show that the effects of the shared environment are significant for all the variables chosen for analysis. Thus, C cannot be deleted from the model for conservatism, church attendance, political affiliation or educational attainment in either population. Removing C from the model ("No C" in Table 10) worsens the fit significantly and, in many cases, substantially. Since the model assumes random mating, it is possible that some or all of what passes for C in the model may actually be the excess genetic correlation between relatives generated by the correlation between mates. The impact of assortment on these measures will be evaluated below.

Table 11: Relative contributions (% total variation explained) of genetic transmission on outcome measures (twin data, without spouse pairs).

	Sex	*Males*			*Females*		
Trait	Source	Genes	Shared E	Unique E	Genes	Shared E	Unique E
Stature	US	84.1	2.1	13.8	69.9	15.4	14.7
	Au	84.1	2.4	13.5	65.7	17.1	17.2
Conservatism	US	58.5	2.7	38.9	39.7	23.7	36.6
	Au	32.5	29.3	38.2	27.4	41.4	31.2
Neuroticism	US	15.6	15.6	68.8	41.2	0.0	58.8
	Au	26.5	5.0	68.5	41.9	0.0	58.1
Church	US	46.4	28.2	25.4	19.7	50.5	29.7
attendance	Au	0.0	49.8	50.2	40.1	25.4	34.6
Political	US	24.8	27.0	48.1	14.4	41.5	44.1
affiliation	Au	0.0	77.8	22.2	25.9	49.9	24.3
Educational	US	65.8	21.9	10.3	34.7	50.1	15.2
attainment	Au	56.4	15.3	28.3	40.1	34.0	25.8

The fit of the model to the twin data is significantly poorer when genetic effects are dropped from the model for all the variables studied in both populations ("No genes" in Table 10). The loss of fit is dramatic in the case of stature and significant, though far less so, even for political affiliation providing some evidence, albeit not as strong as for the other outcomes, for the role of genes in political preference. Table 11 summarizes the estimated contributions of additive genetic effects, shared environment, and individual unique experience to individual differences in the chosen measures. There is no evidence of genetic effects on church attendance and political preference in Australian males. Stature is indeed the most heritable trait in this set.

Table 12: Parameters of Model for Twin Resemblance (Including Spouse Pairs).

		Parameter estimates (c.f. Figure 5)								Model comparison statistics				
					Environment									
Trait	Country	Mating	Genes		Shared		Unique		Full	No Sex	No C	No Genes	Sex-specific C	
		μ	h_m	h_f	c_m	c_f	e_m	e_f	$\Sigma\delta^2_{(1)}$	$\Delta\delta^2_{(2)}$	$\Delta\delta^2_{(2)}$	$\Delta\delta^2_{(2)}$	$\Sigma\delta^2_{(2)}$	$\Delta\delta^2_{(1)}$
Stature	US	0.220	0.837	0.923	0.342	0.000	0.427	0.384	2.51	3.61	3.16	202.89	133.34	69.55
	Au	0.204	0.903	0.909	0.112	0.000	0.415	0.418	4.80	0.03	0.02	141.80	102.16	39.64
Conservatism	US	0.619	0.620	0.800	0.418	0.000	0.664	0.678	2.68	13.91	9.27	88.26	44.91	43.55
	Au	0.683	0.708	0.598	0.359	0.575	0.607	0.559	1.84	5.00	2.09	22.81	10.94	11.87
Neuroticism	US	0.092	0.384	0.641	0.403	0.000	0.831	0.767	4.71	11.19	6.69	69.61	34.89	34.72
	Au	0.058	0.502	0.646	0.244	0.000	0.829	0.766	6.71	5.46	0.54	59.85	43.89	15.96
Church	US	0.821	0.758	0.857	0.396	0.000	0.607	0.518	2.95	7.73	7.57	43.45	13.24	30.21
attendance	Au	0.754	0.612	0.810	0.411	0.029	0.676	0.587	0.15	12.70	5.10	43.84	21.69	41.15
Political	US	0.642	0.545	0.396	0.475	0.633	0.691	0.665	1.85	3.71	10.47	11.83	4.53	7.30
affiliation	Au	0.831	0.000	0.509	0.822	0.706	0.471	0.493	0.35	4.98	3.54	13.28	4.80	8.48
Educational	US	0.569	0.836	0.933	0.392	0.000	0.383	0.359	4.37	7.08	6.75	91.63	45.14	46.49
attainment	Au	0.509	0.733	0.871	0.317	0.000	0.550	0.491	1.52	4.05	2.18	67.34	37.24	30.10

Estimates of the shared environmental effect are large for many of the variables of concern to social and political scientists. Indeed, the shared environment is clearly the largest contributor to individual differences in political preference and is comparable to or greater than the estimate of genetic effects in many cases.

Table 12 summarizes the model fitting results when the correlations between mates are included in the data and the model is adjusted to reflect the corresponding increase in the genetic correlation between DZ twins, γ. The goodness-of-fit tests of the model tell a similar story, as do the tests for the effects of genetic differences. This finding is expected, because the information about the significance of genetic effects comes from the observed difference between MZ and DZ correlations which is not affected by allowing for assortment. However, including the correlation between mates reduces the estimates and significance of the shared environmental effects because any genetic effects that might have biased estimates of the shared environment due to assortative mating are now correctly removed and transferred to estimates of the genetic component. Thus, the change in fit due to removing C from the model is much smaller in Table 12.

Table 13 shows the revised estimates of the proportions of variance after including the correlation between mates in the data and allowing for phenotypic assortment in the model. Estimates of the unique environmental contribution (E) barely change because these are almost entirely determined from the differences within monozygotic twin pairs. As expected, allowing for assortative mating tends to increase estimates of the genetic contribution and reduce the apparent influence of the shared environment.

Table 13: Relative contributions (% total variation explained) of genetic transmission on outcome measures (twin data, with spouse pairs).

	Sex	*Males*			*Females*		
Trait	Source	Genes	Shared E	Unique E	Genes	Shared E	Unique E
Stature	US	70.0	11.7	18.2	85.2	0.0	14.8
	Au	81.5	1.3	17.2	82.5	0.0	17.5
Conservatism	US	38.4	17.5	44.1	64.0	0.0	36.0
	Au	50.2	12.9	36.9	35.8	33.0	31.2
Neuroticism	US	14.7	16.2	69.0	41.1	0.0	58.9
	Au	25.2	0.06	68.8	41.7	0.0	58.3
Church	US	57.4	15.7	26.9	73.4	0.0	26.6
attendance	Au	37.4	16.9	45.7	65.6	0.1	34.3
Political	US	29.7	22.6	47.7	15.7	40.1	44.2
affiliation	Au	0.0	77.8	22.2	25.9	49.9	24.3
Educational	US	70.0	15.4	14.7	87.1	0.0	12.9
attainment	Au	59.8	10.0	30.2	75.9	0.0	24.1

Correcting for assortative mating has relatively little effect on the estimated contribution of the shared environment to political affiliation, since the genetic component is relatively small in the first place. The impact of assortative mating on the genetic correlation between siblings/DZ twins is largest when both the heritability and marital correlation are large. By contrast, allowing for assortative mating for educational attainment all but eradicates the case for shared environmental effects on this trait because the difference between MZ and DZ correlations is far greater in the first place.

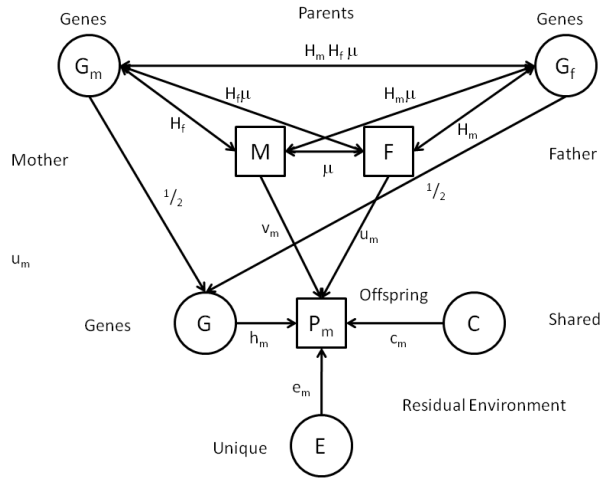
Integrating Twin and Nuclear Family Data.

Considered separately, twin and nuclear family data have their specific strengths and shortcomings. Although there is much variation between the specific conclusions for individual outcomes and samples,

the broad picture from nuclear families is that there is substantial and highly significant familial clustering of individual differences between and within generations. Parents and offspring and siblings correlate very highly. That being said, between 30 and 90% of variation does not depend on family clustering but on individual unique random environmental effects and/or the segregation of genetic differences within sibships in accordance with Mendel's first law. Nuclear family data do not generally help discriminate genetic and cultural models for transmission between parents and children. The fit of both tends to be similar to the identical data. However, models that invoke the genetic transmission of a latent genetic variable tend to be more parsimonious. The most elaborate model for direct phenotypic transmission from parent to offspring tends to lead to larger estimates of the residual shared environmental correlation than a model invoking latent genetic transmission. That is, the "genetic model" for nuclear families explains more, more simply. If genetic transmission is indeed a significant component in family resemblance for social traits, then there is an important implication that explaining transmission genetically also explains part of the residual variation between and within sibships without invoking new principles. If it were possible to "find the genes" then there is a chance of explaining multiple statistical sources of individual differences by reference to the same underlying mechanism. Such speculation, however, is unwarranted in advance of a convincing demonstration that the data are not merely consistent with genetic theory but actually demand it. For the most part, the twin data suggest strongly that a genetic explanation of some of the variation is required if the equal environments assumption is justified. The problem of the twin study lies with its relatively blunt analysis of the environment. Almost all the variables we studied required a model that involved some shared environmental effects, although their source was not clear. When allowance was made for the genetic consequences of assortative mating, the apparent effects of the shared environment were often reduced. This finding is consistent with the conclusion from the nuclear family data that a genetic explanation of parent-offspring correlation, with appropriate allowance for assortative mating, also reduces the apparent residual effects of the shared environment compared with models that assume direct transmission between the measured phenotypes of parents and offspring.

The principal limitation of the models so far is that they have treated genetic and cultural transmission as alternative explanations of parent-offspring transmission. This limitation arises because nuclear family and twin correlations considered separately do not make it possible to consider both mechanisms simultaneously. A joint analysis of the twin and nuclear family data makes it possible to estimate the genetic and environmental contributions to transmission and variation simultaneously. Figure 5 shows the elements of an integrated model for genetic and vertical cultural inheritance for nuclear families. The diagram only includes parents and a single male offspring. The diagram for female offspring is identical in shape with the substitution of u_f, v_f, h_f, c_f and e_f for the path coefficients u_m, v_m, h_m, c_m and e_m respectively. The key to the integrated transmission model lies in the fact that the diagram shows intergenerational paths from parental genotypes and phenotypes to offspring genotype and phenotype (Figure 5). Furthermore, the model allows for the correlation between mates and the implications of phenotypic assortative mating for family resemblance. The offspring phenotype, P_m (in males) is the linear sum of the contributions of genes (G_m), parental phenotypes (M and F) and residual shared (C) and unique (E) environmental effects. The diagram may be extended to include a sibling, DZ cotwin, or MZ cotwin . We focus only on one offspring in the diagram to minimize confusion.

Figure 5. Combined model for biological and cultural inheritance in nuclear families.



Note: Diagram is shown for male offspring only. The diagram for females is generated by replacing subscript "m" by "f" on paths where appropriate. Cultural inheritance is assumed to occur directly between the phenotypes of parents and offspring. Other models are possible (see e.g. Heath et al., 1985)

An important implication of joint genetic and cultural inheritance is that the *correlations* between genotype and phenotype, H_m and H_f, are not the same as the *path coefficients* between genotypes and phenotypes, h_m and h_f respectively. Thus, from the diagram, it can be seen that, if H_m and H_f are the genotype-phenotype correlations in mothers and fathers, the expected correlation between offspring genotype and (male) phenotype is not h_m but

$$H'_m = h_m + \tfrac{1}{2}H_m(v_m + \mu u_m) + \tfrac{1}{2}H_f(u_m + \mu v_m).$$

Similarly:

$$H'_f = h_f + \tfrac{1}{2}H_m(v_m + \mu u_m) + \tfrac{1}{2}H_f(u_m + \mu v_m).$$

If the intergenerational paths and correlation between mates do not change over time then $H'_m = H_m$ and $H'_f = H_f$. These constraints may be imposed numerically when estimating the parameters, allowing identification of the model parameters. Whether or not a given population attains equilibrium under joint biological and cultural inheritance with assortative mating is questionable for traits that are historically labile. However, analytical and simulation studies (e.g. Medland and Keller, 2009) show that the approach to equilibrium is quite rapid so violations of the constraint may not be too critical.

There is no single way of parameterizing the same model and no one "best" conception of how environmental transmission should be modeled. For other path models using slightly different conceptions of non-genetic inheritance see, e.g. Heath et al, (1985) Maes et al. (2009). Generally, detailed subtleties of transmission are difficult to resolve with realistic sample sizes.

The algebraic expectations for the correlations between twins and relatives within nuclear families are given in Table 14. The model as presented in the table makes some simplifying assumption. In particular, it is assumed that the same genes and shared environmental effects affect males and females, although there may be sex differences in the effects of these components on the phenotype. In performing the analysis, we test this assumption by relaxing this constraint and allowing for sex-specific genetic and environmental effects i.e. effects are expressed in one sex but not in the other. The expectations for the

more complex model are not tabulated but have been implemented in the general algorithm for model-fitting.

Table 14: Expected correlations between relatives under model for joint biological and cultural inheritance

Relationship	*Expected Correlation*
Spouses	μ
Mother-Daughter	$v_f + \mu u_f + \frac{1}{2} h_f(H_F+\mu H_m)$
Mother-Son	$v_m + \mu u_m + \frac{1}{2} h_m(H_F+\mu H_m)$
Father-Daughter	$u_f + \mu v_f + \frac{1}{2} h_f(H_m+\mu H_f)$
Father-Son	$u_m + \mu v_m + \frac{1}{2} h_m(H_m+\mu H_f)$
Male siblings	$\gamma h_m^2 + u_m^2 + v_m^2 + 2\mu u_m v_m + h_m[v_m(H_f + \mu H_m) + u_m(H_m+\mu H_f)] + c_m^2$
Female siblings	$\gamma h_f^2 + u_f^2 + v_f^2 + 2\mu u_f v_f + h_f[v_f(H_f + \mu H_m) + u_f(H_m+\mu H_f)] + c_f^2$
Male-Female siblings	$\gamma h_m h_f + u_m u_f + v_m v_f + \mu(u_f v_m + u_m v_f) + \frac{1}{2} h_m[v_f(H_f + \mu H_m) + u_f(H_m+\mu H_f)] + \frac{1}{2} h_f[v_m(H_f + \mu H_m) + u_m(H_m+\mu H_f)] + c_m c_f$
Male DZ	$\gamma h_m^2 + u_m^2 + v_m^2 + 2\mu u_m v_m + h_m[v_m(H_f + \mu H_m) + u_m(H_m+\mu H_f)] + c_m^2 + t_m^2$
Female DZ	$\gamma h_f^2 + u_f^2 + v_f^2 + 2\mu u_f v_f + h_f[v_f(H_f + \mu H_m) + u_f(H_m+\mu H_f)] + c_f^2 + t_f^2$
Male-Female DZ	$\gamma h_m h_f + u_m u_f + v_m v_f + \mu(u_f v_m + u_m v_f) + \frac{1}{2} h_m[v_f(H_f + \mu H_m) + u_f(H_m+\mu H_f)] + \frac{1}{2} h_f[v_m(H_f + \mu H_m) + u_m(H_m+\mu H_f)] + c_m c_f + t_m t_f$
Male MZ	$h_m^2 + u_m^2 + v_m^2 + 2\mu u_m v_m + h_m[v_m(H_f + \mu H_m) + u_m(H_m+\mu H_f)] + c_m^2 + t_m^2$
Female MZ	$h_f^2 + u_f^2 + v_f^2 + 2\mu u_f v_f + h_f[v_f(H_f + \mu H_m) + u_f(H_m+\mu H_f)] + c_f^2 + t_f^2$
Where:	$\gamma = \frac{1}{2}(1+\mu H_m H_f)$
Subject to:	$1-h_m^2 + u_m^2 + v_m^2 + 2\mu u_m v_m + h_m[v_m(H_f + \mu H_m) + u_m(H_m+\mu H_f)] + c_m^2 + t_m^2 + e_m^2 = 0$
	$1-h_f^2 + u_f^2 + v_f^2 + 2\mu u_f v_f + h_f[v_f(H_f + \mu H_m) + u_f(H_m+\mu H_f)] + c_f^2 + t_f^2 + e_f^2 = 0$
	$H'_m - h_m + \frac{1}{2} H_m(v_m + \mu u_m) + \frac{1}{2} H_f(u_m + \mu v_m) = 0$
	$H'_f - h_f + \frac{1}{2} H_m(v_m + \mu u_m) + \frac{1}{2} H_f(u_m + \mu v_m) = 0$

Note: The model tabulated does not incorporate genetic dominance or sex-specific genetic and environmental effects.

The expectations for the twin correlations contain two additional parameters, t_m and t_f, to allow for the possibility that MZ and DZ twins may share greater environmental similarity than siblings. This parameterization assumes that twins do not experience qualitatively different environments from non-twin siblings but that the residual environments of both types of twins correlate more highly than siblings. Note that this does not deal with the equal environments assumption because the model still assumes that the environments of MZ twins are no more highly correlated than those of DZs. It is apparent from the expectations that there are four non-linear constraints to be imposed on the parameter estimates. As written, the model assumes that gene action is additive. It is not possible to estimate the effects of dominance at the same time as the shared sibling environment. However, if we are prepared to assume that $c_m=c_f=0$ their shared environmental parameters may be replace by parameters d_m and d_f to allow for the effects of dominance in males and females. The dominance variance component in MZ twins is d_m^2 in males and d_f^2 in females. The coefficient of the dominance component in siblings and DZ twins is ¼. Dominance does not contribute to parent-offspring resemblance and is not affected by assortative mating in polygenic systems (e.g. Fisher, 1918; Falconer and MacKay, 1995).

Four equality constraints allow the model to be identified (c.f. Table 14). Two constraints result from the fact that we are analyzing correlations so the total phenotypic variance is constrained to be unity in males and females. The other two follow from the assumption of equilibrium for the correlations H_m and H_f under the combined effects of genetic and non-genetic inheritance in the presence of phenotypic assortative mating. The expectations and constraints were coded for the *Mx* package for structural modeling. The model was fitted simultaneously by weighted least squares to all 12 sets of 13 correlations between relatives. Convergence required 30 seconds CPU time on a Dell Inspiron 1420 laptop computer.

The model in Table 14 has 11 free parameters that are estimated from 13 correlations for each variable in each sample. Thus, the residual weighted sum of squares for the full model has 13-11 d.f. When allowance is made for sex-specific residual sibling shared environmental and excess shared twin environmental effects two additional parameters are required and the model should fit perfectly ($\Sigma\delta^2 = 0$) unless there are active boundary constraints on one or more parameters. Goodness of fit tests for the full model and for a series of reduced models are shown in Table 15. In no case does a purely additive genetic model (Model 3) account for the pattern of family resemblance. Similarly, the combined data do not generally justify ignoring the role of genetic factors (Model 7). Political affiliation is the only apparent exception to this rule. A model that excludes genetic factors gives quite a good fit to the correlations for political affiliation ($\Sigma\delta^2_{(2)} = 4.53$ and 6.00 in Virginia and Australia respectively).

Beyond this basic conclusion, there is clearly no "one size fits all" answer to the question of the relative sizes and types of genetic and environmental effects on the variables included in this investigation. Different variables show different patterns of genetic and environment influence. The goodness of fit tests in Table 15 suggest a high degree of consistency between the best-fitting models for the different outcomes in the two large samples from Virginia and Australia. The models that seem to give the most consistent acceptable fit in both samples are indicated in bold type in Table 15. In each case, we identify a model that has the same structure in both populations. Thus, although it is impossible to exclude genes and all sources of shared environmental variance for conservatism, church attendance and education, a model that eliminates non-genetic parent-offspring transmission (Model 2) gives an acceptable fit to these variables. The "best" model for political preference does not require genetic inheritance overall (Model 7). For stature and neuroticism an adequate model includes the effects of genetic dominance (Model 8). The picture is summarized in Table 16 which shows the parameter estimates for the "best" model for each variable. Blank cells represent parameters that were fixed at zero in the selected reduced models. The models incorporate additional parameters (h'_f, c'_f etc.) to allow for sex-specific effects (see e.g. Truett et al., 1994; Maes et al., 2009). Zero estimates of these parameters are consistent with the absence of sex specific effects of the corresponding outcome. The large estimates of d'_f for stature and neuroticism are consistent with sex-specific effects of genetic dominance on these variables.

Although many of the variables provide evidence for significant residual shared environmental resemblance between siblings and twins, perhaps the most striking feature of the overall findings is that the best fitting models do not generally provide strong support for direct non-genetic influence of parental phenotypes on the phenotypes of their offspring. The only exception is political affiliation in both populations where large parent-offspring correlations and small differences in MZ and DZ correlations conspire to favor a purely environmental explanation of parent-offspring resemblance. Political affiliation also reflects some of the larger effects of other shared environmental influences including the increased environmental similarity specific to twins.

Table 15: Model Comparison Statistics: Combined twin and nuclear family data.

			Weighted residual SS = $\Sigma\delta^2$											
	Trait		Stature		Conservatism		Neuroticism		Church attendance		Political affiliation		Educational attainment	
	Model	d.f.	US	Au	US	Au	US	Au	US	Au	US	Au	US	Au
1	Full model: genes+VCI[1]	0[2]	0.00	1.93	0.00	0.00	1.43	9.18	0.00	0.18	0.00	5.79	0.00	0.10
2	No vertical cultural inheritance ("VCI")	4	5.42	12.22	**9.08**	**5.35**	3.78	13.77	**2.26**	**6.31**	15.49	32.67	**9.52**	**9.25**
3	No shared environment/dominance	10	43.81	43.25	38.73	75.07	32.40	41.36	45.58	56.12	49.78	38.60	239.32	341.56
4	No twin or sibling shared environment	6	18.31	15.13	9.77	34.76	14.79	22.51	29.78	7.38	37.61	29.96	161.77	134.89
5	No sibling shared environment	3	2.92	5.49	3.67	28.97	1.44	11.01	10.70	5.10	10.22	5.81	103.89	85.32
6	No twin shared environment	3	6.18	9.42	1.71	8.42	10.63	16.78	10.97	5.45	30.58	28.25	12.06	17.23
7	No genes	2	133.34	102.16	44.91	10.94	34.02	43.89	13.24	21.71	**4.53**	**6.00**	45.14	37.24
8	Model 3 plus sex-specific A and D	6	**7.81**	**13.37**	5.39	33.13	**7.75**	**8.41**	15.68	17.57	42.36	36.87	88.49	109.82

Notes: [1] No dominant genetic effects; sex differences in effects of genes and shared environment, but no sex-specific genetic effects. [2] This model should give a perfect fit in the absence of non-additive genetic effects.

Significance levels of χ^2

	P (%)				
d.f.	10	5	1	0.5	0.1
2	4.61	5.99	9.21	10.60	13.82
3	6.25	7.81	11.34	12.84	16.27
4	7.78	9.49	13.28	14.86	18.47
6	10.64	12.59	16.81	18.55	22.46
8	13.36	15.51	20.09	21.95	26.13
10	15.99	18.31	23.21	25.19	29.59

Table 16: Parameter estimates for best-fitting models for genetic and environmental components of family resemblance.

Parameter		Stature		Conservatism		Neuroticism		Church attend.		PID		Educational att.	
		US	Au	US	Au	US	Au	US	Au	US	Au	US	Au
Additive genetic	h_m	0.853	0.827	0.670	0.697	0.480	0.536	0.797	0.659			0.771	0.583
	h_f	0.817	0.818	0.751	0.747	0.522	0.438	0.788	0.700			0.764	0.638
	h'_f	0.185	0.286			0.146	0.270						
Sibling shared environment	c_m			0.217	0.304			0.121	0.232	0.438	0.157	0.378	0.428
	c_f			-0.009	0.184			-0.163	0.261	0.313	0.015	0.373	0.278
	c'_f			0.186	0.274			0.185	0.000	0.106	0.000	0.210	0.387
Dominant Genetic	d_m	0.346	0.403			0.332	0.237						
	d_f	0.119	-0.087			-0.010	-0.135						
	d'_f	0.367	0.221			0.358	0.358						
Twin shared Environment	t_m			0.269	0.239			0.299	0.254	0.324	0.420	0.343	0.390
	t_f			-0.067	0.211			0.107	-0.074	0.231	0.124	0.044	0.264
	t'_f			0.170	0.000			0.191	0.278	0.408	0.384	0.300	0.165
Vertical cultural transmission	u_m									0.268	0.464		
	u_f									0.206	0.331		
	v_m									0.176	0.233		
	v_f									0.321	0.535		
Marital	μ	0.223	0.207	0.618	0.685	0.092	0.060	0.821	0.753	0.642	0.834	0.567	0.507
Goodness-of-fit[1]	$\Sigma\delta^2$	7.81	13.37	9.08	5.35	7.75	8.41	2.26	6.31	4.53	6.00	9.52	9.25
	d.f.	6	6	4	4	6	6	4	4	2	2	4	4
	P%	>10	5-10	1-5	>10	>10	>10	>10	>10	>10	5	1-5	5-10

Note: [1] Probability (P%) assumes weighted residual sum of squares is distributed as chi-square.

Table 17 contains the estimated proportions of the total variance attributed to each of the sources in the best-fitting model for each of the outcome measures in males and females. The estimates allow for any effects of assortative mating on the total phenotypic variance.

Table 17: Proportions (%) of variance in phenotype attributed to sources in best-fitting model.

		Males						Females					
		V_A	V_D	V_E	V_{EC}	V_{ET}	V_{CI}	V_A	V_D	V_E	V_{EC}	V_{ET}	V_{CI}
Stature	US	72.8	11.9	15.3				76.7	14.9	14.4			
	Au	68.3	16.2	15.4				76.2	5.7	18.1			
Conservatism	US	44.8		43.2	4.7	7.2		56.5		36.7	3.5	3.3	
	Au	48.6		36.5	9.3	5.7		55.8		28.9	10.9	4.4	
Neuroticism	US	23.1	11.0	65.9				29.5	11.8	58.7			
	Au	28.7	5.6	65.7				26.6	14.7	58.7			
Church Attendance	US	63.5		26.1	2.1	8.9		62.1		27.1	6.1	4.8	
	Au	43.4		44.8	1.5	6.5		49.0		36.0	6.8	8.3	
Political Affiliation	US			51.8	19.2	10.5	18.5			46.4	10.9	20.0	20.7
	Au			21.9	2.5	17.6	58.1			28.8	0.0	16.3	54.9
Educational Attainment	US	59.4		14.5	14.3	11.8		58.4		14.1	18.3	9.2	
	Au	34.0		32.5	18.3	15.2		40.7		26.9	22.7	9.7	

Note. Sources of variance: V_A =Additive genetic variance; V_D = Dominance genetic variance; V_E = Residual, unique environmental variance within sibships; V_{EC} = Residual shared environmental variance among sibships; V_{ET} = Additional shared environmental variance between twin pairs; V_{CI} = Variance due to non-genetic ("cultural") inheritance from parental phenotype.

The estimates for stature provide a benchmark for what is typical of a variable for which family resemblance is entirely genetic. When allowance is made for the modest degree of assortative mating, additive and dominant genetic effects together account for 80-85% of the total variation, the remainder being due to random, unique environmental effects that are uncorrelated between family members. The estimates are remarkably consistent across sexes and between the two populations sampled. The same model fits the data on neuroticism but, for this personality variable, the effects of genetic differences account for only 35-40% of the phenotypic variance. The finding that variation in the major dimensions of personality is only modestly heritable and caused primarily by non-familial environmental effects has been long-established in the behavioral genetics literature (e.g. Eaves et al., 1989; Plomin et al., 2001).

For the purposes of the current investigation, the results for the other variables are challenging and compelling. Firstly, the best model for political affiliation in both populations does not require genetic influences. All the variance is due to the environment. The effects of the environment span all the sources included in the model. Although the basic model is the same, the actual proportions explained by the sources of variance differ greatly between the two contexts. There are small but statistically significant effects of the residual shared environment among sibships (V_{EC}) and excess shared environmental similarity between twins (V_{ET}). Together these effects account for 15-30% of the total variation in outcome. Both populations show large effects of the environment transmitted by parents and influences specific to individual subjects. However, the balance of these contributions is markedly different between the US and Australia. In the US, unique environmental effects account for c. 45-50% of the total variance, compared with only 20-30% in Australia, depending on sex. The direct contribution of parents to the political preferences of the adult offspring (V_{CI}) accounts for a much smaller proportion of the variance in the US (c.20%) compared with Australia (c. 55-60%). Although it is dangerous to read too much out of such findings, they would suggest that there is far less intergenerational mobility in the political affiliations of Australians than Americans.

At least part of this difference may be due to the fact that the items address somewhat different aspects of political affiliation in the two studies. In the US, the item comprised a categorical rating of the strength of

preference for one or another of the two major political parties. In Australia, subjects were asked to report their voting choice among the principal political parties.

Many social scientists will be surprised by our claim the data are consistent with the contribution of additive genetic factors to individual differences in conservatism, church attendance and educational attainment. The fact that other variables (stature and political affiliation) support two extremely different models for family resemblance, one purely genetic (stature) and one purely environmental (political affiliation) imply that the models and methods are not inherently biased against environmental explanations. Taken at their face value, the results for conservatism, church attendance and educational attainment imply that much of the evidence for the shared environment from twin studies disappears when allowance is made for the genetic consequences of mate selection and that genetic effects account for c. 35-50% of the total variance depending on measure and context. The effects of residual shared environmental effects on conservatism and church attendance are significant but relatively small (c.5-16% of the total). For educational attainment the residual shared environmental effects are larger accounting for approximately 30% of the total variance.

Testing Assumptions About Mate Selection.

The above analyses of resemblance between twins and members of nuclear families make it very clear that spouses are significantly correlated for all the outcomes chosen for study and that these correlations are especially large for those socially significant variables that comprise the backbone of the current investigation: social attitudes; voting behavior; religious practice and educational attainment. Correlations between mates for these variables are among the largest in the literature and often comparable to those for monozygotic twins. By contrast, the correlations between mates for stature and neuroticism are much lower. This finding is generally consistent with those for other anthropometric, physiological and personality traits in the literature (see e.g. Pearson and Lee, 1903; Eaves et al., 1989) and in our own data (e.g. Eaves et al., 1999).

The consequences of the correlation between mates for the correlations between other relatives and for the family clustering of socially important traits depends significantly on the mechanism underlying spousal resemblance. Our analyses so far make the strong and untested assumption that the correlation between mates is due to assortative mating, i.e. that "like tends to marry like" for the traits in question and that the attitudes and behavior of spouses does not converge significantly due to their mutual interaction during their life together. Furthermore, our model has assumed that assortative mating is primarily based on the actual phenotype being measured for the partners themselves (stature, personality, church attendance) etc. and not on some other correlated variable such as income, or even the phenotypes of other family members such as parental or sibling education, political affiliation or religious belief.

There is no single mechanism, or taxonomy of mechanisms of spousal resemblance, nor are they mutually exclusive. Further, there is no one "best" approach for their resolution. An exhaustive treatment is a chapter in its own right. Here we focus on outlining the principal mechanisms and illustrate one approach that allows them to be compared on a common footing with the minimum of complex structural modeling. The integrating framework for our treatment of assortative mating is the recognition that mates select one another for some feature, measured or un-measured, of their phenotypes. People may choose one another because they prefer people who share their religious, social or political values or they may be brought together by shared background variables, perhaps including aspects of the homes or neighborhoods in which they grew up. Our treatment of alternative models for assortative mating begins by considering how different selection criteria influence the correlations between relatives and their spouses. If all we were given were a set of correlations between mates, we would find it hard to distinguish between the different processes of mate selection. However, the unique structure of the Virginian and Australian samples allows us to examine the impact of different types of assortment on the correlations between MZ and DZ twins and their spouses, and the correlations between siblings and their

spouses. It turns out that these additional relationships can shed significant light on the causes of the observed correlations between mates.

Table 18 summarizes the data that will form the backbone of our analysis of mate selection for the variables chosen to illustrate this introduction. They comprise the correlations between twins, siblings and spouses for the six illustrative variables and those between the various combinations of in-laws that can be obtained when we include the spouses of siblings and twins in the data set. Correlations are tabulated for both the Virginian and Australian samples.

Ultimately, we want to use all the data for each variable to test alternative theories for the process underlying spousal resemblance between these measures, much as we fitted multiple models for biological and cultural inheritance to the correlations in Table 3. However, we begin by exploring some of the basic concepts and models that may be used to analyze patterns of spousal resemblance and show how they affect the correlations between twins, siblings and their spouses.

Phenotypic Assortative Mating Suppose initially that mate selection is based directly on the measured trait under investigation, for example, we measure voting behavior or conservatism in pairs of twins, T_1 and T_2, and their spouses, S_1 and S_2 (Figure 6). The correlation between twins for the "mate selection" phenotype is *t* and that between spouses is μ as before. Since mate selection is assumed to depend only on the selection of a spouse by his/her partner and not on characteristics of the other spouse or cotwin, the correlations between a spouse and the cotwin of his/her partner is a secondary consequence of the phenotypes of the partners themselves. Thus, $r_{S1T2}=r_{S2T1}=\mu t$. Similarly, the correlation between the spouses of twins is expected to be $r_{S1S2}=\mu^2 t$ (See Heath et al, 1985, for a more extensive treatment of the correlations between the relatives of spouses under different mechanisms of assortment).

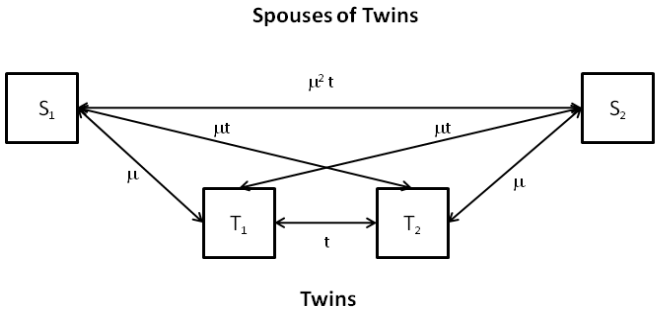
If this simple model is adequate to account for mate selection for a given trait (e.g. "conservatism") then it should be possible to predict the correlations r_{S1T2}, r_{S2T1} and r_{S1S2} from the correlations between twins, *t*, and the correlations between spouses, μ. Thus, if the basic model for assortment is correct, we expect $r_{S1T2}=r_{S2T1}=r_{T1T2}r_{S1T1}$ and $r_{S1S2}=r_{T1T2}r^2_{S1T1}$. If the model is wrong, then these expectations will not apply and a better model will be called for.

Table 18: Raw Correlations Between Married Relative Pairs in US and Australia

Relationship		*Stature*		*Conservatism*		*Neuroticism*		*Church attendance*		*Political affiliation*		*Educational Attainment*	
		N	r	N	r	N	r	N	r	N	r	N	r
Spouses	US	4850	0.238	4737	0.648	4886	0.087	4831	0.757	4440	0.648	4839	0.565
	AU	3383	0.215	3415	0.666	3446	0.056	3406	0.819	2551	0.832	3379	0.509
Male sibling	US	870	0.415	864	0.397	884	0.116	887	0.356	820	0.369	886	0.567
	AU	866	0.395	871	0.426	876	0.153	859	0.437	648	0.535	895	0.411
Female sibling	US	1871	0.436	1821	0.444	1874	0.202	1866	0.368	1664	0.297	1883	0.586
	AU	2169	0.396	2180	0.441	2222	0.144	2185	0.481	1531	0.487	2208	0.486
Unlike-sex sibling	US	2312	0.407	2283	0.374	2324	0.157	2347	0.378	2096	0.306	2344	0.568
	AU	2678	0.376	2696	0.389	2721	0.122	2669	0.420	1969	0.489	2746	0.354
DZ male	US	337	0.484	324	0.395	330	0.196	337	0.449	303	0.426	329	0.618
	AU	174	0.383	171	0.538	175	0.086	176	0.513	147	0.774	211	0.498
DZ female	US	532	0.550	498	0.480	526	0.331	529	0.435	449	0.436	520	0.689
	AU	520	0.502	508	0.534	528	0.217	517	0.528	420	0.531	579	0.523
DZ unlike-sex	US	690	0.393	645	0.365	682	0.087	679	0.295	589	0.379	684	0.576
	AU	451	0.485	453	0.446	459	0.125	450	0.450	364	0.555	529	0.466
MZ male	US	506	0.841	493	0.600	504	0.372	496	0.476	469	0.480	493	0.865
	AU	324	0.857	323	0.574	327	0.314	317	0.660	269	0.670	363	0.732
MZ female	US	898	0.818	844	0.642	895	0.425	890	0.584	818	0.513	895	0.812
	AU	879	0.851	874	0.648	903	0.431	894	0.656	718	0.671	948	0.766
Spouses of DZM	US	102	0.118	101	0.196	101	-0.024	102	0.482	89	0.319	104	0.203
	AU	35	-0.169	36	-0.313	35	0.201	36	0.507	25	0.718	34	0.173
Spouses of DZF	US	126	0.150	126	0.339	124	0.113	123	0.364	115	0.303	124	0.361
	AU	67	-0.121	70	0.294	70	0.013	67	0.630	54	0.360	68	0.254
Spouses of DZMF	US	172	0.045	169	0.194	169	-0.039	166	0.189	156	0.209	169	0.421
	AU	67	-0.114	68	0.426	67	-0.237	66	0.202	40	0.739	67	0.189
Spouses of MZM	US	179	0.195	178	0.338	186	-0.076	181	0.331	169	0.361	184	0.334
	AU	78	0.079	79	0.456	79	0.115	80	0.264	54	0.716	75	0.389
Spouses of MZF	US	306	0.240	295	0.261	302	0.041	305	0.403	287	0.195	305	0.476
	AU	178	0.200	186	0.260	183	0.032	181	0.388	129	0.533	185	0.431
Cotwin-Sp DZM	US	343	0.174	346	0.289	349	-0.058	354	0.311	309	0.404	349	0.392
	AU	151	0.169	147	0.273	152	0.101	153	0.534	115	0.686	159	0.387
Cotwin-Sp DZF	US	449	0.178	436	0.335	445	0.032	445	0.349	399	0.272	447	0.403
	AU	356	0.103	358	0.332	363	-0.011	353	0.517	281	0.169	374	0.291
Cotwin-Sp1 DZMF	US	296	0.154	286	0.220	292	-0.037	290	0.148	258	0.218	291	0.343
	AU	160	0.125	161	0.365	165	-0.093	157	0.324	123	0.463	179	0.277
Cotwin-Sp2 DZMF	US	334	0.175	316	0.354	332	-0.038	327	0.256	293	0.249	332	0.390
	AU	163	0.128	163	0.432	164	0.093	164	0.340	116	0.531	169	0.467
Cotwin-Sp MZM	US	524	0.300	519	0.476	536	0.077	530	0.393	495	0.358	529	0.475
	AU	296	0.211	295	0.446	301	0.053	296	0.467	217	0.610	303	0.348
Cotwin-Sp MZF	US	888	0.189	842	0.414	886	0.078	884	0.467	825	0.295	886	0.455
	AU	703	0.174	710	0.390	717	-0.006	705	0.442	530	0.451	726	0.498
H of FS-Bro-in-law	US	321	0.181	317	0.260	322	0.057	322	0.234	290	0.299	322	0.490
	AU	475	0.148	478	0.396	481	-0.064	468	0.439	329	0.319	481	0.304
H of FS-Sis-in-law	US	471	0.138	461	0.231	474	0.082	468	0.317	414	0.075	479	0.504
	AU	702	0.089	715	0.325	721	0.004	701	0.425	493	0.354	715	0.314
W of MS-Bro-in-law	US	254	0.143	258	0.360	259	0.033	260	0.164	236	0.138	264	0.342
	AU	266	0.129	274	0.361	271	0.061	265	0.409	190	0.458	266	0.272
W of MS-Sis-in-law	US	268	-0.001	268	0.178	272	0.164	274	0.329	239	0.220	272	0.427
	AU	333	0.121	327	0.251	339	0.091	336	0.282	233	0.298	327	0.367

Notes: All subjects selected for being married or living with partner. Unmarried subjects excluded from correlations. *Description of relationships involving DZMF pairs*. Cotwin-Sp1 DZMF = male twin, with husband of female co-twin. Cotwin-Sp2 DZMF = female twin, with wife of male co-twin

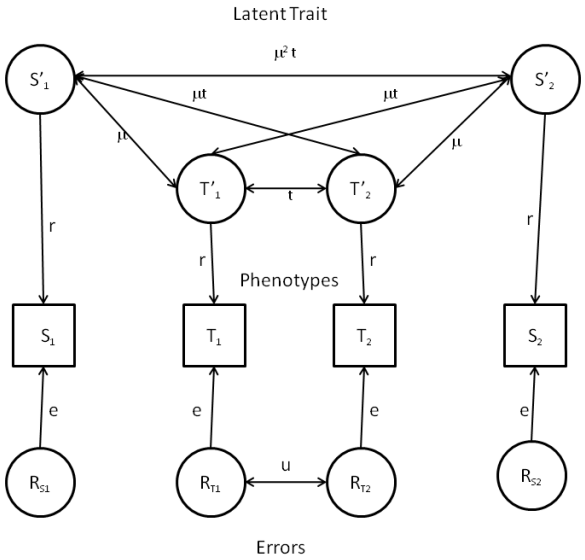
Figure 6. Pattern of correlation between twins and spouses when selection is based on measured phenotype.



Consider the case of assortment for church attendance. The correlation between mates in the US (Table 18) is 0.757. In Australia, it is 0.819. The correlations between female siblings are 0.368 and 0.481, respectively. If mating is assortative, we expect the correlation between husbands of female siblings and their sisters-in-law is expected to be 0.757x0.368=0.278 in the US and 0.819x0.481=0.394 in Australia. The observed correlations are 0.317 and 0.425 respectively. Although the agreement between the observed correlations and their expected values is not perfect, they lie well within the sampling errors of their differences suggesting, at first sight, that assortment for church attendance may be based on the mutual preference of church-goers for people like themselves in that respect and not for some other, unmeasured variable that influences mate selection.

Obviously, this is only an illustration to give the idea. In practice, we need to employ all the correlations, including those of other types of sib-pairs and the five kinds of twins. Also, estimates and tests of goodness of fit should embrace all the data, as they did in our analysis of the effects of genetic and cultural inheritance above. However, before doing that, we have to consider how the pattern of correlations might change under different theories of mating.

Figure 7. Error-prone measurement of mate selection for a latent trait ("phenotypic assortative mating plus error").



Phenotypic assortment for a latent trait. Suppose, now, that the correlation between mates is not based on the actual trait that is measured, but that the measured trait is an unreliable index of another trait on which mate selection is truly based according to the model in Figure 6. For example, we may measure the

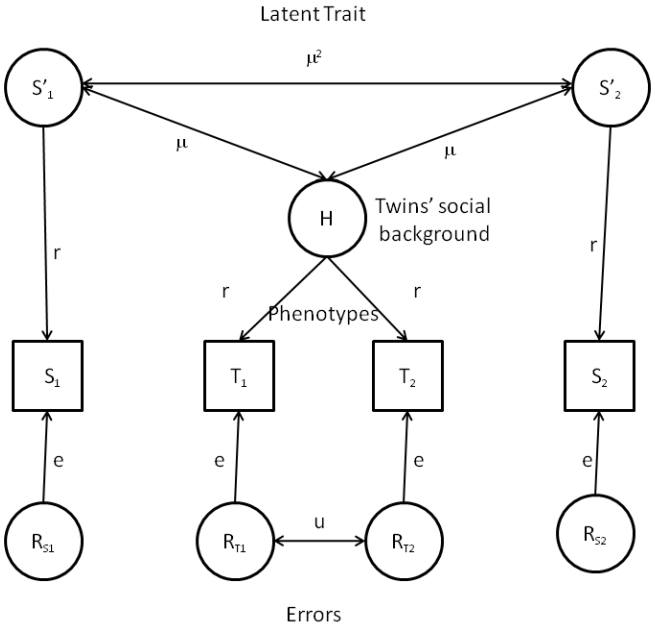
correlations between relatives using an IQ test, but mate selection is not based directly on test scores but on the actual cognitive abilities of the people being tested. We may call this model "phenotypic assortative mating with error" (see Heath et al., 1985). Figure 7 illustrates this model. Mate selection is now based on the latent variables S'_1, S'_2, T'_1 and T'_2 (in circles in the figure) while the actual measured values are S_1, S_2, T_1 and T_2 respectively (in squares). We let the path, *r*, denote the regression of observed trait on the latent variable ("true score") and $e=(1-r^2)^{½}$ the (standardized) path from the residuals, r_{S1}, r_{T1}, r_{T2}, and r_{S2} respectively (Figure 7). Note that in this case, the residuals are assumed to be uncorrelated. Furthermore, in this simple illustration, we assume that the path *r* is the same for men and women. The model in Figure 7 allows the residuals to be correlated between twins and siblings. This adjustment will allow us to test simple models for social homogamy (see below). However, initially we assume that the twin correlation, *u*, between residuals is zero.

If we derive new expected correlations from the model parameters we find $r_{S1T2}=r_{S2T1}=\mu tr^2$ for the correlations between spouses and the co-twins of their partners and $r_{S1S2}=\mu^2 t\, r^2$ for the correlation between the spouses of twins. These two correlations cannot now be predicted from the twin and spousal correlations alone since the observed correlation between twins is now expected to be tr^2 and that between mates should now be μr^2. A little algebra will show that the correlation between spouses and their partners' cotwins is no longer expected to be $r_{S1T2}=r_{S2T1}=r_{T1T2}r_{S1T1}$. Rather, we expect $r_{S1T2}=r_{S2T1}=\mu tr^2 > r_{T1T2}r_{S1T1} = \mu tr^4$ and $r_{S1S2}=\mu^2 tr^2 > r_{T1T2}r_{S1T1}{}^2 = \mu^2 tr^4$. A first guess at the path, *r*, is thus given by $[(r_{T1T2}r_{S1S2})/r_{S1T2}]^{½}$. Substituting the values for the correlations for church attendance in the example above we obtain rough estimates of r=0.936 and 0.905 for the US and Australian samples respectively. These values are very close to unity, suggesting that reported church attendance is a very reliable index of the underlying trait for which spouses select one another in this domain. Again, more rigorous analysis would integrate all the correlations in a single analysis

Social Homogamy. The above model (Figure 7) assumes that the residual twin correlation, *u*, is zero and that the correlation between twins is less than unity and the twin correlation varies as a function of zygosity. If the latent trait is completely correlated in both MZ and DZ twins (and, for simplicity in siblings) we arrive at the model in Figure 8. The model in Figure 8 is identical to the previous figure, except that the latent variables in the twins are assumed to be identical. That is, they are assumed to be completely correlated in the same way that the effects of the "shared environment", C, are perfectly correlated in twins. This does not mean that there are no genetic effects on the measured phenotype but, in this simple model, they are assumed to contribute to the correlation between the residuals for biological relatives, *u*, and not to assortative mating. An important implication of this model is that, regardless of the residual correlation between twins, *u*, if mate selection is based purely on family background, H in the Figure, then the correlation between spouses and their partners, is expected to be the same as that between spouses and their partners' co-twins. That is, under this model for social homogamy, we expect $r_{S1T2} = r_{S1T1} = \mu r^2$ regardless of the residual twin correlation, *u*.

Even the most cursory examination of the correlations for church attendance, say, in Table 18, are enough to show that the correlations for church attendance between spouses and in-laws are all substantially smaller than the correlations between spouses. Thus, the data from Australia and Virginia provide very little support for a predominant role of purely social homogamy in mate selection for this trait. Indeed, the patterns of correlation for other variables for which there is marked assortative mating (Table 18) provide very weak support for predominantly social homogamy in preference to phenotypic assortment.

Figure 8: Social homogamy in twins and their spouses.



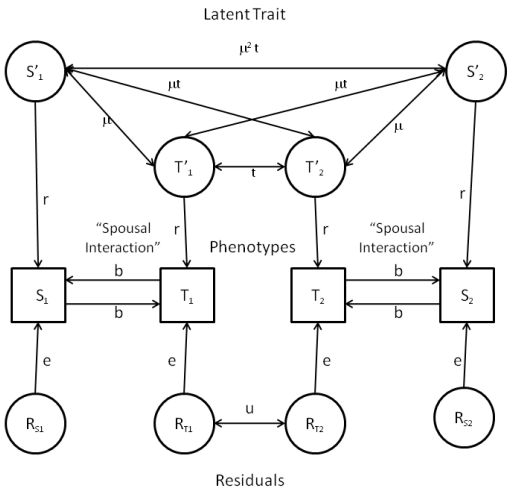
Spousal Interaction. All the models so far assume that the correlation between spouses is entirely due to assortative mating, i.e. to the mutual selection of spouses on the basis of latent or measured phenotypes and not to any mutual interaction after pairing. Thus, it is assumed that once mates have chosen each other their similarity is fixed for ever. They neither converge nor diverge with age or length of marriage. There are, of course, many ways of testing for marital interaction with spouse pairs , including examining the correlations or differences between spouses as a function of age or duration of marriage. This has been done many times, and the effects are modest at best, suggesting that convergence does not occur or that it occurs very rapidly after mate selection so that its effects are already established and completed within an interval that is beyond the resolving power of many cross sectional studies of age change in spousal correlation. Here we follow Heath (1987) and explore an alternative approach which examines the effect of spousal interaction on the correlations between twins (or other biological relatives) and their spouses. In its simplest form, this model assumes only that partners have been together long enough for convergence to be complete. More elaborate forms of the model can take into account variations in the duration of the partnership.

Before examining the algebra of spousal convergence, it may be helpful to think about a simple example. Imagine firstly, that twins are correlated for a trait of interest and that each chooses and interacts with a random partner. Thus, at the start of the relationship twins will be correlated and spouses will not be. Suppose now that each twin interacts with his/her partner so that, to some extent, each spouse is influences and is influenced by the other ("reciprocal spousal interaction"). With the passage of time, two things are expected to happen. One, obviously, is that pairs of spouses will become correlated, even though their correlation was zero initially. The second may not be so obvious, but now each twin is exposed to the influence of a separate randomly chosen environment (the spouse). In so far as the spouses of the twins are not correlated at the start, their effects on the twins will tend to increase the variance of the individual twins and lower their correlation compared with the initial value prior to mate selection.

Figure 9 is an extension of the model in Figure 7 to allow for the effects of spousal interaction; the parameter, *b*, represents the reciprocal influences of husbands and wives on each others' phenotypes. It is not necessary that the influence of husbands on their wives is the same as that of wives on husbands. The

basic mathematical treatment of spousal interaction is an application of the general model for the interaction between manifest variables in linear structural ("LISREL") models, such as those developed by Karl Joreskog in the late 1960's.

Figure 9: Extending the Model to Include Spousal Interaction.



We begin with the (4x4) correlation matrix, ***R***, between twins and spouses before interaction. If spouses are not correlated at the start of their relationship, ***R*** will have the form:

$$\begin{matrix} 1 & 0 & 0 & 0 \\ 0 & 1 & t & 0 \\ 0 & t & 1 & 0 \\ 0 & 0 & 0 & 1 \end{matrix}$$

where the rows and columns correspond to S1, T1, T2 and S2 respectively. The twin correlation at the start is *t.* The model then specifies the pattern of interaction between family members. Assuming for simplicity that the only interaction occurs between spouses (we could also allow for interaction between twins, see Carey, 1985), and that the coefficient *b* measures the reciprocal paths between S1 and T1 and between T2 and S2, we form the matrix ***B*** thus:

$$\begin{matrix} 0 & b & 0 & 0 \\ b & 0 & 0 & 0 \\ 0 & 0 & 0 & b \\ 0 & 0 & b & 0 \end{matrix}$$

The matrix is null except for those cells corresponding to those variables involved in the interaction. Element b_{12} of ***B*** denotes the path from the second individual in the family (the first twin in this example) to the first (his/her spouse). b_{21} measures the reciprocal effect of the spouse on the twin. The reciprocal effects do not have to be equal. Indeed, if twins affected their spouses but spouses did not affect their partners b_{21} would be zero. If spouses reacted against one another, *b* would be negative.

It may be shown (see e.g. Carey, 1985; Neale et al., 2004, Appendix D) that the covariance matrix between the relatives evolves during interaction to a stable value $\Sigma = \mathbf{(I-B)^{-1}\,R\,(I-B)'^{-1}}$ as long as **(I-B)** is positive definite, when the correlations between twins and their spouses after spousal interaction may

be obtained by standardizing Σ. To give an idea of how spousal interaction affects the correlations, assume that the twin correlation, *t*, is 0.6 at the time of mate selection and that spouses choose each other at random (i.e. the initial correlation between mates is zero). Now allow for the effects of reciprocal interaction with a value of, say, *b=0.4.* Substituting for *t* in ***R*** and *b* in ***B*** the above matrix multiplication yields the following standardized covariance matrix after spousal interaction:

1.00 0.69 0.21 0.08
0.69 1.00 0.52 0.21
0.21 0.52 1.00 0.69
0.08 0.21 0.69 1.00

That is, the correlation between mates has risen from zero at mate selection to 0.69 after spouses have interacted and the twin correlation has dropped from 0.6 to 0.52. Note also that the other correlations are no longer zero. Twins correlate 0.21 with the spouses of their co-twins and the spouses of twins now correlate 0.08. It is instructive to predict these two correlations under the alternative model that the correlation between mates is purely due to classical phenotypic assortment rather than spousal interaction. In this case, twins are expected to correlate 0.69x0.52=0.36 with the spouses of their cotwins and the correlation between the spouses of twins is expected to be 0.69x0.69x0.52=0.24. Both these two values are substantially greater than their observed values. The starting values have been chosen to illustrate some of the basic implications of spousal interaction for the correlations between relatives, and suggest how different theories of the correlation between mates predict different correlational patterns for in-laws. Thus, we may use the observed patterns of correlation between in-laws (e.g. the spouses of twins and siblings) to resolve alternative models for spousal resemblance as a precursor to analyzing the mechanisms of transmission within families.

As a rough guide, we suggest asking how the spouse-cotwin correlation and the correlation between the spouses of twins compare with what would be expected if the similarity between spouses were due only to phenotypic assortative mating. The previous treatment suggests that:

1. Phenotypic assortment for a latent variable, including social homogamy, generally produce spouse-cotwin and spouse-spouse correlations that are too high compared with those predicted from the twin and spousal correlations under the hypothesis of phenotypic assortment;
2. (Positive) spousal interaction leads to spouse-cotwin and spouse-spouse correlations that are too low compared with those predicted under phenotypic assortment.

Fitting Models for Spousal Resemblance.
It is helpful for the reader to examine some of the correlations in Table 18 to get a basic idea of which of the various models for assortment may best account for the observations. Remember that the sample sizes vary and that confidence intervals of correlations based on small numbers may be surprisingly large. For example, small correlations based on N=400 have CIs approximately +/- 0.1 so it is important not to over-interpret small differences.
We used the method of weighted least-squares described above to fit a series of models for mate selection to the six sets of correlations in Table 18 for the Virginia and Australian samples separately. The models considered are not exhaustive, but embrace some of the principal basic possibilities. They may be enumerated as follows.

1. No assortative mating or spousal correlation. This model is included as a baseline. Note that the model includes parameters for the correlations between five types of twins and three types of sibling pairs but assumes that the correlation between mates and all in-laws are zero. The

number of free parameters is 8 and there are 24 unique correlations for each sample, yielding 24-16 d.f. to test the goodness of fit, $S^{2.}$

2. Phenotypic assortative mating. This model corresponds to that in Figure 6. All the correlations between in-laws are assumed to be predicted from the marital correlation and the 8 correlations between biological relatives. Thus the model involves 8+1=9 parameters, yielding 15 d.f. for errors of fitting, and a difference of 1 d.f. (16-15) for comparing the fit of the model of phenotypic assortment with that assuming no assortment from the difference in weighted residual sums of squares, ΔS^2.
3. Phenotypic assortment plus error. This model corresponds to that in Figure 7. Separate estimates of *r* are specified for males and females. The residual effects are assumed to be uncorrelated between twins and siblings (*u=0).* Thus, this model has 9+2 free parameters and yields 13 d.f. for testing goodness of fit.
4. Spousal interaction. The model for spousal interaction is a reduced version of that in Figure 9. It assumes twins and sibling correlations initially take their own value and that all other correlations are zero at the start of the relationship between spouses. The correlations between mates and in-laws are assumed to result only from the interaction between twins (or siblings) and their spouses. Such an assumption is restrictive. The residual paths are assumed to be zero, and the reciprocal paths between spouses (*b*) are assumed to be symmetrical.
5. Social homogamy. The effects of social homogamy are approximated by the model in Figure 8. Mating is assumed to be based on the shared family background, H, which is perfectly correlated in MZ and DZ twins and siblings. Thus, social homogamy contributes equally to all the correlations between first-degree collateral relatives, and any differences among these correlations are explained by differences in the residual correlations between biological relatives. This model, thus, has 10 free parameters: the eight residual correlations between biological variables, the spousal correlation for family background and the path from family background to the outcome.

The above set of models is selected to illustrate the main characteristics of the mate selection process. The models can be made more elaborate by combining multiple elements in a single model, but problems of model identification, i.e. what combinations of effects can be estimated reliably, soon arise in this relatively simple data set so we combine ourselves to a restricted set of comparisons. Table 19 summarizes the goodness of fit tests of the selected models. Parameters estimates for conservatism, church attendance, political affiliation and educational attainment are summarized in Table 20. Those for stature and neuroticism are omitted since the correlations between spouses are smaller and there is little to be gained from a more detailed examination of the models for these variables in this context. Also given in Table 20 are the P-values associated with a goodness-of fit-test for each of the models on the assumption that the weighted residual sums of squares are approximately distributed as chi-square with their corresponding d.f..

Table 19: Goodness-of-fit statistics (weighted residual sums of squares, S^2) for selected models for assortative mating in the US and Australia

Model		Random mating	Phenotypic assortment (P)	P+Error	Spousal Interaction	Social Homogamy
d.f.		16	15	13	14	11
Variable	Sample	S^2	S^2	S^2	S^2	S^2
Stature	US	449.179	31.363	24.423[1]	78.930	28.786
	AU	239.827	12.947	11.817[1]	31.694	25.353
Conservatism	US	2535.373	14.845	12.143	118.266	328.491
	AU	2041.407	31.627	29.669	113.276	239.123
Neuroticism	US	63.371	17.811	See note[2]	20.226	19.458
	AU	28.337	17.444	See note[2]	15.583	22.807
Church attendance	US	3375.872	15.187	12.841	103.042	611.006
	AU	3019.544	22.140	21.548[1]	76.574	403.950
Political affiliation	US	2213.625	22.254	18.500	87.889	429.819
	AU	2337.500	34.183	32.537	70.696	322.685
Educational attainment	US	2477.957	46.210	28.207	243.100	57.774
	AU	1430.440	44.146	18.624	160.747	82.086

Notes: [1]Estimated regression of male outcome on latent trait on upper bound (1.000). [2] This model is poorly identified for Neuroticism because the correlation between mates is close to zero. Stable parameter estimates are not available.

The residual sums of squares are all enormous for models assuming random mating. That is consistent with the results of the models fitting to twins and nuclear families and confirms that the correlations between spouses and pairs of relatives by marriage are all significantly greater than zero on average. Even the residuals for neuroticism are statistically significant under the random mating model, although the spousal correlation is too small to contribute substantially to the overall pattern of family resemblance. All attempts to allow for assortative mating lead to a much better fit than models that assume random mating. However, for none of the variables in Table 20 do simple models of social homogamy or spousal interaction come close to fitting the data, suggesting that either model, by itself, is not adequate to account for spousal resemblance. Between these two models, invoking social homogamy fits much better, though still extremely badly, for conservatism, church attendance and political affiliation and assuming spousal interaction does better, though also badly, for educational attainment. In general, the parameter estimates under the social homogamy model are also strange. The estimated correlation between the social backgrounds of spouses is almost always unity, the only exception being that for educational attainment in the US sample. Furthermore, the residual correlations between DZ twins and siblings are often very small and frequently close to zero while those for MZs, while not large are often more than twice those for DZs and siblings. This would arise if the assumption that assortment was based only on the latent *non-genetic* aspects of family background led to overestimation of the contribution of family background to the pattern of family resemblance.

Table 20: Models for assortative mating, parameter estimates.

Outcome	Conservatism								Church Attendance							
Model	Phenotypic(P)		P+Error		Social Homogamy		Spousal Interaction		Phenotypic(P)		P+Error		Social Homogamy		Spousal Interaction	
Sample	US	AU	US	AU	US	AU	US	AU	US	AU	US	AU	US	AU	US	AU
t_{MZM}	0.645	0.613	0.698	0.655	0.279	0.200	0.840	0.852	0.496	0.610	0.510	0.612	0.252	0.252	0.798	1.000
t_{MZF}	0.637	0.627	0.655	0.658	0.343	0.358	0.793	0.745	0.600	0.614	0.629	0.628	0.284	0.284	0.757	0.847
t_{DZM}	0.411	0.464	0.444	0.487	0.000	0.133	0.541	0.718	0.456	0.575	0.470	0.576	0.000	0.000	0.727	0.958
t_{DZF}	0.498	0.526	0.513	0.554	0.046	0.150	0.604	0.617	0.451	0.575	0.476	0.592	0.018	0.018	0.559	0.768
t_{DZMF}	0.389	0.492	0.411	0.525	0.000	0.000	0.489	0.612	0.288	0.430	0.299	0.436	0.000	0.000	0.413	0.657
t_{SibM}	0.414	0.440	0.462	0.477	0.000	0.000	0.506	0.584	0.335	0.448	0.348	0.448	0.000	0.000	0.508	0.713
t_{SibM}	0.435	0.446	0.450	0.475	0.000	0.000	0.509	0.505	0.374	0.488	0.403	0.505	0.000	0.000	0.442	0.639
t_{SibMF}	0.371	0.402	0.397	0.433	0.000	0.000	0.446	0.493	0.376	0.462	0.398	0.434	0.000	0.000	0.496	0.616
μ	0.657	0.677	0.701	0.721	1.000	1.000	0.000	0.000	0.765	0.819	0.807	0.831	1.000	1.000	0.000	0.000
r_M	1.000	1.000	0.942	0.956	0.745	0.730	1.000	1.000	1.000	1.000	0.978	1.000	0.739	0.739	1.000	1.000
r_F	1.000	1.000	0.982	0.966	0.738	0.740	1.000	1.000	1.000	1.000	0.956	0.980	0.721	0.721	1.000	1.000
$b_{M->F}$	0.000	0.000	0.000	0.000	0.000	0.000	0.371	0.326	0.000	0.000	0.000	0.000	0.000	0.000	0.397	0.527
$b_{F->M}$	0.000	0.000	0.000	0.000	0.000	0.000	0.454	0.549	0.000	0.000	0.000	0.000	0.000	0.000	0.650	0.737
P(%)	46.3	0.7	51.6	0.5	<0.1	<0.1	<0.1	<0.1	43.8	10.4	46.0	6.3	<0.1	<0.1	<0.1	<0.1
Outcome	Political Affiliation								Educational Attainment							
Model	Phenotypic(P)		P+Error		Social Homogamy		Spousal Interaction		Phenotypic(P)		P+Error		Social Homogamy		Spousal Interaction	
Sample	US	AU	US	AU	US	AU	US	AU	US	AU	US	AU	US	AU	US	AU
t_{MZM}	0.516	0.715	0.589	0.755	0.094	0.215	0.631	1.000	0.850	0.720	0.922	0.880	0.668	0.548	1.000	0.960
t_{MZF}	0.493	0.638	0.496	0.640	0.208	0.278	0.649	1.000	0.816	0.801	0.959	1.032	0.583	0.606	0.949	0.839
t_{DZM}	0.494	0.807	0.571	0.852	0.000	0.462	0.591	1.000	0.628	0.536	0.682	0.665	0.061	0.154	0.815	0.673
t_{DZF}	0.440	0.433	0.442	0.435	0.083	0.000	0.559	0.727	0.694	0.526	0.821	0.680	0.311	0.197	0.801	0.552
t_{DZMF}	0.377	0.587	0.405	0.605	0.000	0.000	0.476	0.873	0.600	0.501	0.684	0.641	0.013	0.101	0.722	0.580
t_{SibM}	0.352	0.538	0.420	0.580	0.000	0.000	0.419	0.737	0.568	0.418	0.624	0.528	0.000	0.008	0.696	0.528
t_{SibM}	0.280	0.477	0.280	0.478	0.000	0.000	0.345	0.742	0.608	0.493	0.751	0.656	0.082	0.135	0.669	0.520
t_{SibMF}	0.315	0.471	0.348	0.489	0.000	0.000	0.370	0.690	0.585	0.374	0.682	0.489	0.000	0.000	0.675	0.420
μ	0.653	0.824	0.717	0.854	1.000	1.000	0.000	0.000	0.595	0.553	0.664	0.673	0.869	1.000	0.000	0.000
r_M	1.000	1.000	0.894	0.957	0.653	0.761	1.000	1.000	1.000	1.000	0.952	0.882	0.770	0.638	1.000	1.000
r_F	1.000	1.000	1.000	1.000	0.621	0.738	1.000	1.000	1.000	1.000	0.894	0.857	0.741	0.637	1.000	1.000
$b_{M->F}$	0.000	0.000	0.000	0.000	0.000	0.000	0.420	0.703	0.000	0.000	0.000	0.000	0.000	0.000	0.304	0.153
$b_{F->M}$	0.000	0.000	0.000	0.000	0.000	0.000	0.376	0.577	0.000	0.000	0.000	0.000	0.000	0.000	0.443	0.504
P(%)	10.1	0.3	13.9	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	1.2	13.5	<0.1	<0.1	<0.1	<0.1

Note: Estimates are not given for stature and neuroticism. Estimates for random mating models are omitted from table. In the case of the social homogamy model the twin and sibling correlations refer to the correlations between residuals (I.e., *u* in Figure 8). Otherwise, the correlations are those between twins and siblings for the manifest or latent variable on which mate selection is based.

Overall, models that assume phenotypic assortment perform much better, with or without the inclusion of uncorrelated errors of measurement. Assuming that the residuals are distributed as chi-square, the simple model of phenotypic assortment gives an adequate, even "good", fit to conservatism, church attendance and political affiliation in the US sample, but it is necessary to invoke error components to give a tolerable fit to the Virginia data on educational attainment. Even when allowance is made for errors of measurement in the model for phenotypic assortment, the paths from "true" score to phenotype are typically large suggesting that the measures used provide a good approximation to the traits on which assortment is based. Although, in Australia, models of phenotypic assortative mating give much better fit than those for social homogamy and spousal interaction for all the variables discussed, the fit is still relatively poor for conservatism and political affiliation. Further analysis, perhaps involving elements of more than one model ("mixed homogamy", Heath et al., 1985) would be needed to see if the fit could be improved further.

The above analyses of the resemblance between spouses, twins and in-laws, suggest that phenotypic assortment, perhaps with some allowance for random residual effects gives a much better account of the correlations between spouses and in-laws than models that assume spousal resemblance results only from their mutual interaction or stratification of the pool of mates by features of the family background. We do not claim that this is a definitive analysis, but it suggests how the study of extended kinships can elucidate the principal features of human mate selection and gives some justification for the assumption of phenotypic assortment in our previous analysis of biological and cultural inheritance.

Development of Social and Political Attitudes in Childhood

All the above analyses deal with differences between *adults*. By the time these studies have begun, the subjects have already passed through two decades or more of biological development and social learning – from their parents, teachers, peers and media. Infants do not appear with measurable social attitudes. Their world and its perception matures and expands as they mature. The world itself changes over time. Contexts and salience change with age. Thus, when we consider differences in behavior, especially differences in social attitudes and behavior we expect change. Such changes may not merely affect how behavior itself changes over time with age, the so-called "main effects" of age and secular change, but the actual *causes* of individual differences may change with age and environment. For example, different kinds of environment may be important to different times in the life cycle, different genes may be expressed at different ages or in different social contexts. Thus, it is important to look at the role of social and genetic influences on political attitudes from a life course perspective.

Political socialization research has traditionally focused on how elements of the social environment influence and interact with individual development (Sigel 1989). Within the social environment, familial, and especially parental, influences have long been considered a primary source of political learning over the life course. This focus on the parent seemed well-founded as numerous studies have found a strong correspondence between ideological orientations of parents and their children (e.g., Alwin, Cohen and Newcomb 1991; Jencks et al. 1972; Jennings and Niemi 1982; Miller and Glass 1989). The need to take a developmental perspective extends as much to the study of genetic differences as it does to the effects of socialization. Although the genes may be fixed at conception, the effects of genes are not but emerge as part of an ontogenetic process that extends across the whole of life, with different patterns of expression and regulation arising in response to the adaptive challenges at different times in the life span and historical setting. Eaves et al. (2008) found that the genetic and environmental contributions of religious practices and church attendance (religiosity) vary with age. In principle, genetic differences expressed at birth may be eradicated by the cumulative effect of the post natal environment or they may be imperceptible early on and increase over the lifespan as a result of constant environmental reinforcement (e.g., Eaves, Long and Heath 1986). Shared environmental factors accounted for the vast majority of

variance in children and adolescents, but decreased in importance during late adolescence and young adulthood, while genetic influences on religiosity increase over the same period. This is important as political ideology is believed to stem from a socialization process similar to that affecting religion in that both share the same high levels of parent-child concordance (Niemi and Jennings 1991).

We demonstrate the importance of a more dynamic conception of the roles of genes and environment in social behavior by examining data from published population-based twin surveys that address both the pattern and sources of political attitudes over the life course (see Hatemi et al., 2009 for details). We combine two studies of twins. The first examines cross-sectional age changes in adult MZ and DZ twin correlations from the Virginia 30,000 (see Eaves et al., 1997 for more details). The second employs longitudinal data on the attitudes of twins gathered as part of a study of cardiovascular risk and function through adolescence.

Figure 10 summarizes the correlations for conservatism between MZ and DZ twins in twins from the Virginia 30,000, by age divided into 5 year cohorts between 18 and 75+. The individual correlations fluctuate somewhat with age, but the principal feature of the data is that the correlations for MZ twins are roughly the same across ages and virtually always greater than those for DZs. A simple statistical test of these correlations (see Eaves et al, 1997) shows that variation over ages is largely due to sampling error, while that between MZ and DZ correlations is not. That is, there is consistent evidence for the role of genetic effects on social attitudes in adulthood that does not alter in magnitude through adult life.

Figure 10: Twin correlations for conservatism in twins aged 18+

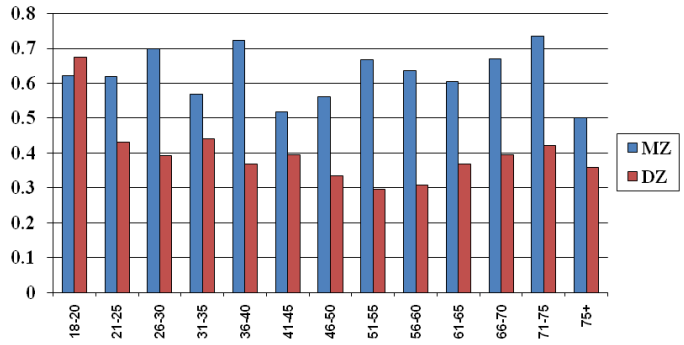
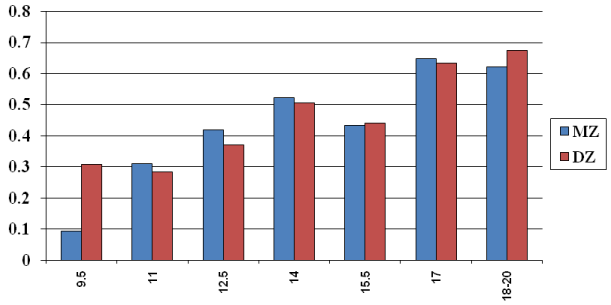


Figure 11 shows the correlations for a measure of conservatism obtained in a study of juveniles. Note that we have deliberately included twins aged 18-20 in both figures.

Figure 11: Twin correlations for conservatism in juvenile twins aged 9-18.



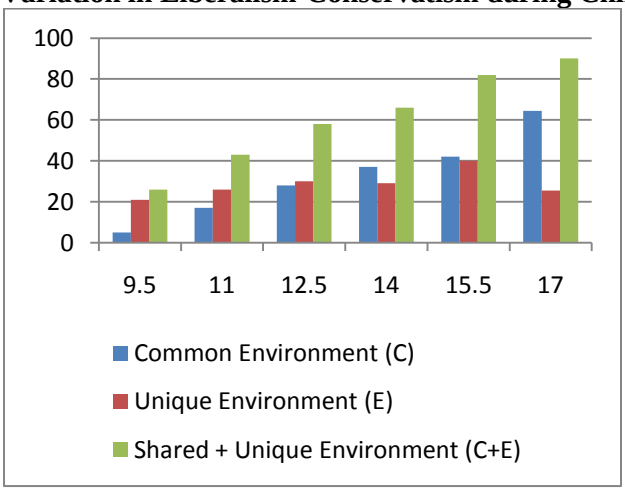
The results for juveniles stand in marked contrast to those for adults. In juveniles, the correlations visibly increase with age during adolescence. Furthermore, and very strikingly, there is virtually no difference

between the correlations for MZ and DZ twins. The increase in correlation with age and the lack of any significant difference between MZ and DZ correlations in adolescence are both confirmed by a simple statistical test (Eaves et al., 1997). The analysis of the age trends in correlation suggests a remarkable developmental transition in the contributions of genes and environment across the life-span. Prior to age 20, the pattern is one of purely social determination with no significant hint of genetic effects. After that age, the picture changes with emergence of the marked genetic differences that persist through the rest of adult life. It appears that ages 18-25 reflect a pivotal developmental transition in the contributions of the social environment to social attitudes. Prior to (say) age 21, the overwhelming effects are those of the environment shared by family members. In so far as these effects depend on parents, they are largely shared and social and not detectably genetic in any form. After age 20, the pattern changes. The abiding effects of parents on their adult offspring seem to be genetic rather than social (exaggerated by the effects of strong assortative mating for social attitudes, see above). As a result, the effects of Mendelian segregation are finally expressed in the adult phenotype, resulting in a sharp and marked reduction in the DZ correlations relative to those for MZs because genes do not only contribute to the resemblance of parents and offspring but also to differences within pairs of dizygotic twins and siblings.

The other impressive feature of the correlations in Figure 11 is the consistent increase in twin correlation with age, suggesting that the relative effects of the shared environment on twins' attitudes increase with age. In fact, the figure obscures the fact that the increase in the effects of the shared environment is even more marked. The correlations are standardized within each age cohort, so the figure only reflects the contribution of shared influences *relative to the total at each age.* Figure 12 summarizes estimates of the absolute amount of variation in conservatism contributed by shared and unique environmental influences across ages.

The figure shows that the *total* variance in conservatism increases almost four-fold between ages 9 and 17. This is not surprising as the items concern issues that are barely salient to younger children and responses likely to be far less structured and organized as they are in older juveniles who are becoming aware of, and influenced by, their social and poltical universe. The variance contributed by the unique environment (E) fluctuates around the same level (20 units) across the whole age range or shows, at best, only modest accumulation between 9½ and 17. This is consistent with the view that much of what contributes to E is due to relatively short-lived random fluctuation that contribute typically to measurement error. The effects of the shared environment, in contrast, increase from close to zero in the youngest twins to about 60 units in 17 year-olds.

Figure 12. The Relative Contributions of Shared and Unique Environmental Influences on Variation in Liberalism-Conservatism during Childhood and Adolescence.



Source: Hatemi et al., 2009.

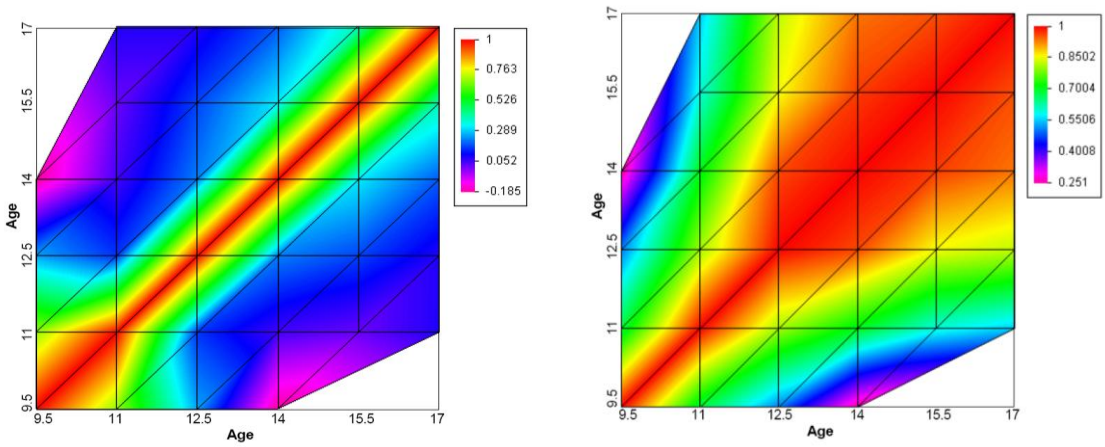
It is tempting to conclude that the effects of the shared environment at any stage during adolescence represent the persistent and cumulative effects of previous environmental experiences. Obviously, this hypothesis cannot be tested with cross-sectional data. However, for this adolescent cohort, we are fortunate that a large part of the sample was studied longitudinally so it is possible to reconstruct how the effects of the shared and unique environment persist and accumulate over time (See Hatemi et al, 2009). The logic is simple. Just as we can use cross-sectional data on MZ and DZ twins to estimate the contributions of genes and environment to individual differences *within* ages, so we can use the cross-twin correlations *between* ages to estimate the conttibution of genes and environment to stability of behavior *between* ages. For example, suppose genetic effects persist over time but the effects of the shared environment are age-specific. Cross-sectional twin data would show that the DZ correlations, while less than the MZ correlations are nevertheless greater than half the MZ correlations (see the above introduction to the analysis of twin resemblance). If the effects of the shared environment are age-specific, however, they will not contribute to the correlation between first twins measured at one age and second twins measured at another. So, for example, in our thought experiment, if genetic effects are stable over time, the correlation between first twins at one age and second twins at another in MZ twins is expected to be twice that for DZ twins. Indeed, we can use the cross-twin cross-age correlations to estimate the extent to which the same genes (or environments) contribute to individual differences at different ages. That is we can estimate the *genetic correlation* between measures made at different times, an index of the extent to which the same or different genes are expressed at differents ages. Generally, a high genetic correlation implies that a high proportion of the genes affecting a trait are showing effects that are stable over time and age. The same logic can be applied to estimate the *shared environmental correlation* and the *unique environmental correlation* from longitudinal data on MZ and DZ twins. This basic insight allows a variety of models to be elaborated for the effects of genes and environment on development (see Hatemi et al. 2009 and references in Eaves et al., 2005) and for developmental changes in the effects of genes and environment. Figure 13 shows "heat maps" of the cross-age correlations for the effects of the unique (Figure 13a) and shared (Figire 13b) environment on adolescent liberal-conservative attitudes.

High correlations (close to 1) are shown in red and low correlations (close to zero) in blue. The diagonal (from lower left to top right) represents the correlations between effects for twins of the same age. Obviously these are all unity for both unique and shared environmental effects. The color shades from red to blue as the graph moves from the leading diagonal. As the age differences between the stages of measurement get greater, the correlations get smaller because somewhat different environmental effects operate at different ages. However, the difference between the graphs for the unique and shared environmental effects is very marked. The correlations between the effects of the unique environment fall off very sharply with increasing age difference, rapidly approaching zero. Furthermore, there is very little tendency for the correlations to change as a function of increasing age. Thus, the individual-specific effects of the environment, not shared with cotwins, are relatively transient. They neither persist nor accumulate with age.

The pattern for the shared environment differs greatly. Firstly, the correlations do not decay anything like as rapidly as those for the unique environment, supporting the view that the effects of the shared environment – parents, teachers, peers and media – are far more persistent than those which make individual twins differ within the family. Second, as twins get older, the correlations between measures at different ages also get larger.

Figure 13 Cross-age Correlations Between Environmental Effects on Conservative Atttitudes during Childhood and Adolescence (Source Hatemi et al. 2009).

a. Unique (within-family) environment b. Shared (between-family) environment

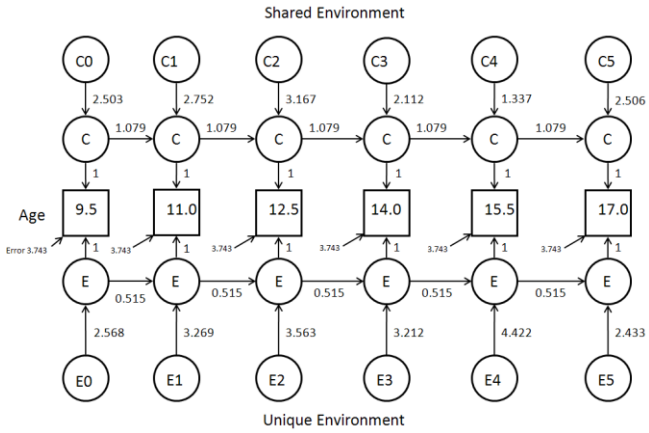


This can be seen in the way the band of red and orange in Figure 13b spreads out more from the diagonal as the twins approach 17. This finding is consistent with the view that the effects of the shared environment not only persist over time, they also accumulate. Each new occasion may expose each pair to a new set of shared environmental experiences, but these persist throughout adolescence and new shared environmental experiences build up these, leading to the pattern we observe of increasing effects of the shared environment with age (Figure 12), and high and increasing cross-temporal correlations in the shared environmental components (Figure 13b). By contrast the effects of the unique environment show much less dramatic changes over time (Figure 12) and are much less stable and accumulate less with age (Figure 13a).

The Figures are suggestive of a developmental model for social attitudes in which influences of the unique environment are time-specific and short-lived whereas those of the shared environment show continuous, persistent and cumulative input from before adolescence into early adulthood. These basic ideas can be captured and tested more formally in a variety of linear time series models that specify the input and transmission of environmental influences over time. Hatemi et al. develop and test such a model for the longitudinal twin data we have summarized. Their "best" model, in the sense of that which yields good overall fit without over-elaborating model complexity, is summarized in Figure 14.

Squares represent the measured trait (conservatism-liberalism) for each age listed. Circles represent latent "input" varibles. E is the unique environment. C is the shared or common environment. There are assumed to be specific environmental inputs at each age that are independent of each other over time (E0..E5; C0-C5). E0..E5 and C0..C5 are assumed to be scaled to unit variance. These effects, however, accumulate through the mediating effects of variables E and C respectively. The paths between successive values of E and C represent the strength of the forward persistence of previous environmental influences. These are allowed to differ between E and C, but are assumed, for simplicity, to be constant over time.

Figure 14: First-order Autoregressive Model for the Effects of the Shared and Unique Environment on the Liberalism-Conservatism Index during Childhood and Adolescence



Source: Hatemi and Eaves, 2009

There are assumed to be occasion-specific, individual-specific errors of measurement that do not persist. These are repesented by the arrows labelled "error" in the figure. Hatemi et al. present the results for other versions of the model. The figure presents maximum likelihood estimates of the parameters of their final model for the covariance structure. If anything, the time-specific effects of the unique environments, E0…E5, are somewhat larger than those of the shared environment, C0…C5. Thus, at any one time the unique environments are highly variable, perhaps more so than the shared environments. However, the persistence of the unique environment across time (0.515) is far less that that of the shared enviroment (1.079). That is, the effects of the shared environment, once they occur, do not decay over time but persist to the threshold of adult life whereas unique environmental experiences are soon forgotten. Details of the model-fitting process and model-comparison statistics are summarized by Hatemi et al.

These compelling findings for the development of social attitudes during adolescence also present an abiding problem. Why is it that effects that persist and accumulate so robustly during adolescence are so rapidly dissipated in favor of genetic influences once the young adults leave home? Is our finding for conservatism the footprint of changing roles of social and genetic family influences in the transition from parent-dependent adolescence to adult independence? Late adolescence is seen as the formative phase for establishing social orientations (Inglehart 1977). Our data imply a much more nuanced process where social *orientations* are not established in this period, but social *learning processes* are. The latter comprise the mainspring for the subsequent development of social *orientations*. Only when children leave home do their own longer term orientations manifest themselves. What children may be gaining from their parents is a default heuristic; a process of heuristic acquisition in adolescence which then changes to heuristic application in adulthood, thereby explaining the significant correlation between parents and children but also allowing for idiosyncratic deviations. Thus, offspring begin with the attitudes learned from their parents, but as they leave home, their own experiences and individual genetic disposition interact to modify those attitudes. The underlying mechanisms behind this remarkable transition over a relatively short part of the lifespan remains unclear.

We note in passing that our data on adolescent and adult attitudes present a problem for simple-minded versions of the frequent criticism that MZ twins resemble one another more than DZs because they share more similar patterns of early socialization, with parents and others tending to emphasize the similarities of MZs and the differences in DZs. Proponents of this view have to develop a model that explains why it takes until the twins leave home for these early differences in socialization to occur, yet at the very time they are living together, going to school and playing with friends there is not the slightest hint, at least for the variables in question, that MZ twins are in the least bit more similar than DZs.

Limitations

At a conceptual level, the model we employ has a number of limitations that require analysis of further sets of relationships and variables. The model ignores genotype x environment interaction, GxE, that is a known feature of genetic systems in other species (see above). Some credible examples of GxE have been published in humans although such studies require very large samples stratified by environmental (or genetic) risk factors. These studies have still to be undertaken with the US and Australian samples with respect to political and social attitudes and behavior. The extended twin kinship design yields far more correlations between relatives than we chose to illustrate the essentials of the model. Altogether, the studies yield no less than 80 distinct relationships (ignoring three-generational data). These additional relationships not only allow the current model to be tested for its ability to account for a much broader set of kinship relationships but also allow for the development and testing of a richer set of alternatives including different mechanisms of mate selection and intergenerational transmission (see e.g. Truett et al., 1994; Keller et al., 2009; Maes et al., 2009). The Australian and Virginia samples comprise a far richer set of relationships than those we have chosen to analyze for didactic and illustrative purposes (see e.g. Eaves et al., 1999; Kirk et al., 1999; Lake et al., 2000). These additional correlations not only generate more data points that provide broader-ranging tests of the generalizability of simpler models but permit a wider range of models, involving greater numbers of parameters, to be fitted and tested (see e.g. Keller et al., 2009; Maes et al., 2009).

Critics (see Exchange in *Perspectives on Politics* June 2008, October 2008) have argued that the reliance of such estimates of genetic contributions on patterns of twin resemblance which is inherently flawed due to the equal environments assumption (EEA), i.e that MZ twins are typically treated more alike than DZs as children (e.g. Loehlin and Nichols, 1976) and have more contact with each other as adults (e.g. Kendler et al. 1993). This assumption has been tested for numerous behavioral traits in every fashion the data would allow (for a review see Medland and Hatemi 2009; Hatemi et al 2009a). For example, Posner et al. (1996) showed that the degree of environmental sharing in adult Australian twins is better explained empirically as a consequence rather than a cause of phenotypic differences within MZ and DZ pairs. The analysis of adolescent attitudes discussed above (see Hatemi et al. 2009) have demonstrated that the differences between MZ and DZ correlations for conservatism arise only in adult life and that MZ and DZ correlations are identical during adolescence. These data preclude the obvious implication that additional similarities in the juvenile MZ environment are having a substantial effect on the development of attitudes. Typically, critics of the EEA have yet to provide a theory of how greater early environmental differences within DZ twin pairs compared with MZs have latent effects that only affect the outcome of interest when the twins leave home. It could still be argued that the persistence of greater MZ similarity in adult attitudes could reflect the abiding greater attachment of MZ twins compared with DZs. The implications of this possibility for adult political and social behavior remain to be explored empirically with the current data. For example, it remains to be explained how the effects of the "MZ special environment" metastasize through entire pedigrees, such as the extended kinships of twins including such a wide range of social and biological relationships as cousins related through MZ and DZ twins, the in-laws of MZ and DZ twins and so forth. That is, if the "special MZ environment" or lasting MZ attachment, is a real component of their behavior, then it has to be such that it pervades virtually every feature of adult life, including marriage, mating and parenting. Even if such explanations were plausible, in the absence of concrete proposals about their broader implications for family resemblance, they remain an *ad hoc* refuge of despair in the face of more theoretically-grounded genetic models and have the same heuristic value as theories of intelligent design in evolutionary biology (Charney 2008). The general consistency of twin data with the growing array of data confirming the contributions of large numbers of identifiable genetic polymorphisms to behavioral outcomes is a further strand in the web of coherent support (c.f. Urbach, 1977; Murphy, 1997) for the view that parsimonious statistical genetic models, grounded in a wide interwoven history of experimental and observational study cannot be dismissed so summarily because the conclusions appear counter to our personal preferences (c.f. Russell; 1946, p.).

Conclusions and Future Directions

In this chapter we provide a step by step account of formulating, fitting and testing a series of competing and complimentary models for the causes and familial transmission of individual differences in six key domains of human behavior and attributes: group affiliation ("Party ID"), ideology (Conservative-Liberal attitudes), religiosity (Church Attendance), education (Educational Attainment), personality (Neuroticism) and physical appearance (stature).

We illustrate the approach with two of the largest and potentially most informative datasets available to us, using different constellations of relatives separately and cumulatively: nuclear families and twins. Within limits, the more subtle the models we wish to identify, the greater the range of relationships we need to study. We contrasted, and then combined, models of non-genetic transmission and models that include additive genetic transmission. Our dataset, which is the largest of any genetically-informative study on socio-political attributes, supports a model which includes genetic transmission for all six traits analyzed except for party identification. Thus, our data from Australia and the US concur in showing, to a first approximation, that individual differences in party affiliation are due entirely to social and unique environmental influences without any apparent genetic influence. This finding sets party affiliation, along with religious affiliation (see e.g. Eaves et al., 1990), apart from the majority of traits studied from a genetic perspective almost all of which show at least some contribution of genetic differences. Educational attainment, church attendance, conservatism-liberalism and party affiliation are all remarkable for the very high correlations between spouses. In this respect, these variables are also distinct from almost all other physical and behavioral traits, including neuroticism and stature for which the correlations between mates are far smaller if not close to zero. When our models for family resemblance include assortative mating it becomes clear that much of what is assigned to the effects of the shared environment in twin studies may well be a reflection of the genetic consequences of assortative mating The choice of mate as one of the most significant ways through which humans extend their influence to the next generation.

The actual process that generates spousal resemblance in these initial analyses is moot. It was assumed without test in the analysis of the twin and nuclear family data that the correlation between mates was a matter of mutual selection of each spouse for the measured phenotype of the other. A series of follow-up analyses exploit the additional information in the patterns of resemblance between the spouses of biological relatives (twins and siblings, in our case) to begin to test alternative hypotheses about the process of mate selection and to discriminate similarity due to assortative mating from that due to spousal interaction. For those variables where the correlations between mates are large, analysis of the correlations between the spouses of twins and siblings strongly supports the assumption of the initial model-fitting that the principal source of spousal resemblance is indeed the selection of spouses for the actual phenotypes in question and not for aspects of the family background that helped shape them ("social homogamy") or simply due to the mutual social interaction that is expected to occur between spouses.

The Virginia 30,000 data and the Australian 20,000 comprise samples that are entirely adult and cross-sectional. Thus, the analyses of these samples only consider the long-term outcome of development. They do not attempt to analyze the ontogenetic process by which attitudes emerge during adolescence. We have summarized the findings of a recent analysis by Hatemi et al. (2009b) in which the development of liberal and conservative attitudes was studied in a longitudinal sample of MZ and DZ twins assessed repeatedly every 18 months between 9.5 and 17 years of age. The results of this analysis are remarkable in showing that, prior to young adulthood, the influences shaping conservative and liberal social attitudes are exclusively environmental and notably the environments that twins share regardless of zygosity. That is, the genetic effects on attitudes expressed in adulthood only emerge in the transition from dependence on parents to relatively independent adult life. During adolescence, the effects of the shared environment are highly persistent and cumulative, whereas those of the individual within-family environment are short-lived and show little, if any, long-term accumulation. A model that elucidates the actual mechanism

underlying this apparent "switch" from purely social to partly genetic causation of differences in attitudes will require the careful longitudinal study of a genetically informative sample during this period of transition. Unfortunately, such data are not available to us at this time.

The results of these analyses, based on large sample of informative relatives in two populations addressing a common set of constructs suggest strongly that the theory of the "Promethean genotype" is not a solid foundation for understanding why people vary in their social and political behavior. Evolution has not entirely emancipated humans from the influences of their biological inheritance. This being said, the results imply a far more subtle understanding than the simple-minded assumption of pure genetic or social inheritance. Although social attitudes and political behavior could not exist without culture, individual differences in the cultural options to which individuals migrate is, at least in part, a function of inherited genetic differences (c.f. Martin et al., 1986).

Some may be concerned that our approach raises the specter of genetic determinism. There is no practical or epistemological difference between genetic and environmental accounts of human behavior. Both are inherently determinist because they address "cause." Neither precludes nor necessarily facilitates behavioral change or intervention. Neither precludes human agency as a potent factor in individual or societal transformation. Neither is fixed. The real scientific challenge is to reveal what it is about the phylogeny and ontogeny of human behavior that makes humans capable of transcending perceived biological or social limitations.

The conclusion that genetic differences are a significant component underlying variation in some socially and politically important dimensions of human behavior raises the question of the number and genomic location of these heritable differences. Any demonstration that human differences are partly genetic in origin definitely does not imply that there is a single gene, or even a small handful of genes "for" specific features of complex human behavior. In this respect we concur with Lewontin et al. (1984). Genetic research in the 90 years since Fisher's (1918) seminal paper, and especially in the last decade, appears to be reaching a consensus that, after analyzing variation throughout the genome on an extraordinarily large number of subjects for a wide range of physical, physiological and clinical outcomes, the number of genes is very large and their individual effects sometimes vanishingly small (e.g Visscher, 2008; International Schizophrenia Genetics Consortium, 2009). Research strategies which recapitulate failed attempts to find individual genes of large effect on other traits are, in the opinion of these authors, unlikely to unlock the underlying biological mechanisms that bridge the developmental gap between the DNA and outcomes of concern to the social or political sciences. Indeed, those who incline towards non-genetic explanations may well agree that the same is true for environmental predictors. The strongest correlations are the empirical correlations between relatives and their resolution into specific material causes remains elusive. Classical twin and family designs provide population estimates of the genetic and environmental components of certain behaviors or attitudes, but they cannot explain why a specific individual has a certain attitude.

How should findings about familial, environmental and genetic sources of variation be interpreted and incorporated into the research program of political science? The short answer may be "in fear and trembling" because it is very easy to overstate and misrepresent the practical and cultural importance of any scientific conclusion. This being said, the data on family resemblance offer a pressing invitation to address genetic and biological mechanisms underlying some, but not all, aspects of political behavior. Models that incorporate only presumed "environmental" influences may miss one of the most significant factors underlying human motivation and behavioral development. *Individual* differences are paramount in individual behavior and the origin of individual differences is, apparently, not purely social. Political scientists continue to debate whether political preferences and behaviors are matters of social influence or only matters of personal conscious and rational choice. The approach discussed in this chapter makes it possible to explore both genetic and social influences within an integrated paradigm that excludes neither

a priori and allows the data to decide where to focus research in particular contexts. If the effects of the common environments shared by members of different family clusters are small, it is unlikely that differences in socialization of clusters within a given target population can account for individual differences. In the absence of joint analysis of political choices and other predictors it is impossible to know whether genetic influences on social attitudes and choices are expressed through genetic differences in the cognitive functions that lead to the conscious evaluation of individual utility, differences in the innate structure of individual preferences, or inherited variation in adaptive non-cognitive emotional functions that bear the long-standing imprint of evolutionary rationality. The application to rational choice models are apparent. Resolution or integration of these three perspectives requires the simultaneous collection and analysis of multivariate kinship data.

Political scientists typically assume that all people are genetically identical and or that such genetic differences as do exist are irrelevant to the social and political domain. Such a sweeping generalization risks may distract political scientists from a critical component of human behavior. Clustering people by complete genetic identity, as in the case of identical twins, results in far greater empirical similarity than is found for almost any other type of clustering by relationship or context, except possibly for clustering by marriage. The growing appreciation of the scope of genetic variation at the molecular level creates a strong *prima facie* case for taking seriously the possibility that such differences may reach into the social and political domains. Humans vary enormously at the genomic level with molecular variants currently characterized at several million specific locations across long tracts of the human genome. What proportion of these variants translate into manifest phenotypic differences is still uncertain. Current estimates suggest that there are some 20-30,000 genes. If individuals are the primary unit of decision and analysis, but vary in ways not captured in any current purely social explanatory model, than any account of their behavior may be seriously deficient. This may be in part, why even the best political models of behavior in political science explain less than 30% of the variation (Matsusaka and Palda 1999). Our studies give credence to the view that the best predictors are familial and, in many cases, significantly genetic.

We are under no illusion that the above findings raise more questions than they answer. The models we describe focus on one variable at a time in terms of latent genetic and environmental influences. They do not begin consider the potential causal pathways between manifest variables, for example between personality or cognitive function and political behavior, that might explain how the latent effects of genes and environment translate into observed differences in political behavior. Statistical-genetic methods and models are available that illustrate how to construct and test multivariate models for genetic and environmental influences (see e.g. Neale and Cardon, 2002) including models that attempt to use genetic information to try to tease apart the nexus of causal relationships between multiple variables (e.g. Heath et al., 1993). In the context of political science, Hatemi et al (2007) introduced multivariate genetic models for voter preferences in Australian and showed that the genetic variance found on voting is largely accounted for by covariates. Verhulst, Hatemi and Eaves (2009) addressed the importance of personality on political ideology. Utilizing statistical models of twins they have found that certain facets of personality and ideology were related and the greater part of the relationship was due to shared genetic influence. We reiterate that significant estimates of genetic variance do not imply that individual genes have large effects, or correspond to specific behavioral outcomes. Political scientists typically explore voter turnout, vote choice, attitudes, and ideology by trying to assess all relevant predictors, pathways and best fitting models. An important potential limitation of the models we discuss is that they are linear and additive. The do not take into account possible interactions between genes ("epistasis") or, perhaps more importantly, interactions between genes and environment. In the domain of social and political behavior it is entirely conceivable, yet to our knowledge currently unknown, whether genetic influences on personality (such as differences in sensitivity to reward and/or punishment) or cognitive function modulate the impact of social background and individual situation on political choice. The only barrier to testing for such interactions remains the availability of appropriate data. Our analyses lend credence to a

view that incorporating genetic information in models for political behavior may generate further insight and improve prediction. Political behavior did not end with the introduction of Campbell et al's (1960) American Voter or Downs' (1957) *An Economic Theory of Democracy*. It just began again. There are numerous others avenues ripe to explore almost every experiment, statistical model, and behavioral trait can be re explored using these methods, allowing researchers to further pursue their trait of interest with a whole new set of tools, hypothesis and designs. We look forward to see what comes next.

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