

Political Science, Biometric Theory, and Twin Studies: A Methodological Introduction

Sarah E. Medland

*Department of Genetic Epidemiology, Queensland Institute of Medical Research,
Brisbane, QLD, Australia*

Peter K. Hatemi

*Department of Political Science, University of Iowa, Iowa City, IA 52242
e-mail: phatemi@gmail.com (corresponding author)*

As political scientists begin to incorporate biological influences as explanatory factors in political behavior, the need to present a methodological road map for utilizing biometric genetic theory and twin data is apparent. The classical twin design (CTD) remains the most popular design for initial examinations of the source of variance among social and political behaviors, and a vast majority of advanced variance components models as well as some molecular analyses are extensions of the CTD. Thus, it is appropriate to begin a series of works with the CTD and its most common variants. The CTD has strong roots in biometrical genetic theory and provides estimates of the correlations between observed traits of monozygotic and dizygotic twins in terms of underlying genetic and environmental influences. The majority of these analyses utilize SEMs of observed covariances for both twin types to assess the relative importance of these “latent” factors.

1 Introduction

Within the political science scholarship, there is an increasing interest in the roles that biological influences, specifically genes, may play in influencing political preferences. As evidenced by the article “Are Political Orientations Genetically Transmitted?,” by Alford, Funk, and Hibbing (2005), researchers are beginning to use genetically informative data and designs to draw conclusions about the magnitude and nature of biological influences on social and political behaviors. To date, studies using genetic designs have been published in a number of high-impact political science journals including the *American Political Science Review* and the *Journal of Politics* (e.g., Fowler, Baker, and Dawes 2008; Hatemi, Medland, and Eaves 2009). Given the growing popularity of studies examining genetic hypotheses, the present review provides a guide to the analysis of data collected from twins and relatives and some of the more popular models and analyses used within the fields of Genetic Epidemiology, Psychiatric Genetics, and Behavioral Genetics. The specific aims of the current paper are to provide a background to the methods used to analyze

Authors' note: We thank Michael Neale for access to the Mx program for structural modeling and Lindon Eaves for his mentorship. We also thank the anonymous reviewers and editor for their thoughtful and insightful comments.

© The Author 2009. Published by Oxford University Press on behalf of the Society for Political Methodology. All rights reserved. For Permissions, please email: journals.permissions@oxfordjournals.org

genetically informative data and the assumptions these methods make and their relative strengths and weaknesses. Moreover, it is hoped that the information provided here will assist scholars in initiating new studies as well as critically evaluating research that utilize these designs. This is particularly important as research using these methods become more frequent within the literature.

1.1 *Why Use Genetically Informative Models?*

It is widely accepted, within the medical, academic, and public domains, that many physical, psychological, and behavioral traits, however indirectly, are the ultimate result of a combination of genetic inheritance and the environment (e.g., Bouchard and McGue 2003; Caspi et al. 2002). It is a common observation that family members largely resemble each other in many social and behavioral traits (e.g., Eaves, Eysenck, and Martin 1989; Happonen et al. 2002; Jansson et al. 2004; Krueger, Markon, and Bouchard 2003). Behavior genetic techniques have developed in an attempt to understand individual differences, that is, to understand why individuals in a population differ from one another (Neale and Cardon 1992). These techniques can be used to test a range of hypotheses with regard to the trait (or traits) being studied, such as:

To what extent are the similarities between family members due to shared genes and the home or community environment?

How important are unique experiences (perceptions and understanding of events) in shaping behaviors or attitudes?

Do environmental and gene effects influence males and females in the same way?

Do environmental and gene effects change over time?

Does the correlation between traits reflect a shared genetic or environmental effect?

Do genes and environments interact for a given trait, do certain genes make one more sensitive to some environments, and do certain environments trigger gene effects?

Which if any genes influence the given trait or behavior under examination?

The most common method for initial exploration of these questions is the classical twin design (CTD), which focuses on the variance rather than the means. The primary objective is to examine to extent to which genetic and environmental factors influence variation around a population mean rather than to what extent the mean is influenced by a specific predictor (or group of predictors). The finding that a genetic effect influences a trait does not provide information regarding the number of genes affecting the trait, the direction of these genetic effects (i.e., whether these effects increase or decrease the mean), or the identity of the genes exerting this influence. Rather, they provide evidence that individual differences are genetically influenced and that it is worthwhile to attempt to identify the genes involved.

1.2 *Data Requirements*

Unlike random sampling, the majority of analytical techniques used in behavior genetic analyses derive their explanatory power from comparing the trait values (or *phenotypes*) of related individuals who differ in the amount of genetic or environmental sharing. Studies in which data are collected from only one type of relative, such as a study that only collects

data from pairs of siblings, are limited in their ability to discriminate the source of the similarity between relatives. However, when data are collected from two or more types of relatives (a *genetically informative sample*), such as siblings and cousins, or identical and nonidentical twins, then it becomes possible to distinguish the extent to which similarity between relatives can be attributed to genetic relatedness or shared social influences.

Generally speaking, the more distinct the biological and environmental relatedness of the relative types sampled, the greater the power to distinguish between the sources of resemblance. Adoption studies, where the similarity of adopted relatives is compared to the similarity of nonadopted relatives, usually pairs of siblings, or mother–child dyads, allow researchers to separate biology from environment and are conceptually attractive as they can provide a great deal of explanatory power. This is especially true of studies in which pairs of identical twins who have been raised apart are compared to pairs of identical twins who have been raised together. However, from a pragmatic perspective, adoption studies, particularly those involving twins, are difficult to conduct due to both the legal and ethical responsibilities to protect the child and mother. Twins reared apart are also increasingly rare due to modern adoption policies. There are also a number of methodological confounds that need to be controlled for, including age of adoption, frequency of contact with biological relatives, time spent in protective custody or state care prior to adoption, adoption by relatives, and selective placement (matching of the infants biological and adoptive environments). All of which may bias an adoption sample.

Twin and kinship designs (raised together) are arguably the most powerful and practical alternative to adoption studies. The twin method derives its explanatory power from the fact that *monozygotic* (MZ) twins develop from a single fertilized ovum, whereas *dizygotic* (DZ) twins arise from two different ova fertilized by different sperm (Hall 2003). Genetically, MZ twins are identical, whereas DZ twins share on average only 50% of their segregating genes, meaning they are genetically no different than ordinary siblings born at different times. Twins raised by the same parents, in the same environment and at the same time, act as controls for the effects of familial socialization and many other common environmental forces. Thus, if a trait were influenced in part by genes, one would expect the co-twin correlation of MZ twin pairs to be higher than that of DZ twin pairs. In the absence of genetic influences, the co-twin correlation of MZ twin pairs should be the same as that of DZ twin pairs (Eaves 1977).

Arguably, the most common study designs used to estimate genetic and environmental sources of variance are the CTD (where data are collected from MZ and DZ twins including opposite-sex twin pairs), and the twin and sibling design (in which data are collected from twins and also from any non-twin siblings) (Posthuma et al. 2003). Of these, the twin and sibling design is superior due to the increased power to detect shared environmental effects (Posthuma and Boomsma 2000) and more robust controls for equal environment and sampling assumptions (described in detail later). Twin designs provide a relatively easy and accessible method for the initial study of genetic influences for a given trait. The techniques have been developed and refined for almost a century and have been utilized by researchers across the world to study almost every clinical trait, behavior, attitude, and illness imaginable.

2 Central Concepts Underlying the Twin Model

Genetic methods differ from most other paradigms through the extended use of family data. Although most research designs sample independent randomly selected individuals, behavior

genetic approaches deliberately sample nonindependent data from related individuals drawn at random from the population. Because of this, the unit of analysis is the family, rather than the individual. Thus, analyses must explicitly model the nonindependence or relatedness present within the data. These analyses are usually undertaken using maximum likelihood structural equation modeling techniques, although other methods such as repeated-measures analysis of variance (ANOVA), weighted least squares, or Bayesian modeling are sometimes used (Sham 1998).

The accepted practice within the genetics community for collection of twin data centers around a population-based approach. Typically, birth, school, or government records are used to ascertain twins and relatives in the population for a given location and time period. In many studies, large-scale registries, (ranging from 30,000 to over 150,000 related individuals) are developed and used to explore a host of medical and psychological traits (e.g., see the Australian Twin Registry). In other cases, a certain age or group is ascertained for a single survey, such as the North Carolina Study on Cardiovascular function, which petitioned the school superintendents and parents of every school in North Carolina (Schieken et al. 1992).

For many political scientists, obtaining twin data may appear to be prohibitive, but this is not the case. Although the data are not typically publicly available and likely remain unknown to most social scientists, the registries, studies, and contact information are publicly available. In an effort to address this very issue, and make twin data widely available to all political researchers, these authors and others have initiated twin data collection for a host of modern political and social behaviors. This effort, funded by the National Science Foundation (NSF, grant number 0721353), has already produced data for 2009, which is to be made publicly available in the near future.

However, although the phenotypes being collected contain rich political traits made for and by political scientists, there are a number of twin registries with current data useful to political scientists. Those who both manage the registries and the data obtained from previous studies are easily contactable and have increasingly been working with political and social scientists (e.g., see Hatemi et al. 2007). By far, the best resource to locate an appropriate data source is the journal *Twin Research and Human Genetics*. Roughly every 4 years, they devote an edition to updating what is known about twin registries across the globe (see volume 5, issue 5 [2002] and volume 9, issue 6 [2006] for a list of registries from the United States, Australia, Belgium, Canada, Denmark, Japan, Sri Lanka, and many other countries).

The collection strategies, number of individuals, description of data, contact information, points of collaboration, and other important information are explained in detail.¹ Due to the depth of preliminary analyses (psychometric, medical, and other) required before using twin data for primary research, an initial manuscript, article, or book chapter is often developed to outline the properties of the data in a given study. This initial piece is referenced for data properties in future works indefinitely. The depth of assumptions testing (detailed below) is often too lengthy to report for every manuscript.

Twin and family data present unique challenges in collection, and most registries rely on the “willingness, motivation, cooperation, and generosity” of the volunteer twins and their family members (Busjahn 2002). In every study, however small or large, there is

¹Each registry maintains their own formal processes to protect the twins from oversampling, and to a greater degree, twin sampling has been performed largely for medical traits (e.g., depression, sleep disorders, skin cancer, etc.). In the past, social and behavioral traits were often a minor consideration.

a participation bias, and this bias is typically stated in the preliminary analyses. However, the more recent population registries in Sri Lanka, Italy, Scandinavia, and Korea are based on universal health care records and a centralized database. These are extremely important as they control for ascertainment bias by allowing for comparison of responders and non-responders.

Twin samples also introduce new practical concerns unfamiliar to political scientists. For instance, although the process for research ethics depends on each individual university, many institutions have never experienced twin data collection, and obtaining approval could become difficult. Fortunately, investigators can utilize the expertise of those university ethics departments familiar with twin data collection, such as Virginia Commonwealth University or the University of Minnesota.

However, for interested researchers, there is little need to wait for the NSF data, or even to begin the process of developing a collaborative relationship with existing registries to collect new data. The National Longitudinal Study of Adolescent Health (Add Health, <http://www.cpc.unc.edu/addhealth>), a nationally representative study of children to adulthood, is available to all researchers for a small fee and contains adult twins and family members, genotyped for several important genes and phenotyped for a vast array of social, political, and behavioral traits of interest to political scientists. The data are currently being used for political research by several political scientists (e.g., see Fowler and Dawes 2008).

The population-based approach diminishes sample biases as much as possible, while providing the means to evaluate related individuals.² Importantly, results from a single population should not be generalized; results for a given study are limited to the population and time sampled. However, in many cases, the same results are found throughout populations all over the world, over long periods of time. Such is the case with personality; combined evidence has led to a generalizable conclusion that personality is influenced by both genetic and environmental factors (H. J. Eysenck and M. W. Eysenck 1985).

The need for replication is paramount but in no way diminishes the value of any single study. Assuming the sample under analysis is not overly biased, it is likely that additional studies will provide converging evidence. This was the case with the findings of Alford et al. on political attitudes. Hatemi (2007) found similar results for genetic influences on political attitudes in a large Australian population-based sample. Yet, additional difficulties arise when considering converging results from different countries. Hatemi's (2007) comparison of the genetic and environmental influences of 11 political attitudes in American ($N = 14,000$) and Australian ($N = 9000$) participants sampled during the same time period provided remarkably similar variance component estimates, but some differences were observed, namely for the Death Penalty and Immigration. However, for most items, including Abortion, Divorce, and Nuclear Power, the population means were substantially different. Thus, it was argued that cultural norms and interpretations affect the direction and population means (e.g., socialism in the United States is equated with communism, whereas socialism in Australia is highly related to Universal Healthcare). In this light, the different cultures set the overall veneer or range of options for each population, but genetic and personal experience accounted for the variance within the given cultures to a very similar degree.

This speaks to the larger issue of using population-based samples. Although it is possible that they introduce mean biases, they are a remarkably robust for variance

²See Martin and Witson (1982) for a review of potential sampling biases in twin studies.

components differences (see Neale et al. 1989 for a test of such differences). At first glance, to the casual observer, it may seem that twins who opt-in are more similar to one another, thus biasing the results. However, in the vast majority of studies, data are collected: (1) by mail or phone survey, (2) from participants regardless of whether their co-twin or sibling has chosen to participate, and (3) in such a way that each member of family is interviewed by a different interviewer who usually has no knowledge of the participation of the other family members or often the twins' zygosity. In addition, if only twins with increased similarity responded to a given survey, regardless of zygosity, the end effect of such a bias would actually reduce the estimates of genetic influences. Fortunately, because data are collected from both complete and incomplete twin pairs, the means and variance of participants from complete pairs can be compared to those from incomplete pairs allowing researchers to address the question of whether twins from complete pairs are more similar to their co-twins (i.e., have a smaller variance) than those from incomplete pairs would be.

2.1 Sources of Variance

The most common approach to modeling twin and family data is known as the *variance components approach* (Neale and Cardon 1992). When analyzing data from relatives, researchers partition the variance into that which is *shared* between relatives and that which is *nonshared* or unique to the individual. This partitioning is analogous to the partitioning of variance (or sum of squares) into between- and within-group effects that occurs when using ANOVA techniques.

Nonshared or unique environmental influences (E) represent differences in trait values between members of a family due to individual differences in their personal experiences (e.g., romantic partners, employment, and peers not shared with their siblings). However, unique environmental influences can also include the effects of differences in the perception or understanding of an event or environment at which other family members were present, such as differences in the perception of a candidate's speech. Measurement error, unless repeated or test-retest measures are collected, is also subsumed within this source of variance.

Shared variance can be further partitioned into that which is due to genetic effects (A) and that which is due to the family or *common* environment (C). Generally when modeling genetic influences in the absence of measured genotypes (i.e., the DNA that was passed to an individual by the individual's parents), one can distinguish two broad classes of gene action, additive (A) and non-additive (D), based on the patterns of covariation between relatives (Eaves 1977). A genetic effect is described as additive if the cumulative gene effect is the sum of the individual effects of all the genes involved (Falconer 1960). This type of gene action leads to a distinctive pattern of covariance between different types of relatives whereby the genetic covariation between DZ twins (or between twins and a non-twin sibling) is half that of MZ twins. That is, if a trait were solely influenced by additive genetic and unique environmental effects, the correlation between DZ twins is expected to be half that of MZ twins.

Conversely, if the cumulative gene effect deviates from the sum of the individual effects of all the genes involved, this deviation is described as nonadditive. Nonadditive gene effects arise from interactions either within a gene (known as *dominance*) or between genes (known as *epistasis*) (Falconer 1960). Covariation due to nonadditive genetic effects depend on a relative inheriting the same copies of the gene and acts to reduce the covariation

between DZ twins and sibling pairs who inherit the same copies of a gene about a quarter of the time as compared to MZ twin pairs who always inherit the same copies of a gene. If a trait were influenced by additive and nonadditive genetic effects as well as unique environmental effects, the correlation between DZ twins would be expected to be less than half that of MZ twins.

Common environmental (C) influences are those shared by family members. Traditional sources of the shared environment include variables such as socioeconomic status, parental influence, religion, and access to education. Common environmental influences also create a distinctive pattern of covariation. It is assumed that the common environment influences a trait to the same extent for both MZ and DZ twins. Thus, common environmental effects increase the similarity of DZ twins and siblings relative to MZ twins.

When working with data limited to twins who were reared together, common environment and dominant genetic effects are confounded and cannot be estimated within the same model. The decision of whether to fit an *ACE* (additive genetic, common environment, and unique environment) or an *ADE* (additive genetic, nonadditive genetic, and unique environment) model is based on a simple heuristic. If $r_{DZ} \geq .5r_{MZ}$, an *ACE* model will usually fit better than an *ADE* model. Conversely, if $r_{DZ} < .5r_{MZ}$, an *ADE* model will usually fit better than an *ACE* model. Nonadditive genetic influence such as genetic dominance is typically detected in highly biological traits such as electrical activity within the brain or tooth formation, but it may be possible to be present in certain social and behavioral traits. Given that dominance is seldom detected for social traits, we used the term *ACE* model; however, the same analytic techniques and limitations apply to *ADE* models.

2.2 Assumptions of the Twin Method

As with any method of analysis, twin studies make certain assumptions regarding the nature of the data and processes underlying observed effects. However, only an abbreviated set of limitations and assumptions are often included in the genetic literature due to their commonality (similar to that of using regression in the social sciences, i.e., few if any modern journal articles include the limitations of regression methods). Many of the assumptions are implicit within the CTD, and when publishing data collected from twins, authors are expected to consider each of these assumptions in preliminary analyses. It is important to recognize that if an assumption is violated, it does not negate the use of the twin design, family models, or biometric methods in general. Rather, in the case where an assumption is violated, researchers must describe how they tested for the violation and explain the methodological extension used to explicitly model and correct for the violation, akin to something as simple as normalizing data. The general assumptions are described in detail below.

2.2.1 Zygosity testing

Arguably, the best test of zygosity is provided by genotype data. Genetic studies commonly use panels of high polymorphic markers, similar to that used in forensic DNA testing. Blood provides the most easily processed and highest quality DNA sample, but saliva samples are also used. However, more common in twin samples is a series of parent and self-report zygosity questions which have been shown to establish zygosity with above 95% accuracy in comparison to blood typing (Ooki et al. 1990).

2.2.2 Distributional assumptions

A key assumption used to model twin and family data is that the distribution of a trait at a family level will be multivariate normal (Neale and Cardon 1992). This can be verified by examining each family's contribution to the overall fit of the model and the influence of each family's values using a metric such as the Mahalanobis distance and removing or modeling extreme outliers, as is standard practice in genetics modeling. The maximum likelihood approach is robust in the face of minor deviations from normality, but if the data are seriously skewed or kurtotic transformation is suggested, significance tests can be obtained via permutation.

2.2.3 Homogeneity assumptions

Genetic models of twin and family members assume that there are no differences in the means and variances between the different groups in the analysis (i.e., MZ, DZ, siblings, etc.). A similar assumption is made regarding the prevalence when analyzing ordinal data. If such differences exist and are not accounted for, the results of the analysis may be biased. Fortunately, it is easy to customize a model to account for these differences (Neale and Cardon 1992). Prior to fitting models to the data, it is common practice to run a series of analyses, known as *assumption checking*, regarding the homogeneity of means and variances or prevalences within the data.

2.2.4 Twin/singleton differences

It is important to consider whether twins may differ from singletons in a systematic manner for the trait under analysis. The most common test of this assumption is to compare the trait values of twins to their non-twin siblings who are matched for social background and family influences after modeling age and sex effects. Although twins do differ from singletons for some traits, especially those relating to prenatal growth, most studies in general do not find differences in studies of personality and social traits (Evans, Gillespie, and Martin 2002).

2.2.5 Equal environments

Broadly speaking, it is assumed that the contribution of familial or shared environmental influences will not differ between siblings with respect to the trait under analysis. More specifically, it is assumed the magnitude of shared environmental influences on the trait under analysis will not be influenced by zygosity. That is, the shared environment is hypothesized to influence the trait in the same way and to the same extent for MZ and DZ twins. The central concern is that the influence of the shared environment may be larger for MZ than DZ twins or alternatively that there is an additional shared environment specific to MZ twins. If this were the case and the model did not accommodate this difference, the proportion of variance due to genetic and environmental influences would be biased and the genetic variance of the trait would be overestimated, whereas the common environment would be underestimated.

There is little argument that MZ twins are treated more similarly than DZ twins in certain aspects. As young children, they more often share a bedroom and are dressed alike; they are more likely to share common friends and stay in closer contact once they leave home (Cohen et al. 1973; Kendler et al. 1987; Loehlin and Nichols 1976). However, the central questions of the equal environment assumption (EEA) are whether these differences

influence the specific trait under analysis and if these environmental differences are manifestations of the genetic similarity of MZ twins.

Several methods have been used to test the EEA, including (1) correlating perceived similarity with the trait while controlling for actual zygosity (Kendler 1983; Matheny et al. 1976; Plomin, Willerman, and Loehlin 1976; Scarr and Carter-Saltzman 1979); (2) direct observation of family members and others to examine their self-initiated and twin-initiated behaviors toward the different twin types (Lytton 1977); (3) correlating the similarity of the twin environments with the trait while controlling for actual zygosity (Heath, Jardine and Martin 1989; Kendler et al. 1987; Martin et al. 1986); (4) extending the classical ACE model by further partitioning the common environment into the usual common environment, C_{residual} , which is completely correlated for all twin pairs and that which is influenced by the perceived zygosity, C_{specific} , which is parameterized to be completely correlated if both twins perceive themselves to be MZ, completely uncorrelated if both twins perceive themselves to be DZ and correlated at .5 if the twins disagree about their perceived zygosity (Hettema, Neale, and Kendler 1995; Kendler et al. 1993; Scarr and Carter-Saltzman 1979; Xian et al. 2000). This model may also be used to include specific measures for the common environment other than zygosity; (5) when data have been collected from non-twin siblings checking for differences between the DZ covariance and the twin-sibling and sibling-sibling covariances can provide an additional test of the EEA. Arguably, if the more similar treatment of MZ twins were affecting their trait values, one might also expect more similar treatment of DZ twins as compared to regular siblings; (6) when using ordinal data, equality of the thresholds of MZ and DZ twins indicate no differences in variances between MZ and DZ twin pairs, excluding the possibility of an extraenvironmental influence specific to MZ twins. Although MZ and DZ differences have been found for traits such as birth weight and similar dress, no EEA violations have been reported for intelligence, personality, or political preferences.

The most recent method to remove equal environments biases allows heritability to be estimated from non-twin siblings. In summary, rather than assuming that DZ twins or full siblings share of average 50% of their segregating genes, Visscher et al. (2006) used molecular data to get exact measures of genetic sharing of sibling pairs (which in a sample of 4401 sibling pairs ranged from 37% to 61%). This small variation in genetic similarity between siblings was sufficient to allow Visscher et al. to estimate the heritability of height in this sample. Notably, the heritability estimate was very similar to that derived from MZ and DZ twin analyses, providing a powerful validation to the CTD.

To date, there is no evidence of increased MZ environmentally influencing MZ co twin correlations for political preferences. For example, it is difficult to conceive of a population where parents of MZ twins would purposely or unconsciously socialize their children to support the same political party, whereas parents of DZ twins, simply because the twins do not look alike or are not genetically identical, socialize them for opposing political values. This would run counter to all that political scientists know regarding political socialization.

2.2.6 Power and Sample Size

An important limitation in twin analyses, and a by-product of using population-based samples, is the necessity for large sample sizes in order to have confidence in the results. This is particularly important with the use of ordinal data (Neale, Eaves, and Kendler 1994). As shown in Fig. 1, the number of twin pairs required to detect the presence of additive genetic or common environmental effects in traits with low prevalence can be prohibitive. Samples in the hundreds offer very little statistical power, and only full model results should be considered in small samples.

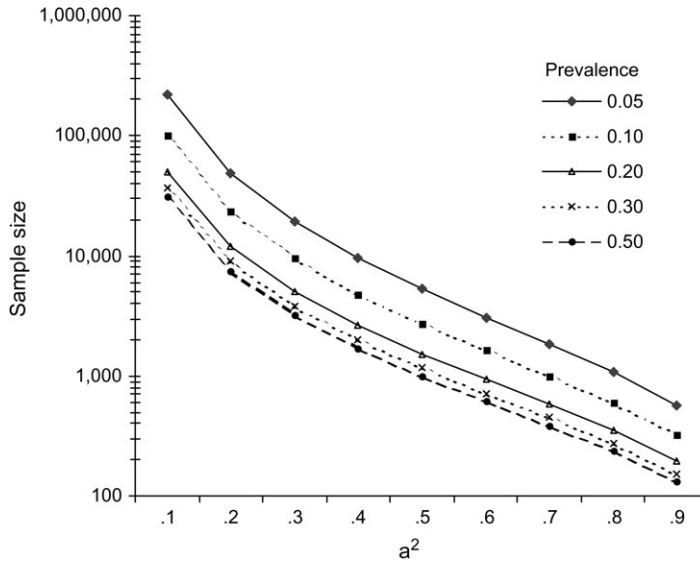


Fig. 1 The graph depicts the effects of prevalence and heritability (a^2) on the power to reject a common and unique environmental (CE) model when the true model is an additive genetic and unique environmental (AE) model at the .05 level of significance and 80% power.

2.3 Alternative Sources of Variance

The classical twin model (CTD) assumes that genetic and environmental influences are orthogonal and that parents do not share any genes in common. However, it is possible that genetic and environmental influence may correlate or interact and that parents, while not related, may share similar genetic factors. Although each of these possibilities would bias the estimates in a CTD, extensions of the CTD exist to model the interaction of genes and environment.

Genotype by environment correlation (r_{GE}) refers to the hypothesis that an individual's genes may influence their exposure to certain nonrandom environmental stimuli. In effect, one is "choosing" their environment based in part on their genes (Caspi et al. 2003; Eaves 1969; Eaves, Eysenck, and Martin 1989; Saudino 1997). This correlation may be classified as active, where the individual's own genes influence their exposures to certain environments or passive in which the environment of an individual is influenced by the genes of a relative. Unmodeled active r_{GE} may either inflate or deflate the estimates of genetic influences. Unmodeled passive r_{GE} may increase the estimates of common environmental effects. Traditionally longitudinal data have been required to test these effects.

Genotype by environment interaction ($G \times E$) arises when individuals with different genotypes differ in their response or sensitivity to the environment. The presence of $G \times E$ means that the estimates of genetic and environmental influences will differ within the sample based on environmental stimuli. If the environmental stimuli are not shared among the siblings, the estimates of the unique environment will be inflated. However, if the environmental influences are shared among the siblings, estimates of additive genetic influences will be inflated. The presence of $G \times E$ may be assessed by testing for a correlation between the sum and absolute differences of data from MZ co-twins (i.e., heteroscedasticity) (Jinks and Fulker 1970; van der Sluis et al. 2006) or by partitioning the variance

components into that which is linked to variation within the environmental influence and that which is residual (see Purcell 2002).

Assortative mating refers to the tendency for individuals to choose romantic partners who are similar to themselves and is widely addressed in nearly all disciplines examining social behavior, including political science (Stoker and Jennings 1995), sociology (Kalmijn 1994), psychiatry (Merikangas 1984), and genetics (Heath and Eaves 1985; Heath et al. 1987; Vandenberg 1972). The classical twin model, as well as analyses based upon random sampling, implicitly assumes that the population mates randomly with respect to the trait under analysis. For social and political attitudes, this assumption is violated (e.g., see Hatemi et al. 2007; Eaves et al. 1999; Martin et al. 1986). However, if data are collected from the parents of the twins (or the spouses of adult twins), then assortative mating can be explicitly modeled. Hypothetically, when two individuals who are similar for the trait of interest have children, and this trait is genetically influenced, genetic similarity of the siblings for the trait of interest will be increased so that genetic sharing of DZ twins or regular siblings will be greater than the .5 assumed in most genetic models. In a CTD, such an effect would lead to inflation in the estimation of the common environment and an underestimation of genetic influences.³

3 Twin and Family Data Modeling Techniques

Differential equation methods often referred to as Holzinger (1929) or Falconer (1960) transformations were popular with early twin studies and provide estimates of the proportions of variance due to additive genetic (A), nonadditive genetic effects (D), common environment (C), and unique environment (E) effects from the MZ and DZ correlations as follows:

$$\begin{aligned} A &= 2(r_{MZ} - r_{DZ}) \text{ assuming } D \cong 0; \\ C &= 2r_{DZ} - r_{MZ}; \\ D &= 2r_{MZ} - 4r_{DZ}; \\ E &= 1r_{MZ}. \end{aligned}$$

In addition, if non-twin sibling data were collected, a special twin effects could be calculated as

$$T = r_{DZ} - r_{\text{Sibling}}.$$

In response to statistical limitations, improved methods, typified by the seminal papers of Eaves (1969, 1977) and Martin and Eaves (1977), focused on the use of structural equation modeling using weighted least-squares analyses of precomputed variance–covariance matrices. Increases in computational power and access led to modeling of raw data for both continuous and ordinal traits for a wide range of complex situations, thereby allowing behavioral geneticists to develop models that more accurately reflect the complexities of human behavior and development (Mehta and Neale 2005). To this end, there is a focus on explicitly modeling the effects of covariates and interaction effects rather than correcting for these effects prior to modeling the data.

³The nuclear family analyses by Hatemi et al. (2008) included assortative mating and provided evidence that genetic influences on political preferences were underestimated in twin-only analyses.

Arguably, structural equation modeling within a maximum likelihood (ML) framework is the most common approach to data analyses with twin and family data (Posthuma et al. 2003).⁴ Using the ML approach, the algorithm minimizes the goodness-of-fit between observed covariance and mean/prevalence matrices and those predicted by models of genetic and environmental influence. This optimization procedure considers how well the model fits the data for a range of parameter values and arrives at the solution (converges) when it finds the parameters that produce the lowest log-likelihood. The parameter values that produced the optimized solution are estimates of the magnitude of the “latent” sources of variance. The reliability of these estimates are expressed as a 95% confidence intervals, which are the positive and negative deviations from these estimates that result in a change in the fit of the model (minus twice log-likelihood, $-2LL$) of 3.64 (equivalent to χ^2 , $p = .05$) (Neale et al. 2003).

Dropping parameters from the model allows the researcher to test whether the trait is a function of genes, social environment, and unique environment, or some combination thereof. This is accomplished by comparing the fit of the full model in which the parameters are freely estimated to the fit of the model in which one or more of the parameters have been set to zero (known as dropping the parameter) or fixed to a specified value. For instance, if one wanted to test whether personality was largely due to genes and personal experience only, the researcher would simply drop the C term from the model and examine the model fit in comparison to the full ACE model. The difference in model fits, assuming that the models are nested (one model is a submodel of the other), is asymptotically distributed as a chi-square distribution with the degrees of freedom equal to the difference in the number of estimated parameters between the two models.

4 Common Univariate Models

When analyzing family data, it is required to specify the model that will be fitted to the variances and covariances. In addition, when analyzing raw data, it is also necessary to provide a model for the means (or prevalences in the case of ordinal data) which can include the effects of covariates such as age, social economic status, etc. When modeling continuous data, three sources of information are available: the mean, variances, and covariances of each subgroup within the sample. When modeling ordinal data, one typically employs the multifactorial liability threshold model which postulates that the data collected is an indirect and imprecise measure of an underlying latent trait (typical referred to as the distribution of liability) that has not been, or cannot be, measured precisely. Instead, liability is measured as a series of ordered categories, characterized by phenotypic (trait) discontinuities that occur when the liability reaches a given threshold. The multifactorial liability is assumed to reflect the combined effects of genes and environmental factors (Neale and Cardon 1992). This underlying continuous latent variable is usually assumed to follow a standard normal (z score) distribution that allows the prevalence of each ordered category to be expressed in terms of cut points, expressed as z scores, which divide the distribution. Under this model, differences in thresholds between subgroups or samples imply differences in variances.

⁴A comparison of the methodological assumptions and flexibility of correlation transformations, correlation matrices, and raw data approaches is available online at http://www.vipbg.vcu.edu/_sarahme/methods.

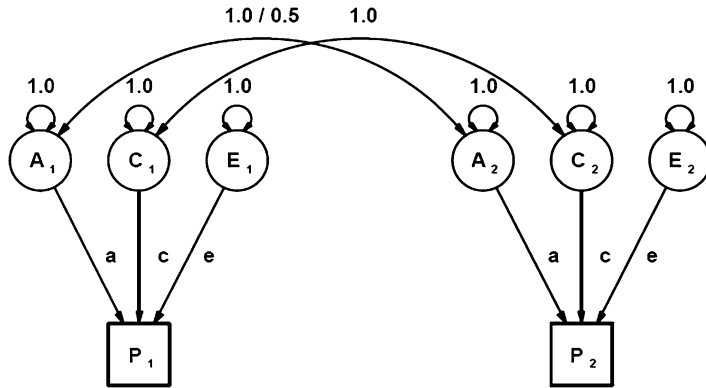


Fig. 2 ACE path diagram with labeled paths. Notes: Squares denote measured traits. Circles denote latent variables. Upper-case letters denote variables. Lower-case letters denote path coefficients. Single-headed arrows or paths represent hypothesized causal relationships between variables (the causal variable is the source of the arrow). Double-headed arrows represent covariances between variables or the covariance of a variable with itself.

To illustrate this point, consider the analyses of voter preference by Hatemi et al. (2007). Given the predominantly two party system in Australia, the data were effectively reduced to a binary variable: those who voted for the more conservative Liberal/National Coalition and those who voted for the less conservative Australian Labor Party. The binary variable of vote choice could be viewed as an indirect measure of the voters’ overall ideological issue positions on the grounds that voters choose between the parties on the basis of their issue platforms and that these platforms represent positions on an ideological scale. Of the 94% of participants who voted for one of these two main parties, 56.95% voted for the conservative coalition. This prevalence can be mapped onto an underlying hypothetical distribution and expressed as a z-score of 0.175.

The main advantages of working with thresholds are that they directly map onto current conceptualizations of the cumulative effects of genetic and environmental influences. An additional convenience of this approach is that when analyzing data under the threshold model, the variances of the variables are set to 1; this means that the estimates of variance effects (ACE) are standardized estimates which can be easily compared across samples.

Figure 2 presents the path diagram for a univariate ACE model. The expected covariance between two variables, or the expected variance of a variable, is computed by (1) multiplying together all the coefficients in a chain and then summing over all possible chains and (2) trace backward, change direction at a two-headed arrow, and then trace forward.

Applying these rules to the ACE model (Fig. 2), the variance for an MZ twin is calculated as $(a*1*a) + (c*1*c) + (e*1*e) = a^2 + c^2 + e^2$. Figure 3 provides the ACE model from the analyses of Australian voting data described above by Hatemi et al. (2007); the population-level additive genetic effects accounted for 24% of the variation ($.48 \times .48$) in vote choice.

Aside from providing an explicit summarization of the model, path diagrams are also helpful tools for translating theoretical models into the matrix algebra used in structural equation modeling. For example, following from Figs 2 and 3, the algebraic statements derived for the variance/covariance matrices of MZ and DZ twins are:

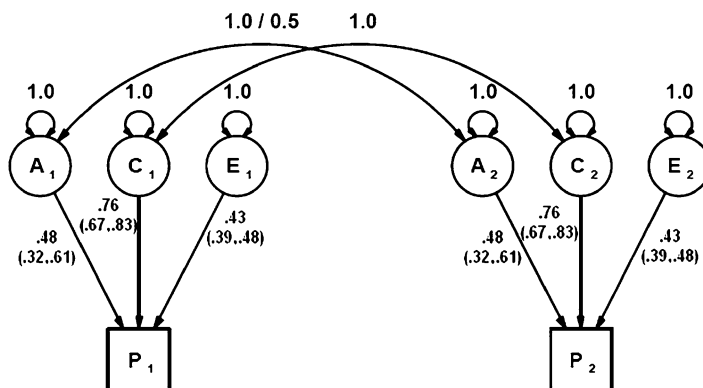


Fig. 3 ACE diagram for vote choice in the Australian vote choice data.

$$\text{Variance – covariance matrix} \begin{bmatrix} \text{var twin1} & \text{cov twin1 \& twin2} \\ \text{cov twin1 \& twin2} & \text{var twin2} \end{bmatrix}$$

$$\text{MZ} \begin{bmatrix} a^2+c^2+e^2 & a^2+c^2 \\ a^2+c^2 & a^2+c^2+e^2 \end{bmatrix} \quad \text{DZ} \begin{bmatrix} a^2+c^2+e^2 & .5 \times a^2+c^2 \\ .5 \times a^2+c^2 & a^2+c^2+e^2 \end{bmatrix}.$$

4.1 Common Extensions of the Univariate Twin Model

Extensions of the classical twin model are often warranted due to the availability of additional familial data (such as non-twin siblings) or in cases where a violation of the fairly restrictive assumptions of the classical twin model are present (as is often the case when modeling sex differences).

4.2 Extended Twin Sibling Model

This model is used when data from twins and their non-twin full siblings are available. The means/threshold model is extended by including an extraparameter for each additional sibling (i.e., if the largest family contains a set of twins and three additional siblings there would be five means parameters). These parameters are usually set to be equal. The variance–covariance of the additional siblings is modeled in the same way as for a DZ twin. Thus, the typical variance–covariance model would be extended as follows to include the data of one extra sibling:

$$\text{Variance – covariance matrix} \begin{bmatrix} \text{var twin1} & \text{cov twin1 \& twin2} & \text{cov twin1 \& sibling} \\ \text{cov twin1 \& twin2} & \text{var twin2} & \text{cov twin2 \& sibling} \\ \text{cov twin1 \& sibling} & \text{cov twin2 \& sibling} & \text{var sibling} \end{bmatrix}$$

$$\text{MZ} \begin{bmatrix} a^2+c^2+e^2 & a^2+c^2 & .5 \times a^2+c^2 \\ a^2+c^2 & a^2+c^2+e^2 & .5 \times a^2+c^2 \\ .5 \times a^2+c^2 & .5 \times a^2+c^2 & a^2+c^2+e^2 \end{bmatrix} \quad \text{DZ} \begin{bmatrix} a^2+c^2+e^2 & .5 \times a^2+c^2 & .5 \times a^2+c^2 \\ .5 \times a^2+c^2 & a^2+c^2+e^2 & .5 \times a^2+c^2 \\ .5 \times a^2+c^2 & .5 \times a^2+c^2 & a^2+c^2+e^2 \end{bmatrix}.$$

Additional siblings may be added in the same way. A major advantage of this sampling design and modeling extra sibling data is that it vastly increases the power to detect common environmental effects (Posthuma et al. 2003). The power to detect common environment is

Table 1 Variance and covariance by sex and zygoty for sex-limited models as compared to the classical twin model

Model	<i>I, General</i>			
	<i>nonscalar sex limitation</i>	<i>II, Nonscalar sex limitation</i>	<i>III, Scalar sex limitation^a</i>	<i>IV, Classical twin model</i>
Female variance	$a_f^2 + c_f^2 + e_f^2$	$a_f^2 + c_f^2 + e_f^2$	$k(a^2 + c^2 + e^2)k'$	$a^2 + c^2 + e^2$
Male variance	$a_m^2 + c_m^2 + e_m^2 + m^2$	$a_m^2 + c_m^2 + e_m^2$	$a^2 + c^2 + e^2$	$a^2 + c^2 + e^2$
MZF covariance	$a_f^2 + c_f^2$	$a_f^2 + c_f^2$	$k(a^2 + c^2)k'$	$a^2 + c^2$
MZM covariance	$a_m^2 + c_m^2 + m^2$	$a_m^2 + c_m^2$	$a^2 + c^2$	$a^2 + c^2$
DZF covariance	$\frac{1}{2}a_f^2 + c_f^2$	$\frac{1}{2}a_f^2 + c_f^2$	$k(\frac{1}{2}a^2 + c^2)k'$	$\frac{1}{2}a^2 + c^2$
DZM covariance	$\frac{1}{2}a_m^2 + c_m^2 + \frac{1}{2}m^2$	$\frac{1}{2}a_m^2 + c_m^2$	$\frac{1}{2}a^2 + c^2$	$\frac{1}{2}a^2 + c^2$
DZOS (opposite sex) covariance	$\frac{1}{2}(a_f a_m) + (c_f c_m)$	$\frac{1}{2}(a_f a_m) + (c_f c_m)$	$k(\frac{1}{2}a^2 + c^2)k'$	$\frac{1}{2}a^2 + c^2$
Compared to				
Model number	IV	IV	II	—
df	1	3	1	—

^a $k = \begin{bmatrix} \frac{Fvar}{Mvar} & 0 \\ 0 & \frac{Fvar}{Mvar} \end{bmatrix}$ for same-sex female pairs and $k = \begin{bmatrix} \frac{Fvar}{Mvar} & 0 \\ 0 & 1 \end{bmatrix}$ for opposite-sex pairs. A prime (') is used to indicate a transpose.

maximized when there are 4 times as many DZ pairs as MZ pairs (Nance and Neale 1989). Including data from extra siblings in the model effectively increases the DZ to MZ ratio. Including parental data in the model is also possible. This model, known as the extended twin family model or the nuclear family model (Hatemi et al. 2008), allows researchers to test whether the maternal and paternal contributions to the familial environment are equal while correcting for assortative mating.

4.3 Sex Limitation

Sex limitation is the term used to refer to sex differences in the magnitude and or proportion of the variance accounted for by genetic and environmental effects (Neale and Cardon 1992). There are three types of sex limitation: scalar, nonscalar, and general nonscalar, each described in turn and summarized in Table 1.

The scalar sex limitation is the simplest and most restrictive of the these models in which the absolute magnitude of the total variance differs between males and females, whereas the proportion of variance accounted for by genetic and environmental effects do not. The name arises from the estimated ratio of male to female variance which is known as a scalar (typically denoted as k within the algebra). As the variances are fixed to unity when working with ordinal data, this model can only be tested when working with continuous data.

In nonscalar sex limitation models, the genetic and environmental effects are estimated separately for males and females. For the general nonscalar (qualitative) model, an extra-genetic or environmental component (m^2) can be modeled for males or females. Conceptually, this model describes a situation in which different genes or environmental factors influence males and females. Differences in both genetic and environmental effects cannot be tested simultaneously when working with twin and sibling data. As such, one would usually run this model twice once specifying m^2 as an additive genetic parameter

(correlated .5 for the DZ twins) and once specifying m^2 as an common environment parameter (correlated 1 for the DZ twins).

In the standard nonscalar (quantitative) model, the sources of variance in males and females are assumed to be the same and the correlation for additive genetic and common environmental influences in the opposite-sex pairs are assumed to be .5 and 1, respectively. Conceptually, this model describes a situation in which the same genes and environmental effects are hypothesized to influence males and females, but the magnitudes of these effects are allowed to differ. The difference in fit between these models can be used to examine whether the same genes or environmental factors are influencing males and females. However, this test can only be conducted if data have been collected from opposite-sex twins (Neale and Cardon 1992).⁵

5 Common Multivariate Models

Human behaviors, attitudes, and decision making processes are seldom characterized as easily measured unidimensional constructs. As a result, the majority of researchers are interested in the simultaneous modeling of more than one variable in order to draw conclusions about the extent to which related variables influenced a given trait. Similar logic applies when modeling genetic or environmental influences. Although biometric designs are still new to the discipline, it is logical to assume more advanced models will soon appear; thus, it is appropriate to address the most common multivariate models (e.g., see Hatemi, Medland, and Eaves 2009). Rather than focus on the total variance caused by one or more independent variables, multivariate genetic modeling partitions out the extent and mechanisms by which one trait relates to another. Twin and family analyses of raw data using structural equation modeling are readily extendable to a variety of multivariate cases.

Genetic analyses of multivariate data draw their explanatory power from the information contained in the variances, cross trait (within individual phenotypic covariance), cross twin (MZ and DZ co-twin) and cross twin–cross trait covariances. These elements are shown schematically in Fig. 4. The magnitude of the cross trait covariance elements provides an indication as to whether the traits under analysis share common etiological influences. The relative magnitude of the MZ and DZ cross twin–cross trait covariances provide information regarding whether these causal influences are likely to be genetic or environmental in nature.

Generally, when beginning multivariate analysis, it is customary to begin by fitting an ACE Cholesky decomposition to the data. This is a simple factor model in which there are as many orthogonal factors, n as there are variables. All variables load on the first factor, $n - 1$ variables load on the second factor and so on, until the final variable loads on the n th factor only. This factor structure is modeled as a lower diagonal matrix (containing estimates of the factor effects known as path coefficients) which is multiplied by its transpose to produce the full factor model. This factor pattern is repeated for each of the three (ACE) concurrently modeled sources of variation (i.e., the *triple Cholesky*). The path diagram for a bivariate ACE Cholesky decomposition is given in Fig. 5. The Cholesky decomposition is typically described as the saturated model and will have the best fit of any multivariate model. However, by definition, the saturated model is also the least parsimonious model that can be applied to the data and provides an ideal comparison point

⁵See Hatemi, Medland, and Eaves (2009) for an example of sex limitation on political attitudes.

		Phenotype1 (P1)	Phenotype2 (P2)	Phenotype1 (P1)	Phenotype2 (P2)
Twin 1	Phenotype1 (P1)	Variance P1 $a^2_{P1} + c^2_{P1} + e^2_{P1}$	Cross trait Covariance $r_{AaP1aP2} + r_{Cp1Cp2} + r_{EeP1eP2}$	Within trait cross twin covariance $(.5/1)a^2_{P1} + c^2_{P1}$	Cross trait cross twin covariance $(.5/1)r_{AaP1aP2} + r_{Cp1Cp2}$
	Phenotype2 (P2)	Cross trait Covariance $r_{AaP1aP2} + r_{Cp1Cp2} + r_{EeP1eP2}$	Variance P2 $a^2_{P2} + c^2_{P2} + e^2_{P2}$	Cross trait cross twin covariance $(.5/1)r_{AaP1aP2} + r_{Cp1Cp2}$	Within trait cross twin covariance $(.5/1)a^2_{P2} + c^2_{P2}$
Twin 2	Phenotype1 (P1)	Within trait cross twin covariance $(.5/1)a^2_{P1} + c^2_{P1}$	Cross trait cross twin covariance $(.5/1)r_{AaP1aP2} + r_{Cp1Cp2}$	Variance P1 $a^2_{P1} + c^2_{P1} + e^2_{P1}$	Cross trait Covariance $r_{AaP1aP2} + r_{Cp1Cp2} + r_{EeP1eP2}$
	Phenotype2 (P2)	Cross trait cross twin covariance $(.5/1)r_{AaP1aP2} + r_{Cp1Cp2}$	Within trait cross twin covariance $(.5/1)a^2_{P2} + c^2_{P2}$	Cross trait Covariance $r_{AaP1aP2} + r_{Cp1Cp2} + r_{EeP1eP2}$	Variance P2 $a^2_{P2} + c^2_{P2} + e^2_{P2}$

Fig. 4 Schematic of bivariate variance/covariance matrix. Notes: The within-twin covariances are shown in gray and the cross-twin covariances are shown in black.

for more restrictive models, allowing researchers to test the trade-off between parsimony and model fit.

The ordering of variables in a Cholesky is important and should be based on theoretical assumptions. In models examining a predictive relationship, variables should be placed in order of hypothesized predictive ability, with the dependent variable as the last variable in the Cholesky. Model simplification proceeds in an *a priori* fashion by testing specific multivariate models based on theoretical expectations.

Interpretation of the results depends on some extent to the model fitted (Loehlin 1996). Broadly speaking, there are three main ways to discuss the covariation between relatives. To illustrate these methods, consider the example of the additive genetic covariation for the situation described in Fig. 5 (the same approaches also apply to common and unique environmental variance). First, interpretation can focus on the standardized path coefficients and the extent to which covariation between variables is due to each factor.⁶ Alternatively, one may discuss the proportions of variance explained and the percent of the phenotypic correlation (*r*) due to additive genetic effects (A) by dividing the estimates of the variance due to A (calculated as XX') by the total variance.

$$\begin{bmatrix} x^2_{11} & x_{11}x_{21} \\ x_{11}x_{21} & x^2_{21} + x^2_{22} \end{bmatrix} \div \begin{bmatrix} \text{var } v1 & \text{cov } v1v2 \\ \text{cov } v1v2 & \text{var } v2 \end{bmatrix} = \begin{bmatrix} \text{heritability } v1 & \% \text{ of } r \text{ due to A} \\ \% \text{ of } r \text{ due to A} & \text{heritability } v2 \end{bmatrix}$$

⁶Researchers typically report the standardized path coefficients because interpretation of the absolute path coefficients is confounded by potential differences in the variance between variables.

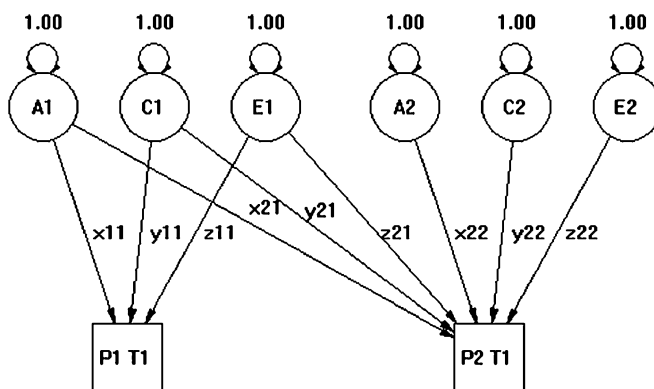


Fig. 5 Bivariate Cholesky. Notes: P1T1 is the measured trait value for phenotype 1, twin 1; P2T1 is the trait value for phenotype 2, twin 1.

Finally, researchers may also discuss the genetic correlations, which are the correlations between the genetic effects of the variables. The additive genetic correlation between the first and second variables in Fig. 5 can be calculated using the following formula:

$$r_A = \frac{x_{21}x_{11}}{\sqrt{x_{11}^2 \times (x_{21}^2 + x_{22}^2)}}$$

Matrix algebra also offers a means to obtain a genetic correlation matrix by pre- and postmultiplying the estimates of the variance due to A by a matrix containing standard deviations on the diagonals. It is important to keep in mind the proportion of variance that is being explained by A when interpreting the additive genetic correlation. For example, an additive correlation of .95 has very little practical meaning when additive genetic effects are only accounting for 5% of the overall variation.

5.1 Models for the Analysis of Three or More Variables

The following is a brief description of the most frequently used multivariate models, the *common* and *independent pathways* models.

The *common pathway model* (shown in Fig. 6) is a restrictive model which hypothesizes that the covariation between variables is due to a single underlying “phenotypic” latent variable. For example, consider the hypothesis that self-reports of the importance of traditional family values, fiscal responsibility, and judicial restraint may all be measures of a latent phenotypic trait called “conservatism” which is itself influenced by both genetic and environmental effects. Thus, genetic and environmental variation in reports of the importance of fiscal responsibility is expected to be due in part to the genetic and environmental effects influencing conservatism with the extent of this transmitted variation being described by the phenotypic factor loadings. The residual variance is expected to be due to genetic and environmental effects that are specific to the reports of the importance of fiscal responsibility itself and are not correlated with the residual variances of reports of the other two traits that make up the latent construct of conservatism, the importance of traditional family values or judicial restraint. This model, though conceptually attractive, often does not fit the data well because the amount of genetic and environmental variation transmitted from the latent factor is defined by the phenotypic relationship between the measured and latent variables. However, genetic variance influencing the latent variable conservatism

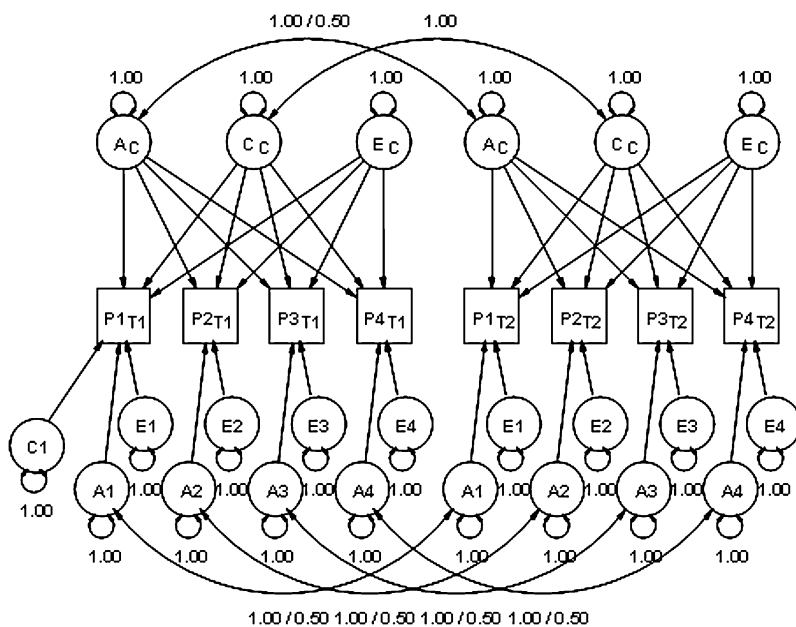


Fig. 7 Independent pathway model.

the main theories of political preferences for each trait specifically. If common environmental influences are not significant, familial socialization could potentially be ruled out as the strongest casual influence for individual differences in a given population for that trait. If this finding were replicated across populations, a more general interpretation may be warranted. If additive genetic influences account for a significant portion of the variance, then rational choice, as traditionally defined, may not be the best model to explain individual differences for that specific trait. It may also begin to provide a source of preferences not typically used in formal models. If genes account for the majority of the variance in any given trait, or are found to have a compounding effect with certain environmental stimuli, the potential for a different theory or modifications of current theories for political behavior is present. The inclusion of variance due to genetic effects, in effect, provides the study of political behavior one means to test the discipline's traditional theories for each trait under analysis.

Importantly, traditional predictive models and variance components approaches are not mutually exclusive or even competing. By integrating the information gained from the source of variance for each predictor, scholars can obtain a more complete picture of the way in which behaviors and attitudes develop for both the dependent variable and each of the predictors. Incorporating both genetic and environmental influences provides a complementary way to study political behaviors thereby increasing the explanatory power of existing political science models rather than replacing them. To be clear, variance component models do not identify the best predictors for any given phenomena, or address mean effects. Rather they partition the source of variation for each of the predictors used and provide information on the relative importance of the predictors.

The biometrical approach can also be used without modeling genetic effects. Specifically, if one chose to only model familial transmission in general, simply combining the

additive genetic and common environmental sources of variance would encompass all transmission that was not unique to the individual (e.g., Eaves and Hatemi 2008b). This partition of unique environmental and family influences alone is extremely valuable in examinations of familial versus unique environmental transmission.

Perhaps, the most important question is why the source of variance should matter if social scientists are interested in mean effects and are agnostic about the inherent source of variation? Why invest in this methodology if it does not offer any predictive ability? Although not immediately apparent, twin studies are just a first step in genetic analyses, albeit a very important and necessary one. Technological advances in both computational and laboratory techniques have led to the integration of variance component analyses with actual genetic information derived from DNA. Once genetic influences are confirmed by twin analyses, researchers may then start searching for the specific genes that are influencing behavior through genome-wide or allelic association (see Hatemi et al. 2008). In simpler terms, once heritability is established, it opens the door to look for specific genes which do provide a predictive capacity. One such example in the political science literature initially used a CTD (Fowler, Baker, and Dawes 2008) to explore voter participation. Fowler and Dawes (2008) followed up by identifying two genes that appear to influence voter turnout.

There are some important challenges to using biometrical methods, primarily the difficulty in gaining access to existing data from related individuals and collecting new data. The latter requires a change in the traditional sampling frame for political studies to purposely include data from related individuals. Although the Add Health and soon to be NSF data will undoubtedly go a long ways in alleviating the initial costs in fiscal terms, the time needed to invest in new processes and methods may still be prohibitive for the early adapters.

Despite the hurdles, it is hoped that this introduction of biometric methods and twin samples will provide a means by which political scientists can both utilize and critically assess future studies of genetic influence in a productive way. More importantly, it is our desire that political scholars will adapt the existing methodologies from biometric studies and tailor them for social science research. Twin designs are merely a first step, and although not definitive, armed with a full understanding of the methodological steps taken to verify the findings, as well as scientific understanding of the assumptions and limitations, they are difficult to dismiss. MZ twins are more alike than DZ twins for reasons other than the environment. Research that began with twin modeling has led to discoveries of genes that influence depression, autism, obesity, and host of important social and medical traits (e.g., Campbell et al. 2006; Frayling et al. 2007; Haberstick, Smolen, and Hewitt 2006). We hope that these methods will be adopted and modified by political scientists to address the complicated and important social and political constructs examined within the discipline, thereby providing an interdisciplinary platform to address the source of preferences from both a social and genetic perspective.

Funding

Sidney Sax National Health and Medical Research Council Australia Public Health Fellowship (443036 to S.E.M.).

References

Alford, J., C. Funk, and J. Hibbing. 2005. Are political orientations genetically transmitted? *American Political Science Review* 99:153–67.

- Bouchard, T. J. Jr., and M. McGue. 2003. Genetic and environmental influences on human psychological differences. *Journal of Neurobiology* 54:4–45.
- Busjahn, A. 2002. Twin registers across the globe: What's out there in 2002. *Twin Research and Human Genetics* 5:V–VI.
- Campbell, D. B., J. S. Sutcliffe, P. J. Ebert, R. Militerni, C. Bravaccio, S. Trillo, M. Elia, et al. 2006. A genetic variant that disrupts MET transcription is associated with autism. *Proceedings of the National Academy of Science of the United States of America* 103:16834–9.
- Caspi, A., J. McClay, T. E. Moffitt, J. Mill, J. Martin, I. W. Craig, A. Taylor, and R. Poulton. 2002. Role of genotype in the cycle of violence in maltreated children. *Science* 297:851–4.
- Caspi, A., K. Sugden, T. E. Moffitt, A. Taylor, I. W. Craig, H. Harrington, J. McClay, et al. 2003. Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science* 301:386–9.
- Cohen, D. J., E. Dibble, J. M. Grawe, and W. Pollin. 1973. Separating identical from fraternal twins. *Archives of General Psychiatry* 29:465–9.
- Eaves, L. J., A. C. Heath, N. G. Martin, H. H. Maes, M. C. Neale, K. S. Kendler, K. M. Kirk, and L. Corey. 1999. Comparing the biological and cultural inheritance of personality and social attitudes in the Virginia 30 000 study of twins and their relatives. *Twin Research* 2:62–80.
- Eaves, L. J. 1969. The genetic analysis of continuous variation. A comparison of experimental designs applicable to human data. *British Journal of Mathematical and Statistical Psychology* 22:131–47.
- . 1977. Inferring the causes of human variation. *Journal of the Royal Statistical Society* 140:324–55.
- Eaves, L. J., H. J. Eysenck, and N. G. Martin. 1989. *Genes, culture and personality; an empirical approach*. London: Academic Press.
- Eaves, L. J., and P. K. Hatemi. 2008a. Transmission of attitudes toward abortion and gay rights: Parental socialization or parental mate selection? *Behavior Genetics* 38:247–56.
- . 2008b. *Do we choose our spouse based on our in-laws?* Unpublished manuscript.
- Evans, D. M., N. A. Gillespie, and N. G. Martin. 2002. Biometrical genetics. *Biological Psychology* 61:33–51.
- Eysenck, H. J., and M. W. Eysenck. 1985. *Personality and individual differences: A natural science approach*. New York: Plenum.
- Falconer, D. S. 1960. *Introduction to quantitative genetics*. Edinburgh: Oliver and Boyd.
- Fowler, J. H., L. A. Baker, and C. T. Dawes. 2008. Genetic variation in political participation. *American Political Science Review* 102:233–48.
- Fowler, J. H., and C. T. Dawes. 2008. Two genes predict voter turnout. *Journal of Politics* 70:579–94.
- Frayling, T. M., N. J. Timpson, M. N. Weedon, E. Zeggini, R. M. Freathy, C. M. Lindgren, J. R. B. Perry, et al. 2007. A common variant in the FTO gene is associated with body mass index and predisposes to childhood and adult obesity. *Science* 316:889–94.
- Haberstick, B. C., A. Smolen, and J. K. Hewitt. 2006. Family-based association test of the 5HTTLPR and aggressive behavior in a general population sample of children. *Biological Psychiatry* 59:836–43.
- Hall, J. G. 2003. Twinning. *Lancet* 362:735–43.
- Happonen, M., P. Lea, K. Jaakko, M. Jaap Van der, J. V. Richard, and J. R. Richard. 2002. The heritability of depressive symptoms: multiple informants and multiple measures. *Journal of Child Psychology and Psychiatry* 43:471–9.
- Hatemi, P. K. 2007. *The genetics of political attitudes*. PhD diss. University of Nebraska-Lincoln.
- Hatemi, P. K., J. Hibbing, J. Alford, N. G. Martin, and L. J. Eaves. 2008. We get opinions from our parents, but not how we think we do: Genetic and social components of the familial transmission of political attitudes. Presented at the Midwest Political Science Association Annual Conference, Chicago, IL, April 2008.
- Hatemi, Peter K., Hibbing John, Alford John, Martin Nicholas, and Eaves Linton. 2009. Is there a party in your genes? *Political Research Quarterly*. Forthcoming.
- Hatemi, P. K., S. E. Medland, and L. J. Eaves. 2009. Genetic sources for the gender gap? *Journal of Politics* 71:1–13.
- Hatemi, P. K., S. E. Medland, K. I. Morley, A. C. Heath, and N. G. Martin. 2007. The genetics of voting: An Australian twin study. *Behavior Genetics* 37:435–48.
- Heath, A. C., and L. J. Eaves. 1985. Resolving the effects of phenotype and social background on mate selection. *Behavior Genetics* 15:15–30.
- Heath, A. C., L. J. Eaves, W. E. Nance, and L. A. Corey. 1987. Social inequality and assortative mating: Cause or consequence? *Behavior Genetics* 17:9–17.
- Heath, A. C., R. Jardine, and N. G. Martin. 1989. Interactive effects of genotype and social environment on alcohol consumption in female twins. *Journal of Studies on Alcohol* 60:38–48.
- Hettema, J. M., M. C. Neale, and K. S. Kendler. 1995. Physical similarity and the equal-environments assumption in twin studies of psychiatric disorders. *Behavior Genetics* 25:327–35.

- Holzinger, K. 1929. The relative effect of nature and nurture influences on twin differences. *J Educ Psychol* 20:241–8.
- Jansson, M., M. Gatz, S. Berg, B. Johansson, B. Malmberg, G. E. McClearn, M. Schalling, and N. L. Pedersen. 2004. Gender differences in heritability of depressive symptoms in the elderly. *Psychological Medicine* 34:471–9.
- Jinks, J. L., and D. W. Fulker. 1970. A comparison of the biometrical-genetical, MAVA and classical approaches to the analysis of human behavior. *Psychological Bulletin* 73:311–49.
- Kalmijn, M. 1994. Assortative mating by cultural and economic occupational status. *American Journal of Sociology* 100:422–52.
- Kendler, K. S. 1983. Overview: Current perspective on twin studies of schizophrenia. *American Journal of Psychiatry* 140:1413–25.
- Kendler, K. S., A. C. Heath, N. G. Martin, and L. J. Eaves. 1987. Symptoms of anxiety and symptoms of depression. Same genes, different environments? *Archives of General Psychiatry* 44:451–7.
- Kendler, K. S., M. C. Neale, R. C. Kessler, A. C. Heath, and L. J. Eaves. 1993. A test of the equal-environment assumption in twin studies of psychiatric illness. *Behavior Genetics* 23:21–7.
- Krueger, R. F., K. E. Markon, and T. Bouchard Jr. 2003. The extended genotype: The heritability of personality accounts for the heritability of recalled family environments in twins reared apart. *Journal of Personality* 71:809–33.
- Loehlin, J. C. 1996. The Cholesky approach: A cautionary note. *Behavior Genetics* 26:65–9.
- Loehlin, J. C., and R. C. Nichols. 1976. *Heredity, environment, and personality*. Austin, TX: University of Texas Press.
- Lytton, H. 1977. Do parents create, or respond to, differences in twins? *Dev. Psychol* 13:456–9.
- Martin, N. G., and L. J. Eaves. 1977. The genetical analysis of covariance structure. *Heredity* 38:79–95.
- Martin, N. G., L. J. Eaves, A. C. Heath, R. Jardine, L. M. Feingold, and H. J. Eysenck. 1986. Transmission of social attitudes. *Proceedings of the National Academy of Science of the United States of America* 83:4364–8.
- Martin, N. G., and S. R. Witson. 1982. Bias in the estimation of heritability from truncated samples of twins. *Behavior Genetics* 12:467–72.
- Matheny, A. P., R. S. Wilson, and A. B. Dolan. 1976. Relations between twins' similarity of appearance and behavioral similarity: Testing an assumption. *Behavior Genetics* 6:343–51.
- Mehta, P. D., and M. C. Neale. 2005. People are variables too: multilevel structural equations modeling. *Psychol Methods* 10:259–84.
- Merikangas, K. R. 1984. Divorce and assortative mating among depressed patients. *American Journal of Psychiatry* 141:74–6.
- Nance, W. E., and M. C. Neale. 1989. Partitioned twin analysis: A power study. *Behavior Genetics* 19:143–50.
- Neale, M., S. Boker, G. Xie, and H. Maes. 2003. *Mx: Statistical modeling*. 5th ed. Richmond, VA: Department of Psychiatry, Virginia Commonwealth University.
- Neale, M. C., and L. L. Cardon. 1992. *Methodology for genetic studies of twins and families*. Kluwer Academic.
- Neale, M. C., L. J. Eaves, and K. S. Kendler. 1994. The power of the classical twin study to resolve variation in threshold traits. *Behavior Genetics* 24:239–58.
- Neale, M. C., L. J. Eaves, K. S. Kendler, and J. K. Hewitt. 1989. Bias in correlations from selected samples of relatives. *Behavior Genetics* 19:163–9.
- Ooki, S., K. Yamada, A. Asaka, and K. Hayakawa. 1990. Zygosity diagnosis by questionnaire. *Acta Genetica Medica et Gemellologica* 39:109–15.
- Plomin, R., L. Willerman, and J. C. Loehlin. 1976. Resemblance in appearance and the equal environments assumption in twin studies of personality traits. *Behavior Genetics* 6:43–52.
- Posthuma, D., A. L. Beem, E. J. de Geus, G. C. M. van Baal, J. B. von Hjelmborg, I. Iachine, and D. I. Boomsma. 2003. Theory and practice in quantitative genetics. *Twin Research* 6:361–76.
- Posthuma, D., and D. I. Boomsma. 2000. A note on the statistical power in extended twin designs. *Behavior Genetics* 30:147–58.
- Purcell, S. 2002. Variance components models for gene-environment interaction in twin analysis. *Twin Research* 5:554–71.
- Saudino, K. J. 1997. Moving beyond the heritability question: New directions in behavioral genetic studies of personality. *Current Directions in Psychological Science* 6:86–90.
- Scarr, S., and L. Carter-Saltzman. 1979. Twin method: Defense of a critical assumption. *Behavior Genetics* 9:527–42.
- Schieken, R. M., M. Mosteller, M. M. Goble, W. B. Moskowitz, J. K. Hewitt, L. J. Eaves, and W. E. Nance. 1992. Multivariate genetic analysis of blood pressure and body size. The Medical College of Virginia Twin Study. *Circulation* 86:1780–8.

- Sham, P. 1998. *Statistics in human genetics*. London: Arnold.
- Stoker, L., and M. K. Jennings. 1995. Life-cycle transitions and political participation: The case of marriage. *The American Political Science Review* 89:421–33.
- van der Sluis, S., C. V. Dolan, M. C. Neale, D. I. Boomsma, and D. Posthuma. 2006. Detecting genotype-environment interaction in monozygotic twin data: comparing the Jinks and Fulker test and a new test based on marginal maximum likelihood estimation. *Twin Research and Human Genetics* 9:377–92.
- Vandenberg, S. G. 1972. Assortative mating or who marries whom? *Behavior Genetics* 2:127–57.
- Visscher, P. M., S. E. Medland, M. A. R. Ferreira, K. I. Morley, G. Zhu, B. K. Cornes, G. W. Montgomery, and N. G. Martin. 2006. Assumption-free estimation of heritability from genome-wide identity-by-descent sharing between full siblings. *PLoS Genet* 2:e41.
- Xian, H., J. F. Scherrer, S. A. Eisen, W. R. True, A. C. Heath, J. Goldberg, M. J. Lyons, and M. T. Tsuang. 2000. Self-reported zygosity and the equal-environments assumption for psychiatric disorders in the Vietnam Era Twin Registry. *Behavior Genetics* 30:303–10.