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Integrating Social Science and Genetics: News from the Political Front

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There has been growing interest in the use of genetic models to expand the understanding of political preferences, attitudes, and behaviors. Researchers in the social sciences have begun incorporating these models and have revealed that genetic differences account for individual differences in political beliefs, behaviors, and responses to the political environment. The first Integrating Genetics and the Social Sciences Conference, held at Boulder, Colorado in May of 2010, brought together these researchers. As a result, we jointly review the last 5 years of research in this area. In doing so, we explicate the methods, findings, and limitations of behavior genetic approaches, including twin designs, association studies, and genome-wide analyses, in their application toward exploring political preferences.

There has been growing interest in the social sciences regarding the possibility that genetic factors contribute to individual differences in political and social behaviors (Fowler and Schreiber 2008). The position that the study of *individual differences* must include both genetic and environmental factors has long been echoed in the life sciences (e.g., Bouchard and McGue 2003; Eaves and Eysenck 1974; Eaves, Eysenck, and Martin 1989; Eaves and Carbonneau 1998; Eaves et al. 1999; Martin et al. 1986; Martin 1987; Olson, Vernon, Harris, and Jang 2001; Posner, Baker, Heath, and Martin 1996; Truett, Eaves, Meyer, Heath, and Martin 1992). However, until quite recently, this literature was largely ignored in the social sciences. Fortunately, both science and society have progressed, and genetic approaches have become an integral part of clinical, developmental, medical, psychological, and now political research. The results from the foundational pieces that identified genetic inheritance on social attitudes more than 25 years ago (Eaves and Eysenck 1974; Eaves et al. 1989; Martin et al. 1986) were further developed and disseminated to the political science community by Alford, Funk, and Hibbing in 2005. Their article in the *American*

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Political Science Review was widely acknowledged in both the mainstream media (Carey 2005) and academic literature (Sigelman 2006) and in large part launched a new era of genetic exploration for the social sciences.

Traditional social science models largely adhere to the assumption that human behavior and social attitudes are almost entirely the product of environmental influences. Accordingly, the focus is on identifying causal relationships wherein investigators do not consider the possibility that both the dependent and independent variables may have heritable or genetic components. Indeed, even though it is common to use biological indicators such as gender as predictors, the effects are interpreted as environmental or socialized. The assumption, made both explicitly and implied through statistical models, is that all people start with the same inherent disposition. Behavioral genetic (BG) analyses, conversely, are concerned with accounting for variation within a population and assume that dependent and independent variables may be a function of both genes and environment. BG approaches largely focus on why individuals differ from one another. Rather than prediction, the focus is on understanding the sources of individual variability and the inherent assumption that people have different dispositions.

The last decade of scholarship in the political sciences has begun to integrate these two approaches into more unified exploration of political behavior. Rather than treating “social” and “genetic” theories as competing models, the goal is to treat them as complementary approaches and to include elements of genes and environment into a unified theoretical approach that more precisely identifies the behavioral precursors and enable a richer understanding of how distinct behaviors are related to each other.

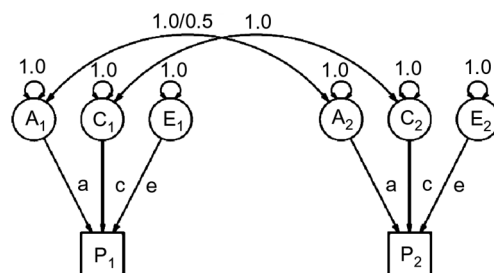
Here we review the empirical evidence from the last 5 years of remarkable growth in this research area. The theoretical framework for evaluating the roles of biological and social inheritance in the transmission of human behavior, however, is largely unknown to the social sciences and remains disconnected from the integral theories of political behavior that currently exist. Herein, we review the extant literature through the lens of the two distinct, but overlapping, behavioral genetic techniques used to explore genetic sources of individual differences on political preferences: twin and kinship models and molecular genetic analyses. Twin and kinship designs allow for population estimates of the amount of variation due to genetic and environmental factors, and molecular genetic approaches identify specific genetic variants related to the trait of interest. Our approach in this review is somewhat didactic; we first explicate the methods used and how these methods inform the study of behavior generally, followed by a review the studies using these methods with regard to political preferences, including how such findings have impacted the field of study. In doing so, we introduce social scientists to a significant and long-standing body of behavior genetic theory and methods, with the goal to more readily allow non-geneticists to incorporate this approach into their own work. For the sake of clarity, we restrict our review to the topics of greatest discussion in the political sciences: attitudes, ideology, partisanship, vote choice, and political participation. However, we recognize that a growing number of political scientists have become more engaged in genetic approaches and have begun to also conduct research in comparative politics, international relations, security studies, trust, public policy, and theory (Boardman et al. 2010; Hatemi 2010a; McDermott et al. 2009; Sturgis et al. 2010).

Twin and Kinship Models: Partitioning Genetic and Environmental Variance

Political scientists who first began to explore genetic sources of influences (e.g., Alford et al. 2005; Fowler, Baker, and Dawes 2008; Hatemi, Medland, Morley, Heath, and Martin

2007) initially followed the paths of their behavior genetic predecessors, Martin et al. (1986) and Eaves et al. (1989), who used large data sets of twins to decompose the population variance of political attitudes into estimates of genetic and environmental influences. The classic twin design, rooted in biometrical theory, relies on the knowledge that monozygotic twins are genetically identical (originate from a single fertilized ovum that splits into two fetuses very early in the gestation period), whereas dizygotic twins develop from two different eggs fertilized by different sperm (they are as genetically similar as non-twin siblings). Using these two types of twin pairs raised by the same parents in the same environment at the same time provides a natural experiment that allows for comparison of the two groups. That is, comparing the co-twin correlations for a given trait between a population of twin pairs who are genetically identical but have the same familial environment to the co-twin correlations of a population of non-identical twin pairs who share on average 50 per cent of their differentiating DNA and who also have the same family environment allows researchers to partition what part of the variance in a given trait is accounted for by genetic and environmental factors (see Neale and Cardon 1992). Thus, variance is typically decomposed into estimates of what portion of individual differences within a population are accounted for by additive genetic (A), common (C), and unique environmental (E) influences. “Additive genetic” is the latent additive effect of all genes (inherited traits). “Common environment” captures the latent influences that are shared by family members, such as familial socialization. “Unique environment” refers to influences from all idiosyncratic environmental stimuli that are unique to the individual (i.e., personal experience). The method is operationalized through differential equation methods (see Holzinger 1929; Falconer 1960), most often used in a structural model within a maximum likelihood (ML) framework (Posthuma et al. 2003) or Bayesian techniques (Fowler et al. 2008). Figure 1 presents the path diagram for a univariate ACE model (for a review of the CTD path model and equations and multivariate extensions see Medland and Hatemi 2009).

The classic univariate twin design and extensions that include parents, non-twin siblings, spouses, and other biological or adoptive relatives have been used to explore the source of individual differences for a wide variety of politically relevant traits, such as ideology, political attitudes, partisan attachment, and voter turnout (see Appendix for a summary of the published ACE findings on political traits). Across a large number of



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

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

Figure 1. Univariate twin design.

Notes: The variance for an MZ twin is calculated as (a*1*a) 1 (c*1*c) 1 (e*1*e) = a2 + c2 + e2, for DZ twins replace the A (genetic) path to 0.5 instead of 1.

studies from different political cultures, the general liberalism-conservatism dimensions and most individual attitudes were accounted for by a function of genetic inheritance (ranging between 0.2 and 0.6) and unique environmental influence (0.4 and 0.8; Alford et al. 2005; Hatemi et al. 2010). In terms of political behaviors, political participation is best accounted for by a function of genes, common environmental, and unique environmental influence (Fowler et al. 2008). However, for party identification, no genetic influence was found (Hatemi et al. 2009a), rather, the vast majority of variation was function of familial influence, a finding consistent with most theories of partisan affiliation (Campbell, Converse, Miller, and Stokes 1960) and a similar pattern to that of religious affiliation (Eaves and Hatemi 2008). Extension of the classical twin design has also been used to explore whether genetic and environmental influences differed by gender regarding political attitudes (Hatemi, Medland, and Eaves 2009). For most traits, no gender differences were present, with the exception of attitudes related to procreation (e.g., divorce and living together), where it was likely a different set of genes were influencing female versus male attitudes, a finding consistent with theories of developmental (evolutionary) differences between the genders.

The results from these univariate ACE kinship studies have fundamentally altered the ways social scientists approach the study of attitudes and present serious challenges to the existing literature. For example, the political affiliations and attitudes of parents and offspring and spouses are highly correlated (Niemi and Jennings 1991). This concordance has long been assumed to be evidence of familial socialization (Campbell et al. 1960; Jennings, Stoker, and Bowers 2009). In large part, this assumption remained untested due to the lack of any other plausible explanation. Certainly, the suggestion that ideological or attitudinal transmission might be other than social was rarely, if ever, considered. In this way, twin models have provided the necessary empirical venue for testing such assumptions. With regard to party affiliation, the extant literature appeared to be largely correct. Hatemi et al. (2009a) found no genetic influence for party identification, and some 80 per cent of individual variation was a function of familial influence. However, for attitudinal transmission, the assumption that the familial correlation was due to mutual socialization was not supported. Instead, regarding attitudes, it appears that children resemble their parents because of their genetic relatedness and not their social upbringing. In this way, whom individuals choose to procreate with (assortative mating) is much more important for attitudinal transmission than how they raise their children. Indeed, attitudinal similarity between spouses is due more to assortation than convergence (Alford, Hatemi, Hibbing, Martin, and Eaves 2011). Such findings have turned the political socialization literature on its head and have inspired a renewed interest in the area to reconcile these findings with the previous literature.

Univariate variance decomposition provides an initial partition of individual difference into a function of genetic, familial, or unique environmental influences. Knowing that attitudes, for example, are to a greater or lesser degree influenced by genes, familial environment or idiosyncratic personal experiences allows researchers to target their focus on the specific sources of variability on which the "social" process is working. However, variance decompositions of a single trait only represent the culmination of the total variance of all possible covariates on the trait. For example, Hatemi et al. (2007) provided evidence that 0.28 of the phenotypic variation in vote choice can be attributed to genetic variation. Taken alone, it would appear that even for a trait so vastly context-dependent and influenced by salient environmental conditions as voting, individual differences appear to be genetically influenced. However, when modeling attitudes, education, and other covariates in a multivariate analysis that simultaneously estimates the proportion of genetic, common

and unique environmental variance of multiple variables (Cholesky decomposition), it was found that *all* of the genetic variance on voting was accounted for by the genetic variance on attitudes about social welfare.

These types of multivariate models have become increasingly important in understanding the level at which relationships between traits exist and for attempting to identify causal pathways. For example, a great deal of literature considers personality to have a causal role in attitudinal formation (Jost, Glaser, Kruglanski, and Sulloway 2003). Such research was based on the assumption that personality was a deep-seated disposition and attitudes were more malleable. However, recent multivariate genetic models have shown that the vast bulk of covariance between personality and political attitudes is due to shared genetic influence (Verhulst, Hatemi, and Martin 2010). This finding allows for an alternative to the general theoretical assumption regarding the nature of the relationship between personality and attitudes; that is, that the relationship between personality and attitudes may not be causal but rather may derive from a common underlying latent genetic trait. The potential for relationships to be due to common underlying latent genetic variability is profoundly different from traditional explorations of causality and represents a fundamentally different approach to studying behavior, particularly when identifying the relationship between disposition traits (e.g., gender, personality, attitudes, cognition). In this way, variance decomposition models have begun to provide a platform to consider alternative pathways to explain the relationships between traits. The next step includes designs that explicitly model causation, accounting for genetic and environmental pathways, to differentiate between these competing theoretical assumptions.

Gene-Environment Interactions

Partitioning the independent effects of genes and the environment is only a first step in explicating the nature of a trait of interest. Virtually every socially relevant behavior is a complex function of both genetic and environmental influences. Thus, it is necessary to incorporate a method whereby genetic and environmental factors interact to moderate the behaviors individuals engage in. Broadly speaking, a gene-environment interaction occurs when genetic factors control the sensitivity to the environment (Kendler and Eaves 1986). Thus, the influence of an environmental factor on a behavior is conditioned by a person's genotype, or conversely, the genotype's effect is moderated by some environmental exposure.

Within a variance decomposition framework that focuses on the latent genetic effects at a population level, a "broad heritability-environment interaction" method (see Purcell 2002) can be used to explain how specific environmental influences alter the contributions of the genetic and environmental variation to a behavior within a population. This gene-environment (GxE) interaction model is a simple modification to the twin design earlier. Rather than modeling attitudes using the linear equation displayed in Figure 1, where the variance (V) of trait (t) is a function of genes (A^2) and environment ($C^2 + E^2$), a GxE twin model expresses the variance associated with each of the three components (ACE) as a linear interaction with the specific environment measured

$$(Vt = (A + \beta a * Event)^2 + (C + \beta c * Event)^2 + (E + \beta e * Event)^2).$$

In this equation, the β s are parameter estimates that moderate the impact of the variance components across different levels of exposure to exogenous events. Using this alteration of the twin model, genetic effects can be partitioned into a baseline or average

influences independent of the environment measured (analogous to an intercept in linear regression), and the marginal effect of the specific environmental event (i.e., slope).

Regarding political traits, so far only one exploration of this type of variance decomposition GxE has been presented in conference symposia, and none has been published in the literature. Hatemi (2010b) explored how life events moderate the genetic and environmental variance attributed to political attitudes (gene-environment interaction) and instances whereby an individual's genes lead them to select into certain environments that correlate with their attitudes (gene-environment covariance). Specifically, the genetic variance reported for certain economic attitudes, such as support for federal housing, was moderated by life events, such as losing one's job (Figure 2).

The findings from these types of models offer the potential for addressing long-standing questions in the social sciences regarding theories of self-interested rational action (Riker and Ordeshook 1968). Indeed, with regard to attitudes toward federal housing, self-interested rational action appears to offer a model theory. On the whole, individuals may be against federal housing but, once losing their job or home, opinions change to support federal assistance and, for the population, the genetic influence that accounts for attitudinal differences dissipates. Accordingly, whether individuals face economic hardship alters the influence of their genes on their attitudes toward federal housing. This pattern did not hold with regard to immigration attitudes: Genetic and environmental influences on opinions toward immigration were not moderated by an individual's employment status, even though immigration attitudes are equally malleable at the phenotypic level. It is important to note that these results offer only a novel first step and leave much to be explained. Why environmental factors moderate the impact of genes on some economic attitudes and not others and what this moderation means for the development of attitudinal theory remains an important question for future research to address.

It is important to note that the interplay of genes with the environment is incredibly complex, and a fundamental puzzle to disentangle is the origin of environmental exposure itself. So far, only a few of the models have been developed that explore this question. If genes motivate people to seek out certain environments or social situations and those environments influence political attitudes, the environments measured are no longer entirely exogenous, and it becomes difficult to quantify the genetic effect on a behavior or attitude separately from its environmental effect (see Moffitt, Caspi, and Rutter 2005; Purcell 2002). For example, Jockin, McGue, and Lykken (1996) found that people "select into" getting a divorce, in part based on their genetic disposition. In this way, the event of getting

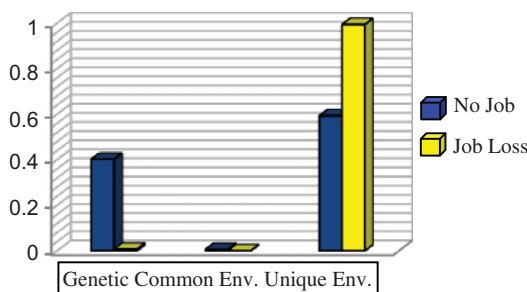


Figure 2. Change in heritability estimates of attitudes on *federal housing* as a function of *losing your job*. (color figure available online)

Note: Figure derived from Hatemi, 2010b.

a divorce could not be considered truly exogenous, and any gene-environment interaction with the event of getting divorced would be confounded. Undoubtedly, getting a divorce would be expected to affect attitudes about interpersonal relationship, procreation, and other politically relevant attitudes.

Limitations of Variance Decomposition

Twin modeling, as are all statistical techniques, is based on a series of assumptions, both theoretical and empirical (for a thorough review of limitations, see Medland and Hatemi 2009). The two assumptions of greatest concern are (1) whether monozygotic (MZ) and dizygotic (DZ) cotwin pairs are influenced to the same degree by their family environments and (2) whether parents are assortatively mating on the trait of interest. If twin environments with regard to the trait of interest are systematically dissimilar depending on twin type (MZ or DZ), the statistical assumption that the common (family) environment can be equated across twin types is violated, and the model will overestimate genetic influences. Alternatively, if parents select each other based on the trait of interest and the trait is genetically influenced, parents are more genetically similar on that trait; thus, DZ genetic similarity will be on average greater than the 0.5 assumed in the model, and results from twin models will then underestimate genetic influences.

Regarding political attitudes, all evidence points toward accepting the first critical assumption. That is, the familial environment equally influences political attitudes regardless of whether an individual is an identical (MZ) or fraternal (DZ) twin. In a direct test of equal environments using a longitudinal sample of children assessed for political attitudes every 2 years from ages 8 to 18 (the most critical periods of childhood socialization), Hatemi et al. (2009b) found that no dissimilarity in environmental influence existed by zygosity type for political attitudes. Furthermore, extended twin models, which included the nuclear family, estimated twin and sibling environments separately and controlled for assortative mating (Eaves and Hatemi 2008; Hatemi et al. 2010), found little evidence of a special twin environment regarding attitudes.

By contrast, the second assumption of random mating for political attitudes is routinely violated. Spouses are remarkably similar on political values and correlate for ideology at around 0.67 and for most attitudes between 0.4 and 0.6. This similarity in attitudes exists prior to marriage (Alford et al. 2011). As such, it appears twin models that do not account for assortative mating may actually be underestimating genetic influences. It is critical to note that only attitudes and ideology have been tested in this manner, and future tests for other political traits and behaviors are needed with regard to assortative mating and equal environments.

Several other statistical aspects of twin models also influence the estimation of the various models used in BG. First, twin studies are rarely, if ever, random samples. Thus, to generalize findings, replication is required. Additionally, sample sizes in twin and kinship models are often a concern, in some ways more so than in traditional social science studies. Most notably, the classic twin design treats the twin pair (or family) as the unit of analysis. Twin samples of fewer than thousands of pairs often lack the statistical power to reliably disentangle genetic and environmental sources of variance. Yet, in many twin studies, sample sizes range in the hundreds (and sometimes fewer). This does not mean those samples with only 500 twins are of no use but rather that the effect sizes must be correspondingly large to find a result, therefore greatly inflating the chances of a type II error. Thus, results from smaller sample sizes must be interpreted with caution and replicated because there is

great potential for sampling errors, and biases can be compounded in samples of related individuals (Neale, Eaves, and Kendler 1994).

Possibly the greatest limitation to twin models is ensuring their accurate interpretation. First, a heritability estimate does not mean that a trait “is” genetic. Rather, a 0.45 additive genetic estimate of abortion attitudes means that 0.45 of the variance of that trait within the population is accounted for by latent genetic influences. Second, estimates from twin models are population- and time-specific. For example, the underlying latent genetic trait that influences attitudes on the death penalty is what is being estimated, not genes for a specific attitude on the death penalty. The meaning and content of the terms *death penalty* have entirely different connotations in medieval Europe than they do in modern day America. Third, genetic influences are not fixed. There is never “a” gene for “an” attitude or any other complex trait for that matter. DNA may not change, but genetic expression is constantly changing, and the genes influencing a trait depend on the context and the accumulation of prior gene expression. One’s genetic expression is as dynamic as one’s life experiences, and genetic influence is far from deterministic. Finally, the latent additive genetic, common environmental, and unique environmental variance can easily be misrepresented. In a univariate model, additive genetic variance will include all the genetic influence from all covariates, some part of gene-environment covariation if it exists, and some part of gene-environment interaction, if it exists. Common environmental influence is also a latent trait and captures the overall effect of the family and social environment on the population, and is only a broad estimate. It can be further partitioned in numerous ways. Finally, the unique environment includes all idiosyncratic experiences, including hormones and nutrition introduced in the womb not shared with a co-twin, and measurement error. Thus, twin models, just like any regression model or correlation matrix, form a statistical estimate that simplifies reality into smaller, more digestible pieces that we can begin to explore, but are far from a perfect representation of the intricacies of the real world.

Identifying the Specific Genetic Markers and Neurobiological Pathways

The variance decomposition and familial models noted earlier have provided important leverage in identifying broad latent pathways of familial transmission and in highlighting the general architecture of genetic and environmental sources of influence for political traits. However, molecular DNA is required to identify the specific neurobiological systems that underlie the genetic variance from heritability estimates. This area is more recent than twin and familial studies, and advances in technology, methodology, and data availability are being continually realized. So far, there are few genetic markers that have been associated with any behavioral outcomes or complex traits that have withstood replication, none of which involve politically relevant traits. Nevertheless, association studies are a valuable step in identifying pathways from genes to complex political behaviors and preferences. Regarding political traits, two basic approaches for identifying genes have been undertaken. The first exploits *a priori* information that suggests a certain gene might be expected to be associated with a specific trait. The second method scans the entire genome for a genetic marker or region that is significantly related to the trait of interest. We detail these methods next and summarize the research undertaken so far in political science.

Candidate Gene/Marker Studies

The goal of a candidate gene association study is to identify within a population whether the incidence of a specific version of a gene, most often an allele, is more common than

would be expected due to chance among individuals exhibiting a particular trait. Candidate genes are typically selected on the basis of previous associations with behaviors that are conceptually related to the trait of interest or because of theoretical expectations about how an environment might affect a genetic predisposition for a trait. Similar to twin studies, the moderating effect of environmental factors on genetic differences may also be studied within the context of an association study.

There are two main research designs typically employed in candidate gene association studies: case-control designs and family-based designs. Unlike variance decomposition, both methods use techniques familiar to the social sciences. Case-control designs compare the frequency of alleles or genotypes among subjects that exhibit a trait of interest (e.g., voting) to subjects who do not and test the null hypothesis of no association. This can be done using a Pearson or Fisher test, analysis of variance, or regression. The second approach, a family-based test, compares whether offspring who exhibit the trait of interest (e.g., vote) receive an allele from their parents more often than would be expected by chance. This second method reduces the probability of false-positive signals that may arise from what is known as *population stratification*. Population stratification occurs when people have different allele frequencies due to their genetic ancestry. The exhibited trait in these groups may be the result of environmental forces, alleles other than the one of interest, or by some unobserved factor. Once these groups mix in a larger population, simply comparing the frequency of an allele among cases and controls would lead to a spurious association (for a review of the statistical methods, see Balding 2006).

Research in this area is relatively new, and candidate gene studies have been limited to identifying specific genetic variants that are associated with voter turnout and partisan attachment. In both cases, the serotonergic and dopaminergic pathways have been implicated. These pathways are known to influence a wide array of social behaviors, including anxiety disorders and neuroticism (Lesch et al. 1996; Lesch and Mossmer 1998), novelty-seeking behavior (Kluger, Siegfried, and Ebstein 2002; Savitz and Ramesar 2004; Schinka, Letsch, and Crawford 2002), and attention disorders (D'Souza et al. 2004; McCracken et al. 2000). The working hypothesis was that genes that regulate these neural systems are likely related to these political traits as pro-social behaviors and attitudes underlie political behaviors such as turnout and partisan attachment.

For example, the MAOA gene controls the production of the enzyme MAOA that breaks down serotonin in the brain. A specific variant of the MAOA gene that is associated with suboptimal levels of serotonin in the brain has been shown to be related to social stress and antisocial behavior. Because pro-social attitudes and behaviors have been shown to positively influence the likelihood of voting (Edlin, Gelman, and Kaplan 2007; Fowler 2006; Jankowski 2007; Knack 1991), Fowler and Dawes (2008) tested whether MAOA was related to self-reported voter turnout and found evidence of a significant association. In addition, a variant of the ANKK1 gene, part of the dopamine pathway, has been found to be associated with the formation of social attachments and generalized feelings of social alienation. However, the precise biological mechanisms linking ANKK1 and social attachment are not yet well understood. As a general willingness to form ties with social groups likely underlies whether individuals join political parties (Green, Palmquist, and Schickler 2002), Dawes and Fowler (2009) hypothesized that ANKK1 would be significantly associated with an individual's propensity to identify with a political party. The authors found evidence consistent with this hypothesis.

These association studies give preliminary support to the conjecture that genes affect turnout and partisan attachment because they influence pro-social behaviors and attitudes that foster cooperation. More broadly, these findings push political scientists to think

more critically about the social component of political behaviors. From an evolutionary perspective, there would have been no selective pressure acting on specific political orientations or actions. However, it is possible to imagine that cooperative behaviors more generally did confer advantage on our ancestors to varying degrees. Considering these more proximal psychological aspects of participation expands our understanding of what participation means. The next step for political scientists is to formally test this conjecture and other possible causal pathways using a mediation model.

Candidate Gene x Environment Interactions

Complex traits, such as political preferences, are the result some combination and interaction of environmental, social, gene, hormonal, neurological, and a host of other neurobiological mechanisms. No one model can fully account for these influences, and the methodological techniques available to explore each individually remain imperfect. However, one approach, gene-environment interaction, has the potential to provide great insight into the “black box” of political preferences. As with twin models discussed earlier, researchers are ultimately interested in better understanding the causal pathways through which genetic variants identified by association studies influence political behaviors and attitudes. However, unlike the work using twin and kinship models, which estimate how environments might moderate the *latent* genetic and environmental variance attributed to political attitudes, candidate gene by environment interaction studies seek to identify the interactions between *specific* genetic variants and specific environmental stimuli are associated with differences in behaviors. Similar to the candidate gene methods described earlier, regression-based techniques are used to test the significance of interaction terms between specific genetic marker and specific environmental stimuli (see Abecasis, Cardon, and Cookson 2000a, 2000b; Fulker, Sham, and Hewitt 1999; Gauderman 2003).

Research in this area remains limited; however, recent papers in conference symposia have begun to emerge regarding the effects of the political context. Continuing with the preceding findings, the serotonergic and dopaminergic pathways are of particular interest. For example, Settle, Dawes, and Loewen (2010) argue that a variant of the 5HTT gene moderates the influence of living in partisan heterogeneity and that people with the short version of the allele are more likely to disengage from politics when confronted with a diversity of opinions in their neighborhood. This finding is consistent with other research that suggests that those who regulate the uptake of excess serotonin less efficiently are more sensitive to social stress (Stein, Schork, and Gelernter 2008). These people may be more likely to select out of politics when confronted with stress, as they will be less able to successfully manage the contention of politics, arrive at a vote decision, and act on that decision. Should this finding be replicated and verified, this knowledge has profound implications for our theory of why competition should increase turnout.

Other work has focused on attitude and ideology. Political scientists have long been interested in the effects of social networks on political attitudes (Huckfeldt, Johnson, and Sprague 2004; Mutz 2002,). This work did not consider the possibility that there may be individual heterogeneity in response to these network environments. However, exposure to diverse viewpoints in adolescence, as measured by the number of self-reported friendships, enhances a liberal political identification for those people who carry the DRD4-R7 allele, who may be genetically predisposed to be novelty-seekers (Settle et al. 2010). People with the R7 allele are no more likely to have a large number of friends than those without it, but the effect of this exposure has a differential effect on the development of their ideology. This finding has important implications for our theory of ideology. As ideology

has stable, genetic components (Eaves and Hatemi 2008), we are also reminded of how genes regulate ideological adoption or change by affecting the way that we interpret and incorporate environmental exposures into our ideological framework.

The ability to accurately estimate the importance of one's genotype in determining sensitivity to environmental factors has only just begun to provide greater understanding of differences in political behaviors and attitudes. As much of political science is concerned with environments outside immediate personal exposures, one of the major potential contributions of the social sciences to behavior genetics is to offer better theory and measurement for why broader social and political experiences should work in conjunction with genes to explain social behavior more generally. Fifty years of work in political science focusing both on the effects of personal social influences and broader sociopolitical context offer many potential interactions to explore, such as interactions between genes and media exposure, campaign mobilization, neighborhood demographics, and electoral rules. Political scientists have begun to lead the way in studying these interactions and, similar to epidemiological studies, are blessed with rich data on environmental exposures. So far, this understanding has remained limited to the individual; however, the potential to estimate aggregate-level effects may provide important guidance to researchers and policymakers as they seek to design environments to improve conditions for citizens.

Genome-wide Analyses

The second approach to identify specific genetic markers, for which appropriate data have only recently become available for the study of political traits, is to systematically scan the entire genome for markers significantly related to the political traits of interest. The processes by which individual genes or groups of genes could be indirectly influencing political behaviors are unknown, and the major benefit of genome-wide approaches is not to find "a" gene "for" a given trait but rather to identify *previously unknown* biological processes and pathways related to the development and maintenance of political orientations. In this way, they are not constrained by prior hypotheses (Pearson and Manolio 2008). Two basic approaches have been used: genome-wide linkage, which requires a large sample of related individuals, and genome-wide association, which relies on a sample of unrelated individuals. The human genome is remarkably identical for all people, but small portions vary from person to person. These polymorphisms make it possible to identify genomic regions inherited in conjunction with the trait of interest.

Regarding political behaviors, only one published study so far has taken this approach. Hatemi et al. (2011) used genome-wide linkage, an exploratory method that identifies large chromosomal regions statistically significant in predicting a trait value, and found four significant or suggestive regions for a liberalism-conservatism scale of attitudes. In each of the regions, glutamate, NMDA, dopamine, and serotonin receptors were located on or near the linkage peaks. This finding was important because neurotransmitters for NMDA and serotonin have been significantly associated with a wide array of behavioral traits with specific involvement in the regulation of cognition and emotion and regulation of fear, stress, and anxiety (Canli and Lesch 2007; Dai et al. 2008; Hariri et al. 2002; Hariri and Holmes 2006). That is, the genome-wide linkage study identified potential candidate genes for political attitudes that are broadly consistent with deeper psychological mechanisms such as fear, anxiety, cognition, and emotion and have opened the door to explore new pathways toward attitude formation.

However, linkage is better suited to detect genes with large effects, and because individual genetic effects are very small for complex traits, more powerful genome-wide

association scans (GWASs) are preferred. GWASs represent a revolutionary advancement beyond linkage and candidate gene studies because they take advantage of high-throughput genotyping techniques to investigate hundreds of thousands of variants across the entire human genome in samples of unrelated individuals to identify specific genes that may be responsible for the trait of interest. This type of analysis requires a dense set of markers that capture a substantial proportion of common variation across the genome. They compare a more “complete” DNA of people on a polymorphism-by-polymorphism basis, rather than a regional approach, and offer a more powerful alternative to linkage that is often underpowered to detect genes with small effects and a much more robust test than candidate gene studies that are biased by the choice of genes included (Baum et al. 2008). However, due to the simultaneous exploration of hundreds of thousands of markers, a GWAS requires large sample sizes and expensive genotyping, and it demands an extremely high threshold for significance: $p < 5 \times 10^{-8}$ or better. GWAS studies for political traits are now underway and should begin producing results in the near future.

Limitations of Candidate Marker and Genome-wide Studies

Molecular genetic studies rely on specific genetic markers rather than estimates of overall latent genetic effects. This approach presents a number of unique considerations and limitations. First, DNA sampling requires a more invasive protocol than typical of most social science studies. Blood or saliva must be collected, and researchers must have training in molecular biology and be cognizant of the additional ethical considerations, subject protections, data safety, and sample collection protocols necessary for dealing with biological specimens, as well as access to proper processing, genotyping, storage facilities and methodological expertise. Furthermore, the funding mechanisms in the social sciences for these additional expenses are limited. In addition, common gene variants that have so far been implicated in social traits are responsible for only a very small fraction of the genetic variation that we know exists. For example, GWASs have been able to account only for some 11 per cent of the genetic variance found by heritability studies for height. Future studies that utilize gene expression methods and account for rare variants have the potential to alleviate some of the missing heritability concerns, but the findings so far caution against any one gene or group of genes being identified that account for the bulk of the genetic variance on any political or social trait.

Each method also contains additional shortcomings and limitations. The genome is extremely large and complex, and what appears to be a significant causal relationship between a genetic variant and a trait of interest may be due to chance. Statistical evidence for an association between an allele and a phenotype comes from one of three situations: (1) The allele itself might be functional and directly affect expression of the trait, (2) the allele might be correlated with another causal allele, and (3) the association could be attributable to chance, artifact of the sample, confounding¹ or selection bias (e.g., population stratification). Of greatest concern is population stratification. The classic example by Knowler, Williams, Pettitt, and Steinberg (1988) showed that a failure to adjust for confounding by population stratification produces a spurious association between variants in an immunoglobulin haplotype and diabetes among residents of the Gila River Indian Community. The association was not causal but instead reflected confounding

¹If unaccounted subpopulations exist within the studies defined groups and if they differed in both allele frequencies, ignoring these differences in a candidate gene studies would lead to confounding.

by a population-stratifying factor: the degree of Caucasian heritage of the individuals in the Gila River Indian Community. In other words, controlling only for self-reported ethnicity resulted in a spurious association. Certain statistical controls such as family-based designs are now preferred to alleviate population stratification concerns, whereas other genomic controls or structured association methods correct for chance association (Balding 2006; for a review of these and other controls, see Cardon and Palmer 2003). In addition, a false-positive may arise due to the fact that candidate gene studies tend to test several variants, possibly only reporting a subset of them, without correcting for this fact. Similarly, when conducting a large number of hypothesis tests, some significant associations will be found that are purely the result of sampling variation. One way to account for this is to use a Bonferroni correction that divides the p -value by the number of tests performed.

So far, very few candidate genetic marker studies have survived long-term replication, and fewer still are validated by genome-wide approaches, which suggest that many significant results may be an artifact of the specific sample being studied rather than a genuine relationship. This is especially important for political science studies focusing on genes related to the dopamine and serotonin systems, as recent meta-analyses have shown that findings for many of these genes may not be as strong as previously believed (Munafo et al. 2009a; Munafo, Durant, Lewis, and Flint 2009b; Munafo, Yalcin, Willis-Owen, and Flint 2008).

A similar concern is that studies that use the exact same genetic markers have been coded in different ways, achieving significance in their respective studies (Freese 2010). For example, the Taq1a variant of ANKK1, part of the dopamine pathway, comes in three forms: TT, TC, and CC. Three studies that used the same AddHealth data coded Taq1a in three different ways. For its relationship with partisanship and voting, Dawes and Fowler (2009) coded the SNP as the number of C alleles (0, 1, and 2). For educational attainment, Shanahan et al. (2008) coded the SNP as C/C versus the combination of TC and TT; and for delinquency, Guo, Roettger, and Cai (2008) coded the SNP as T/C versus the combination of CC and TT. All coding schemes may be valid, as the function of specific SNPs, combination of SNPs, and neurobiological pathways remain largely under-informed and may be different for different traits. However, the lack of a uniform coding scheme makes it difficult to ensure accurate replication and interpretation.

In addition to concerns about spurious associations, there are modeling assumptions made in association studies that must be properly addressed. By methodological design, most studies assume that a single genetic variant influences a behavior or attitude *additively* or independently. That is, candidate gene studies capture only a single polymorphism for a single gene even though it is known that it is unlikely that genes act independently. Rather, there are at least hundreds of genes that influence any given neurobiological pathway, and complex behaviors and attitudes are polygenic, and numerous gene-by-gene associations or interactions (epistasis) may exist that influence a political behavior. This problem is compounded by the need to correct for multiple hypothesis testing as each interaction constitutes an additional test, and the power to detect interactions is much lower than the power to detect main effects. Therefore, including gene-gene interactions will require very large samples to attain a reasonable level of power.

GWASs alleviate some of the concerns found in candidate gene studies. However, as noted, the statistical requirement for a positive association is incredibly strict, and it is quite possible that many genetic markers that are in fact significant are being deemed insignificant. Thus, though candidate gene studies may be over-reporting significance, GWA may be underreporting significance.

Furthermore, similar to twin models, proper interpretation of results is important. For instance, in our preceding example, Settle, Dawes, Christakis, and Fowler (2010) reported that people with 10 friends who have two copies of the 7R allele of DRD4 would have the effect of increasing ideology in the liberal direction by about 40 per cent (on a five-point scale) versus those who do not have two copies of 7R allele but also have 10 friends. A result of this magnitude due to a single variant on a single dopamine receptor is unlikely, if not impossible. However, it is entirely possible to interpret the finding that the 7R allele of DRD4 changes one's ideology by 40 per cent if he or she has many friends. Unfortunately, this is exactly how the results were explained to the public by *Fox News* ("Researchers Find the 'Liberal Gene'"; see Kaplan 2010). This is not to say the analyses were incorrect; rather, the estimates are simply the result of the statistical method and assumptions in the model. In both candidate gene studies, effect sizes are often inflated due to the statistical model. This is of greater concern in small samples and when studies include subjects of different ethnic and racial ancestry into a single sample or genotype only a limited number of biomarkers.

Possibly the greatest concern in candidate gene studies are the false-positives reported in candidate gene-environment interaction studies. Studies that find no main effect but find an interaction do not tend to survive long-term replication. Specifically, finding a significant allelic GxE interaction, but no significant relationship between the genetic marker and the trait of interest (no main effect), suggests the unlikely scenario of a risk environment for one allele's being the protective environment for the other. Regarding the most celebrated of GxE studies, Caspi et al. (2003) found that childhood maltreatment predicted adult-diagnosed depression among individuals carrying at least one copy of the "short" allele of the promoter region of the 5-HTTLPR serotonin transporter. They also found that people with one or more short alleles who were exposed to stressful life events were more likely to develop depression than those homozygous for the long allele. However, the Risch et al. (2009) meta-analysis of more than 14,000 individuals provided evidence that Caspi's findings were not supported.

Conclusion

In this paper, we reviewed the last 5 years of scholarship focused on the study of the genetic influences of political behaviors and attitudes through the main behavioral genetics approaches to study behavior, to include their limitations. The foresight of the geneticists and psychologists who collected relevant political phenotypes decades before political scientists realized their value opened the door for this research. However, those most invested in politics, and who are principally interested in the dynamic and multifaceted nature of the social environment and the mechanisms that link context with the immediate biological substrate, have now taken the lead in the genetics of political and social traits. Remarkably, in just a few short years, political scientists have far surpassed the original behavioral genetics studies that explored political traits.

In doing so, it is not simply that the genetics of political behaviors are being better understood but rather that political behavior is becoming more fully understood. New pathways that link proximal psychological mechanisms informed by genetic differences, such as cognition and emotion, are being identified. In addition, the limits of environmental predictors are being realized. Though it remains quite early in this research program, additional methods and improvements in methodology in each of these domains is continually progressing and offer exciting analyses to further explore genetic influences. Future studies, which begin to unpack gene-environment relationships by including rare variants, measuring gene expression, or incorporating gene-gene interactions will undoubtedly

further this endeavor. The potential of experiments to generate both field and laboratory data is equally promising. Such exploration allows the possibility to further control and refine the nature of the intersecting relationships between genes, environmental stimuli, and relevant political behaviors and preferences.

Only by considering both the environmental and genetic sources of individual differences can we gain a deep understanding of behavior. The more we learn about how genes lead us into environments, affect our interpretations of the exogenous environments we encounter, and how our social environments may change our genetic expression, the more we can contribute to the discipline at large about which environments matter and why.

References

- Abecasis, G., L. Cardon, and W. Cookson. 2000a. A general test of association for quantitative traits in nuclear families. *Am J Hum Genet* 66:279–292.
- Abecasis, G., W. Cookson, and L. Cardon. 2000b. Pedigree tests of transmission disequilibrium. *Eur J Hum Genet* 8:545–551.
- Alford J. R., P. K. Hatemi, J. R. Hibbing, N. G. Martin, and L. J. Eaves. 2011. The politics of mate choice. *J Politics* (forthcoming).
- Alford, J., C. Funk, and J. Hibbing. 2005. Are political orientations genetically transmitted? *Am Political Sci Rev* 99:153–167.
- Balding, D. 2006. A tutorial on statistical methods for population association studies. *Nat Rev Genet* 7:781–791.
- Baum, A. E., M. Hamshere, E. Green, S. Cichon, M. Rietschel, N. M. Noethen, N. Craddock, and F. J. McMahon. 2008. Meta-analysis of two genome-wide association studies of bipolar disorder reveals important points of agreement. *Mol Psychiatry* 13(5):466–467.
- Boardman, J., C. Blalock, and F. Pampel. 2010. Trends in the genetic influences on smoking. *J Health Soc Behav* 51(1):108–123.
- Boardman, J. D., C. L. Blalock, F. Pampel, P. K. Hatemi, A. Heath, and L. Eaves. 2010. Period differences in the genetics of smoking desistence. *Demography* (forthcoming).
- Bouchard, T. J. Jr., and M. McGue. 2003. Genetic and environmental influences on human psychological differences. *J Neurobiol* 54(1):4–45.
- Campbell, A., P. E. Converse, W. E. Miller, and D. E. Stokes. 1960. *The American voter*. Chicago: University of Chicago Press.
- Canli, T., and K.-P. Lesch. 2007. Long story short: The serotonin transporter in emotion regulation and social cognition. *Nat Neurosci* 10(9):1103–1109.
- Cardon, L. R., and L. J. Palmer. 2003. Population stratification and spurious allelic association. *Lancet* 361(9357):598–604.
- Carey, B. 2005. Some politics may be etched in the genes. *New York Times*, <http://www.nytimes.com/2005/06/21/science/21gene.html?scp=1&sq=Some%20politics%20may%20be%20etched%20in%20the%20genes&st=cse> (accessed March 21, 2011).
- Caspi, A., K. Sugden, T. E. Moffitt, A. Taylor, I. W. Craig, H. Harrington, J. McClay, J. Mill, J. Martin, A. Braithwaite, et al. 2003. Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science* 301:386–389.
- Dai, J.-X., H.-L. Han, M. Tian, J. Cao, J.-B. Xiu, N.-N. Song, Y. Huang, T.-L. Xu, Y.-Q. Ding, et al. 2008. Enhanced contextual fear memory in central serotonin-deficient mice. *Proc Natl Acad Sci U S A* 105(33):11981–11986.
- Dawes, C. T., and J. H. Fowler. 2009. Partisanship, voting, and the dopamine D2 receptor gene. *J Politics* 71(3):1157–1171.
- D'Souza, U. M., C. Russ, E. Tahir, J. Mill, P. McGuffin, P. J. Asherson, and I. W. Craig. 2004. Functional effects of a tandem duplication polymorphism in the 5-prime flanking region of the DRD4 gene. *Biol Psychiatry* 56:691–697.

- Eaves, L. J., and R. Carbonneau. 1998. Recovering components of variance from differential ratings of behavior and environment in pairs of relatives. *Dev Psychol* 34(1):125–129.
- Eaves, L. J., and H. Eysenck. 1974. Genetics and the development of social attitudes. *Nature* 249:288–289.
- 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. 2008. Transmission of attitudes toward abortion and gay rights: Parental socialization or parental mate selection? *Behav Genet* 38:247–256.
- Eaves, L. J., A. Heath, N. Martin, H. Maes, M. Neale, K. Kendler, K. 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 Res Hum Genet* 2(2):62–80.
- Edlin, A., A. Gelman, and N. Kaplan. 2007. Voting as a rational choice. *Rational Soc* 19(3):293–314.
- Falconer, D. S. 1960. *Introduction to quantitative genetics*. Edinburgh: Oliver and Boyd.
- Fowler, J. 2006. Altruism and turnout. *J Politics* 68(3):674–683.
- Fowler, J. H., A. Baker, and C. T. Dawes. 2008. Genetic variation in political participation. *Am Political Sci Rev* 102:233–248.
- Fowler, J. H., and C. T. Dawes. 2008. Two genes predict voter turnout. *J Politics* 70(3):579–594.
- Fowler, J. H., and D. Schreiber. 2008. Biology, politics, and the emerging science of human nature. *Science* 322(5903):912–914.
- Freese, J. 2010. The integration of genetic data into social inquiry: The cautionary tale of Taq1a. Paper presented at the Gene-Environment Research Initiative project, Penn State University, State College, Pennsylvania, Nov 15.
- Fulker, D., S. Cherny, P. Sham, and J. Hewitt. 1999. Combined linkage and association sib-pair analysis for quantitative traits. *Am J Hum Genet* 64:259–267.
- Gauderman, W. J. 2003. Candidate gene association analysis for a quantitative trait, using parent-offspring trios. *Genet Epidemiol* 25(4):327–338.
- Green, D., B. Palmquist, and E. Schickler. 2002. *Partisan hearts and minds*. New Haven, CT: Yale University Press.
- Guo, G., M., E. Roettger, and T. Cai. 2008. The integration of genetic propensities into social-control models of delinquency and violence among male youths. *Am Sociol Rev* 73:543–568.
- Hariri, A. R., and A. Holmes. 2006. Genetics of emotional regulation: The role of the serotonin transporter in neural function. *Trends Cogn Sci* 10(4):182–191.
- Hariri, A. R., V. S. Mattay, A. Tessitore, B. Kolachana, F. Fera, D. Goldman, M. F. Egan, and D. R. Weinberger. 2002. Serotonin transporter genetic variation and the response of the human amygdala. *Science* 297(5580):400–403.
- Hatemi, P. K. 2010a. Genetic and neurocognitive approaches for comparative politics: A partnership between science and culture. *APSA-CP Newsletter Symposium on Politics and the Brain* 21(1): 6–12.
- Hatemi, P. K. 2010b. An exploration of gene and environment interaction: The influence of major life events on political attitudes. Presented at the Midwest Political Science Association Annual Meeting, 2010.
- Hatemi P. K., et al. 2011. Genome-wide analysis of political attitudes. *J Politics* (forthcoming).
- Hatemi, P. K., J. Alford, J. Hibbing, M. Keller, N. Martin, S. Medland, and L.J. Eaves. 2010. Not by twins alone: Using the extended twin family designed to investigate the genetic basis of political beliefs *Am J Political Sci* 54(3):798–814.
- Hatemi, P. K., J. R. Alford, J. R. Hibbing, N. G. Martin, and L. J. Eaves. 2009a. Is there a ‘party’ in your genes? *Political Res Q* 62(3):584–600.
- Hatemi, P. K., S. E. Medland, and L. J. Eaves. 2009b. Genetic sources for the gender gap? *J 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. *Behav Genet* 37:435–448.
- Hatemi, P. K., C. L. Funk, H. Maes, J. Silberg, S. E. Medland, N. G. Martin, and L. J. Eaves. 2009b. Genetic influences on political attitudes over the life course. *J Politics* 71(3): 1141–1156.

- Holzinger, K. 1929. The relative effect of nature and nurture influences on twin differences. *J Educ Psychol* 20:241–248.
- Huckfeldt, R. R., P. E. Johnson, and J. Sprague. 2004. *Political disagreement: The survival of diverse opinions within communication networks*. New York: Cambridge University Press.
- Jankowski, R. 2007. Altruism and the decision to vote: Explaining and testing high voter turnout. *Rational Soc* 14(1):55–77.
- Jennings, M., L. Stoker, and J. Bowers. 2009. Politics across generations: Family transmission reexamined. *J Politics* 71(3):782–799.
- Jockin, V., M. McGue, and D. T. Lykken. 1996. Personality and divorce: A genetic analysis. *J Personal Soc Psychol* 71(2):288–299.
- Jost, J. T., J. Glaser, A. W. Kruglanski, and F. J. Sulloway. 2003. Political conservatism as motivated social cognition. *Psychol Bull* 129(3):339–375.
- Kaplan, J. A. 2010. Researchers Find the ‘Liberal Gene.’ FoxNews.com Science: October 28. <http://www.foxnews.com/scitech/2010/10/28/researchers-liberal-gene-genetics-politics/> (accessed March 21, 2011).
- Kendler, K. S., and L. J. Eaves. 1986. Models for the joint effect of genotype and environment on liability to psychiatric illness. *Am J Psychiatry* 143:279–289.
- Kluger, A. N., Z. Siegfried, and R. P. Ebstein. 2002. A meta-analysis of the association between DRD4 polymorphism and novelty seeking. *Mol Psychiatry* 7(7):712–717.
- Knack, S. 1991. Social altruism and voter turnout: Evidence from the 1991 NES Pilot Study. National Election Studies working paper.
- Knowler, W. C., R. C. Williams, D. J. Pettitt, and A. G. Steinberg. 1988. Gm and type 2 diabetes mellitus: An association in American Indians with genetic admixture. *Am J Hum Genet* 43:520–526.
- Lesch, K. P., D. Bengel, A. Heils, S. Z. Zabol, B. D. Greenberg, S. Petri, J. Benjamin, C. R. Muller, D. H. Hamer, and D. L. Murphy. 1996. Association of anxiety-related traits with a polymorphism in the serotonin transporter gene regulatory region. *Science* 274(5292):1527–1531.
- Lesch, K. P., and R. Mossner. 1998. Genetically driven variation in serotonin uptake: Is there a link to affective spectrum, neurodevelopmental, and neurodegenerative disorders? *Biol Psychiatry* 44(3):179.
- Martin, N. J. 1987. Genetic differences in drinking habits, alcohol metabolism and sensitivity in unselected samples of twins. In *Genetics and alcoholism*. eds. H. W. Goedde and D. P. Agarwal. New York: Alan R. Liss, 109–119.
- Martin, N. G., L. J. Eaves, A. C. Heath, R. Jardine, L. M. Feingold, and H. J. Eysenck. 1986. Transmission of social attitudes. *Proc Natl Acad Sci U S A* 83:4364–4368.
- McCracken, J. T., S. L. Smalley, J. J. McGough, L. Crawford, M. Del’Homme, R. M. Cantor, A. Liu, and S. F. Nelson. 2000. Evidence for linkage of a tandem duplication polymorphism upstream of the dopamine D4 receptor gene (DRD4) with attention deficit hyperactivity disorder (ADHD). *Mol Psychiatry* 5:531–536.
- McDermott, R., D. Tingley, J. Cowden, G. Frazzetto, and D. D. P. Johnson. 2009. Monoamine oxidase A gene (MAOA) predicts behavioral aggression following provocation. *Proc Natl Acad Sci* 106(7):2118–2123.
- Medland, S. E., and P. K. Hatemi. 2009. Political science, biometric theory, and twin studies: A methodological introduction. *Political Anal* 17(2):191–214.
- Moffitt T. E., A. Caspi, and M. Rutter. 2005. Strategy for investigating interactions between measured genes and measured environments. *Arch Gen Psychiatry* 62:473–481.
- Munafò, M., N. Freimer, W. Ng, R. Ophoff, J. Veijola, J. Miettunen, M. Jarvelin, A. Taanila, and J. Flint. 2009a. 5-HTTLPR genotype and anxiety-related personality traits: A meta-analysis and new data. *Am J Med Genet Part B* 150B:271–281.
- Munafò, M., C. Durrant, G. Lewis, and J. Flint. 2009b. Gene x environment interactions at the serotonin transporter locus. *Biol Psychiatry* 65:211–219.
- Munafò, M., B. Yalcin, S. Willis-Owen, and J. Flint. 2008. Association of the dopamine D4 receptor (DRD4) gene and approach-related personality traits: Meta analysis and new data. *Biol Psychol* 63:197–206.

- Mutz, D. C. 2002. The consequences of cross-cutting networks for political participation. *Am J Political Sci* 46(4):838–855.
- Neale, M. C., and L. L. Cardon. 1992. *Methodology for genetic studies of twins and families*. Norwell, MA: Kluwer Academic Publishers.
- Neale, M. C., L. J. Eaves, and K. S. Kendler. 1994. The power of the classical twin study to resolve variation in the threshold traits. *Behav Genet* 24(3):239–258.
- Niemi, R. G., and M. K. Jennings. 1991. Issues and Inheritance in the formation of party identification. *Am J Political Sci* 35(4):970–988.
- Olson, J. M., P. A. Vernon, J. A. Harris, and K. L. Jang. 2001. The heritability of attitudes: A study of twins. *J Personal Soc Psychol* 80(6):1–4.
- Pearson, T., & T. Manolio. 2008. How to interpret a genome-wide association study. *JAMA* 299(11):1335–1344.
- Posner, S. F., L. Baker, A. Heath, and N. G. Martin. 1996. Social contact, social attitudes, and twin similarity. *Behav Genet* 26(2):123–133.
- Posthuma, D., and D. I. Boomsma. 2000. A note on the statistical power in extended twin designs. *Behav Genet* 30:147–158.
- Posthuma, D., A. L. Leo Beem, E. J. C. de Geus, G. C. M. van Baal, J. B. von Hjelmberg, I. Iachine, and D. I. Boomsma. 2003. Theory and practice in quantitative genetics. *Twin Research* 6(5):361–376.
- Purcell, S. 2002. Variance components models for gene–environment interaction in twin analysis. *Twin Res* 5:554–571.
- Riker, W. H., and P. C. Ordeshook. 1968. A theory of the calculus of voting. *Am Political Sci Rev* 62(1):25–42.
- Risch, N., R. Herrell, T. Lehner, K.-Y. Liang, L. Eaves, J. Hoh, A. Griem, M. Kovacs, J. Ott, and K. R. Merikangas. 2009. Interaction between the serotonin transporter gene (5-HTTLPR), stressful life events, and risk of depression. *JAMA* 301(23):2462–2471.
- Savitz, J. B., and R. S. Ramesar. 2004. Genetic variants implicated in personality: A review of the more promising candidates. *Am J Med Genet (Neuropsychiatr Genet)* 131B(1):20–32.
- Schinka, J. A., E. A. Letsch, and F. C. Crawford. 2002. DRD4 and novelty seeking: Results of meta analyses. *Am J Med Genet (Neuropsychiatr Genet)* 114(6):643–648.
- Settle, J., C. Dawes, N. Christakis, and J. Fowler. 2010. DRD4 moderates a relationship between adolescent friendships and ideology. *J Politics* 72(4):1189–1198.
- Settle, J., C. T. Dawes, and P. J. Loewen. 2010. Partisan heterogeneity and 5HTT: A GxE effect on voter turnout. Paper presented at the 68th Midwest Political Science Association Conference Annual Meeting, Chicago, Illinois, April 22–25, 2010.
- Shanahan, M. J., S. Vaisey, L. D. Erickson, and A. Smolen. 2008. Environmental contingencies and genetic propensities: social capital, educational continuation, and dopamine receptor gene DRD2. *Am J Sociol* 114:S260–S286.
- Sigelman, L. 2006. Report of the editor of the *American Political Science Review*, 2004–5. *PS: Political Sci Politics* 40:171–173.
- Stein, M. B., N. J. Schork, and J. Gelernter. 2008. Gene-by-environment (Serotonin transporter and childhood maltreatment) interaction for anxiety sensitivity, an intermediate phenotype for anxiety disorders. *Neuropsychopharmacology* 33:312–319.
- Sturgis, P., S. Read, P. K. Hatemi, G. Zhu, T. Trull, M. J. Wright, and N. G. Martin. 2010. A genetic basis for social trust. *Political Behav* 32(2):205–230.
- Truett, K. R., L. J. Eaves, J. M. Meyer, A. C. Heath, and N. G. Martin. 1992. Religion and education as mediators of attitudes: a multivariate analysis. *Behav Genet* 22(1):43–62.
- Truett, K. R., L. J. Eaves, E. E. Walters, A. C. Heath, J. K. Hewitt, J. M. Meyer, J. Silberg, M. C. Neale, N. G. Martin, and K. S. Kendler. 1994. A model system for analysis of family resemblance in extended kinships of twins. *Behav Genet* 24(1):35–49.
- Verhulst, B., P. K. Hatemi, and N. G. Martin. 2010. The nature of the relationship between personality traits and political attitudes. *Personal Indiv Diff* 49(4):306–316.

Appendix

Scale	a^2	c^2	d^2	e^2	Authors	Estimation Technique	Year	Population
Political Conservatism (Females)	0.28	0.30	—	0.42	Truett et al (1992)	ML	1980	Australia
Political Conservatism (Males)	0.30	0.21	—	0.48	Truett et al (1992)	ML	1980	Australia
Left-Right Scale	0.32	0.16	—	0.53	Alford et al (2005)	Corr.	1988	Virginia and National AARP
Political Conservatism (Females)	0.32	0.11	0.13	0.37	Eaves et al (1999)	ML – Assort.	1988	Virginia and National AARP
Political Conservatism (Males)	0.58	0.02	0.07	0.40	Eaves et al (1999)	ML – Assort.	1988	Virginia and National AARP
General Conservatism (Females)	0.24	0.42	—	0.34	Verhulst et al (2010)	ML	1990	Australia
General Conservatism (Males)	0.21	0.41	—	0.38	Verhulst et al (2010)	ML	1990	Australia
Partisan Intensity	0.31	0.17	—	0.52	Hatemi et al (2009)	Corr.	1988	Virginia and National AARP
Party Id	0.07	0.76	—	0.17	Hatemi et al (2009)	Corr.	1988	Virginia and National AARP

(Continued)

Appendix (Continued)

Scale	a ²	c ²	d ²	e ²	Authors	Estimation Technique	Year	Population
Party Id (Con vs Labor) Equality	0.12 0.27	0.70 —	— 0.28	0.19 0.45	Hatemi et al (2007) Olson et al. (2001)	Corr. ML	1990 2001	Australia 1990 Canada
RWA Racial Prejudice (Females)	.50 — .64 0.49	.00 — .16 0.14	— —	.20 — .50 0.37	Bouchard (2004) Truett et al (1992)	Varies ML	Varies 1980	Varies Australia
Racial Prejudice (Males)	0.11	0.40	—	0.49	Truett et al (1992)	ML	1980	Australia
Out-Group Attitudes (Females)	0.31	0.24	—	0.45	Verhulst et al (2010)	ML	1990	Australia
Out-Group Attitudes (Males)	0.27	0.34	—	0.40	Verhulst et al (2010)	ML	1990	Australia
Military Attitudes (Females)	0.05	0.15	0.14	0.63	Eaves et al (1999)	ML – Assort.	1988	Virginia and National AARP
Military Attitudes (Males)	0.13	0.08	0.20	0.54	Eaves et al (1999)	ML – Assort.	1988	Virginia and National AARP
Punishment Attitudes (Females)	0.49	0.09	—	0.42	Verhulst et al (2010)	ML	1990	Australia
Punishment Attitudes (Males)	0.29	0.28	—	0.44	Verhulst et al (2010)	ML	1990	Australia

Treatment of Criminals	0.00	0.51	—	0.49	Olson et al. (2001)	ML	2001	Canada
Sex Attitudes (Females)	0.47	0.13	0.00	0.40	Eaves et al (1999)	ML – Assort.	1988	Virginia and National AARP
Sex Attitudes (Males)	0.36	0.07	0.11	0.45	Eaves et al (1999)	ML – Assort.	1988	Virginia and National AARP
Sexual Conservatism (Females)	0.33	0.27	—	0.41	Truett et al (1992)	ML	1980	Australia
Sexual Conservatism (Males)	0.33	0.27	—	0.41	Truett et al (1992)	ML	1980	Australia
Turnout	0.72	0.20	—	0.09	Fowler et al (2008)	Bayesian	2005	Add Health
Political Participation	0.60	0.18	—	0.23	Fowler et al (2008)	Bayesian	2005	Add Health

Note: a^2 denotes the standardized additive genetic variance component, c^2 denotes the standardized shared environmental variance component, d^2 denotes the standardized non-additive genetic (dominance) variance component, e^2 denotes the standardized unique environmental variance component. In samples restricted to twins, either c^2 or d^2 may be estimated. When parents and other biological relatives are included, both c^2 and d^2 may be estimated and an assortative mating parameter that is combined into the additive genetic estimate in this table. A cell with an em dash indicates that that variance component was not estimated. Attitude items with sex in parenthesis indicate that the analysis was restricted to that gender. As behavioral genetic methods have advanced considerably over the past 30 years, several different analyses have been used. ML denotes Maximum Likelihood based analysis, Assort denotes that the estimates are corrected for assortative mating, corr denotes Falconer calculations based on the differences between the Mz and Dz correlations, and Bayesian denotes a Bayesian analysis technique.