

Genetic and Environmental Approaches to Political Science

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Abstract

Over the past decade, a growing interest in the possibility that biological factors, including genes, might contribute to individual differences in political and social behaviors has emerged. Behavioral genetic techniques have provided a variety of approaches to quantify the effects of genetic and nongenetic inheritance. However, until quite recently, these methods were largely unknown to political scientists. In this essay, we review the general approaches to modeling genetic and social influences on differences in complex human social traits. In so doing, we focus on the “genetics of politics,” including attitudes, ideologies, voting, and partisanship. The emergence of this research reflects a paradigm shift in the study of social traits necessitating the inclusion of biological influences, and recognizing the interdependence of genetic, social, and environmental factors in the development of political behaviors over the life course.

INTRODUCTION

Interest in identifying genetic influences on political traits began in the 1970s by psychologists and geneticists (Eaves & Eysenck, 1974; Martin *et al.*, 1986) and has remained a topic of interest since (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990; Eaves & Hatemi, 2008; Hatemi, Medland, Morley, Heath, & Martin, 2007). Yet, the last decade witnessed major developments in terms of integrating behavior genetic (BG) approaches into explicating political traits in the social sciences (for reviews see Hatemi, Dawes, Frost-Keller, Settle, & Verhulst, 2011; Hatemi & McDermott, 2012a). A multitude of special issues focused on behavioral genetic approaches have appeared in *The Annals of the American Academy of Political and Social Science* (Hibbing & Smith, 2007), *Political Research Quarterly* (McDermott, 2009), *Social Sciences Quarterly* (Lineberry, 2011), *Journal of Theoretical Politics* (Hatemi, Byrne, & McDermott, 2012), *Political Psychology* (Hatemi & McDermott, 2012c), and *Twin Research and Human Genetics* (Hatemi, 2012).

As Hatemi, Byrne, *et al.* (2012) advocate, in order for BG approaches to be integrated into political science, it is necessary for scholars to begin from the same set of starting assumptions regarding the nature and meaning of genetic influences. Thus, we offer a brief description of some of these approaches, results from recent studies, and their implications for understanding political behaviors.

FOUNDATIONAL RESEARCH

WHAT IS A GENE?

Genes regulate the cellular environment and create proteins, the main functional tools in the cell, which in turn instigate or restrict hormonal and other biological pathways in both state and trait circumstances. Thousands of genes interact with countless environmental conditions, both inside and outside the body to produce the chain of mechanisms that lead to a given trait, which may radically differ across the life span. Thus, whenever genetic influence is found for a given trait, whether by twin studies that rely on a latent measure of genetic influence, or molecular studies that rely on specific markers and their expression, it is implied that the genetic influences are not fixed, but conditional upon and interacting with environmental conditions, developmental processes, and other biological mechanisms (Hatemi, Byrne, *et al.*, 2012).

Influences between genes and behavior are mutual and bidirectional. It is believed that DNA has some role in indirectly guiding people into certain environments, and gene expression is affected by and based on exposure to those environments and one's own behavior. In this view displayed in Figure 1, the incorporation of genetic influences on political or social traits are set in a framework of constant interaction between biological and environmental forces that differ at various stages of one's lifetime. In addition, as Hatemi and McDermott (2012a, p. 4) state, "whatever genetic influences exist probably operate through those emotional, cognitive, or rational processes that are instigated when individuals are asked particular questions about their attitudes." Given the complexity of these processes, it is most likely impossible for any single gene to account for any substantial amount of variance for any complex social or political trait. Rather, it is the totality of one's genetic make up, in combination with social and environmental stimuli, which account for different exposure to and selection into experiences, emotive and cognitive states, perceptions, and preferences.

Considering the dynamic nature of genetic mechanisms, how can one accurately identify genetic influence? As Box and Draper (1987, p. 74) eloquently state: "Remember that all models are wrong; the practical question is how wrong do they have to be to not be useful." A model that perfectly captures

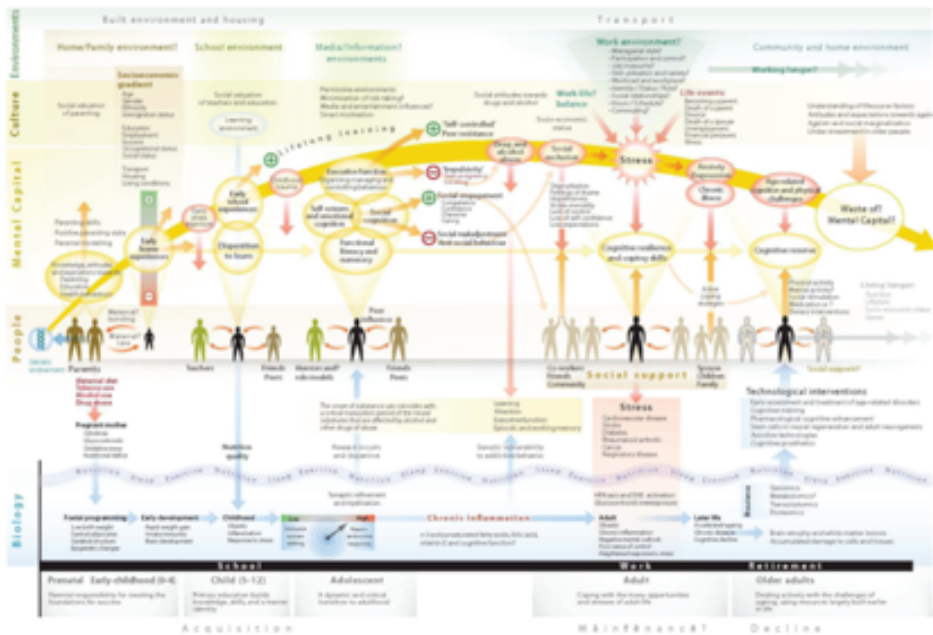


Figure 1 The Interaction of Biology and Environment Over the Life Course. Notes: Figure taken from Hatemi, Byrne, *et al.*, 2012 and originally published by Project Foresight (2008) Mental Capital and Wellbeing Project. London: The Government Office for Science. Available at www.bis.gov.uk/foresight.

one’s genetic, social, environmental, psychological, and physical factors over the life course does not exist. To conduct empirical research, whether using social, environmental, developmental, or biological approaches, or the combination as we advocate, scholars must rely on reductionist models that make assumptions about the world, and such assumptions shape and limit the interpretation of results. All statistical models attempt to simplify the vast complexity of real life in order to allow researchers to test specific hypotheses, and genetic models remain equally informative and fallible as any social science approach (for a detailed discussion, see Verhulst & Hatemi, 2013). Given these limitations, we discuss the two most common approaches to explore genetic influences and how they have been applied to political traits.

CUTTING-EDGE RESEARCH

MODELS OF HERITABILITY

BG analyses guided by biometric theory assume that the variation of a phenotype or trait (P) can be thought of as a consequence of latent genetic (G) and environmental factors (E). Twin and kinship models are among the

most popular approaches to identify the sources of variance on a trait or the sources of covariation between traits. The conventional twin model notation is that the total variance of a trait can be decomposed into additive genetic effects (A) or the sum of the effects of all the individual genetic markers that influence the trait; shared or common environmental influence (C), which captures the factors that are perfectly shared between twins and family members, such as the effects of neighborhood; and unique environmental influence (E), which captures all environmental stimuli not shared between twins, including error ($P = A + C + E$) (Medland & Hatemi, 2009).

This model differs from traditional social science models (SSMs), which seek to identify systematic relationships between two or more characteristics, and predict different outcome levels depending on the variables placement in the regression equation. SSM models assume that all traits, regardless of position, are environmentally determined; the exclusion of biological factors is not a decision based on model fitting, but an *a priori* paradigmatic decision (Smith & Hatemi, 2013).

Extant literature suggests that the assumption of purely environmental determinants of political traits is not warranted and genetics plays an important role in how and why people differ (Hatemi & McDermott, 2012a). For example, numerous studies conducted across decades and in several different countries find individual differences in ideology are genetically influenced (between 0.3 and 0.6 of the variance, see Hatemi *et al.*, 2014). These findings inform theoretical models involving ideology because they steer researchers to understand ideology as a psychological disposition that guides behavior and consequently employ it as a predictor, not an outcome. Contrary to the recently hypothesized notion that the heritability of ideology is simply channeled through personality (Mondak, Hibbing, Canache, Seligson, & Anderson, 2010), a recent stream of research (Verhulst, Eaves, & Hatemi, 2012; Verhulst, Hatemi, & Martin, 2010) shows that genetic influences on attitudes and ideologies are not subsumed by other covariates but specific to ideological differences. The challenge for political science theories becomes more poignant: it is not only that the assumption of no genetic influences for political traits is unwarranted but also these genetic effects are more than some spillover or confounded effects channeled through related traits. These findings brought about the imperative for an integrated theory of ideology, that embraces both genes and environment, yet remain embedded within a developmental framework, that includes parental investment, social groups, education, cognition, perception, aging, and all other critical environmental and neurobiological mechanisms (Eaves, Hatemi, Heath, & Martin, 2011; Fowler & Schreiber, 2008; Hatemi *et al.*, 2009).

In the majority of twin models, the focus is on monozygotic (MZ) and dizygotic (DZ) twin pairs reared together. MZ twins develop from a single

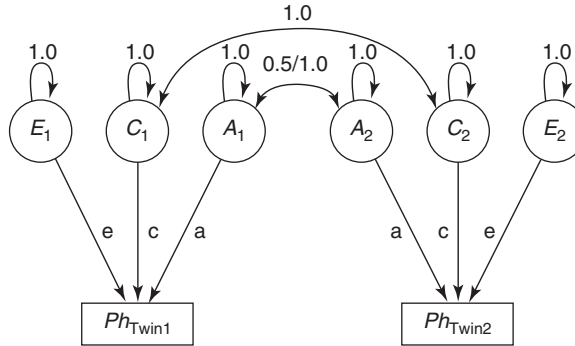


Figure 2 ACE model. Notes: Figure prepared by the authors.

fertilized egg and share 100% of their chromosomal sequence (i.e., “genetically identical”), whereas DZ twins develop from two different eggs fertilized by two different sperms and share, on average, 50% of their chromosomal sequence (Medland & Hatemi, 2009). The most valuable information stems from the covariance between twin pairs for each zygosity type. Different relationships of between twin-pair correlations (r) for MZs and DZs indicate what sort of transmission should we expect: if $r_{MZ} = r_{DZ}$, there is no genetic effect; if $r_{MZ} > r_{DZ}$ genetic and (shared and unique) environmental factors are present; if $r_{MZ} = 2 \times r_{DZ}$, genetic and unique environmental factors drive the variation in the phenotype (no shared environmental effects); and if $r_{MZ} > 2 \times r_{DZ}$, the variation in the phenotype is due to additive genetic effects, nonadditive genetic effects, and unique environmental factors.

Figure 2 shows the ACE variance decomposition and how this decomposition is informed by the properties of twin data. We have information about the phenotype for each twin from a pair, and these are marked in the rectangles. The model stipulates that $P = A + C + E$, where the three factors are unobserved and the relative proportions of variance can be decomposed into these three components that sum to 1. Working with twins reared together, we assume that the shared or common environment influences each twin in the same manner, and thus the C_1 and C_2 are correlated perfectly, $r = 1.0$, where subscripts indicate twin 1 and twin 2. This correlation is independent of zygosity (i.e., the equal environments assumption). Unique environmental factors are defined to capture why the twins are different, and hence they are not correlated. The correlation between the genetic factors (A_1 and A_2) reflects the amount of shared genetic material (MZ twins is set to 1.0. DZ is set to 0.5; for a full description, see Medland & Hatemi, 2009).

To illustrate this method consider, Klemmensen *et al.* (2012) study of political participation in Denmark and the United States. The MZ pair correlation is 0.51, whereas the DZ correlations is 0.32, indicating that

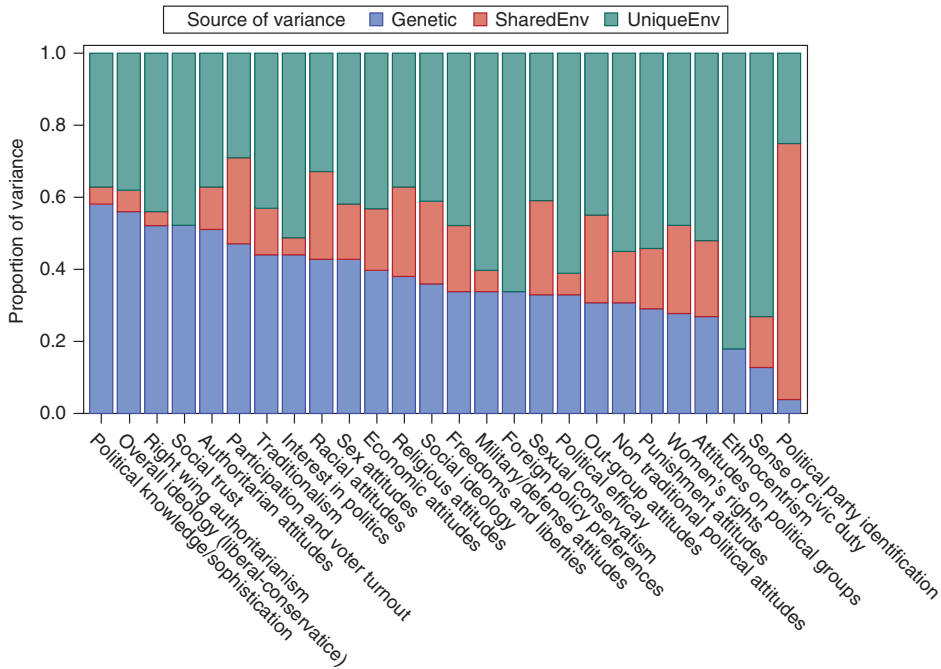


Figure 3 Summary of Relative Genetic and Environmental Influences on Political Traits. Notes: Figure taken from Hatemi and McDermott (2012c, 526).

genetic factors should play an important role in understanding variation in participatory behavior but shared environmental factors could also exert some influence. The univariate ACE model results of $A = 0.39$, $C = 0.12$, and $E = 0.49$ can be interpreted to mean that roughly 39% of the variation (why individuals differ) in participation is accounted for by genetic factors. The 95% confidence intervals for the shared environment (C) are 0.00 and 0.29, which means that the shared environmental effects cannot be distinguished from 0 at the traditional thresholds employed in quantitative analysis, while the bulk of the variation is accounted for by unique factors, and error.

Scores of political traits have been explored relying on twin studies and other models of heritability. Hatemi and McDermott (2012a) combined the findings of all reported twin and kinship studies that estimated genetic and environmental influences on political traits from 1974 to 2012 and aggregated them into 26 domains. Figure 3 displays the relative proportion of variance on each trait explained by additive genetic factors, common environmental influences, and unique environmental influences.

As displayed in Figure 3, individual differences in ideology, political knowledge, trust, authoritarianism, and participation and most political attitudes are accounted for largely by genetic and environmental factors.

However, differences in one's party identification, sense of duty, and ethnocentrism are hardly, if at all, influenced by genetic factors.

ACE models can be extended in numerous ways, including research questions involving the genetic and environmental covariance between two or more variables (Hatemi, McDermott, Eaves, Kendler & Neale, 2013; Neale & Cardon, 1992). Continuing with the Klemmensen *et al.* (2012) example, this approach is suitable for answering whether the genetic influence on political efficacy and political participation is partially shared; or if same latent genetic or environmental factors account for the covariance between these traits. That is, rather than focusing on prediction, or the size of the correlation between traits, multivariate models offer information what is driving the correlation. Is the trait of interest related due to a common genetic factor or due to similar experience or familial environment? Building on the between-trait correlations, in this Klemmensen *et al.* study, those who are politically more efficacious also participate more in politics, however roughly 80–90% of the covariation between efficacy and political participation is driven by a common latent genetic factor and not by environmental similarity, a finding that requires serious rethinking of current theories on how efficacy influences political behaviors.

Further extensions such as direction of causation models are able to test directional hypotheses. Verhulst and colleagues (Verhulst & Estabrook, 2012; Verhulst *et al.*, 2010; Verhulst *et al.*, 2012) employed such a model to explore four causal scenarios: (i) a unidirectional causal model where the variation in personality traits drives the variation in political attitudes; (ii) a unidirectional causal model where the set of genes that influence variation in political attitudes in turn leads to variation in personality traits; (iii) reciprocal causation, where personality traits and political attitudes have a nonrecursive causal structure; and (iv) pleiotropy or a common set of genes that mutually influences both personality traits and political attitudes. Verhulst *et al.* pointed toward the fourth scenario or possibly even the second, finding no causal relationship from personality to political attitudes (also see Dawes *et al.*, 2014; Hatemi & Verhulst 2015), in direct contrast with political science theories (Mondak *et al.*, 2010). DoC models can be extremely powerful tools for testing theories and disentangling causal relationships that are only asserted but remain empirically untested.

UNDERSTANDING HERITABILITY

Notwithstanding the increasing exposure of political science to these methods through detailed methodological papers (Boardman, 2011; Eaves *et al.*, 2011; Hatemi, 2013; Medland & Hatemi, 2009; Verhulst & Hatemi, 2013), there remains an erroneous understanding of how genes operate, what

heritability means and the statistical and theoretical assumptions required to conduct empirical research.

First, an accurate interpretation of genetic influence does not include the word “determined.” With rare exception, genetic influences are not fixed or unmalleable. They are mediated and moderated by environmental conditions and change greatly throughout the life cycle. Heritability estimates account for variance within a population at a given time and are population specific. Instead of explaining the value of trait, they focus on the difference of values on a trait within a population. In the case of the above-mentioned Klemmensen example, it is not that genes explain 39% of political participation; rather, it is that 39% of the variance, or individual differences in political participation within the population, are accounted for by the aggregate of genetic influences. Twin or “ACE” models explain how people differ. They are not to be interpreted to mean that for every person in the population 0.39 of their political participation is due to genes (for more detail, see Hatemi, Byrne, *et al.*, 2012).

ASSUMPTIONS, LIMITATIONS, AND EXTENSIONS

Akin to any statistical model, univariate classical twin models (CTDs) rely on several assumptions (for more detail, see Hatemi *et al.*, 2012a; Medland & Hatemi, 2009); three of them have received the most attention both from critics and advocates of the CTD: equal environment assumption (EEA), no assortative mating, and no correlation or interaction between genetic and environmental influences ($r_{GE}/G \times E$). The EEA stipulates that, on average, MZ cotwins share equally similar environments as DZ cotwins, or that if any differences do exist they have no effect on the traits of interest. This assumption enables researchers to equate the cotwin correlations for the latent common environmental factor (Figure 2). If this assumption does not hold (EEA violations), the CTD would bias the genetic effects upward and the shared environmental influences downward. Many studies have addressed the EEA issues for political traits (Hatemi *et al.*, 2009; Littvay, 2012; Smith *et al.*, 2012) and the results consistently suggest that wherever the similarity in MZ cotwins family environments differ from DZ cotwin pairs, such differences have no effect on the heritability estimates of political traits.

The basic twin model also assumes that no assortative mating for the trait exists; that is, parents (spouses) choose each other randomly and not based on similarities related to the traits under investigation. This assumption however is often not true for social traits. Indeed, overwhelming evidence indicates that this assumption does not hold for political traits as well; spousal correlations for ideology range from 0.62 to 0.68 (Alford, Hatemi, Hibbing, Martin, & Eaves, 2011; Eaves & Hatemi, 2008, 2011; Eaves *et al.*,

1999; Eaves *et al.*, 2011; Hatemi *et al.*, 2010). This is important because in the simplest scenario, if there is assortative mating on the trait of interest and this trait is heritable, genetic influences will be *underestimated* in a classical twin model, and the shared environmental effects will be overestimated (Eaves & Hatemi, 2008).

This lacuna is easily remedied by simple extensions to the CTD. Indeed, the effects of assortative mating were estimated in extended kinship models using information on the twins' parents and twins' spouses, resulting in genetic influences accounting for an even greater portion of individual differences on political attitudes and ideology than reported by the CTD (Eaves & Hatemi, 2008; Hatemi *et al.*, 2010). Extended kinship models offer the possibility to estimate numerous other types of intergenerational transmission including gene–environment covariance and sibling-specific environmental influences. For important political traits such as ideology, analyses carried out on extended family data reinforced the results presented by twin studies, further emphasizing that a serious theoretical discussion of genetic effects on political traits is inevitable if we want to advance our understanding of politics and political preference formation (Hatemi *et al.*, 2010).

Univariate twin models provide only the simplest representation of a highly complex gene to behavior process. That is, similar to simple regression models, they provide a baseline that allows for the exploration of increasingly complex modes of phenotypic transmission (for a description of this progression, see Hatemi *et al.*, 2009). However, numerous extensions to the CTD exist allowing for more complex analyses and the integration with traditional political science theories. Indeed, an emerging stream of research has begun to merge BG theories with psychological and political science theories (Fowler, Baker and Dawes, 2008; Hatemi, Eaves, & McDermott, 2012; Hatemi & McDermott, 2012b; Hatemi *et al.*, 2007; Hatemi *et al.*, 2009; Hatemi *et al.*, 2013; Loewen & Dawes, 2012). In one such example, Fazekas and Littvay (2012) investigate proximity and directional voting principles. Building on rational choice models and employing an operationalization rooted in social psychology, they analyze the heritability of the adoption of these specific voting theories. They use a bivariate twin model to show that there are shared underlying genetic factors influencing both the strength of partisanship and which spatial considerations guide individual vote choice.

Another simple extension to the CTD allows for inclusion of specific environmental measures, thus making it possible to estimate gene–environment interplay. Statistically speaking, when an individual differentially responds to or selects into an environment (actively or passively) as a function of their genotype, they induce an interaction or correlation between their genotype and the environment. Several works have recently provided methodological and theoretical primers on gene–environment interplay specifically tailored

for political scientists (Boardman, 2011; Hatemi, 2013; Verhulst & Hatemi, 2013), while others have provided empirical examples, ranging from the influence of childhood environments on genetic influences of ideology (Smith *et al.*, 2012) to the import of school nutrition programs on the genetic influences on childhood obesity and its impact on public policy (Boardman *et al.*, 2012). Research thus far has demonstrated that for the majority of political traits, biases from passive gene–environment covariance are not significant or substantial (Eaves & Hatemi, 2008; Hatemi *et al.*, 2010). However, this was not the case for gene–environment interaction. Hatemi (2013) found that twins responded to life events differentially based on genetic similarity. In most cases, he found that for the population of individuals who experienced financial problems were laid off or fired or were divorced in the last year, genetic influences on Capitalism and Socialism all but dissipated (Figure 4). However, for the population of individuals who were laid off or fired, genetic influences on Property Tax increased (Figure 5).

These studies are only a small representation of an integrative research framework that has emerged in within political science with has combined latent models of transmission with the foundation works of our field, extending both the theoretical models and empirical toolkit of the discipline.

GENE MAPPING

Gene mapping methods identify associations between-trait levels and specific genetic polymorphisms, by analyzing *a priori* selected genes or by scanning the entire genome for a genetic marker or chromosomal region that covaries with the trait of interest or by measuring the expression of specific genetic marker under certain environmental conditions (e.g., epigenetic). These approaches identify specific biological mechanisms responsible for some portion of the variation in behavior.

Candidate gene studies preselect genes that are believed to be susceptible to be associated with the trait under consideration. Some genes come in alternate forms at a given chromosomal position—labeled as alleles—that reflect one (or more) single-nucleotide polymorphisms (SNPs) or are due to differences in length of specific DNA section (Hatemi, Gillespie, *et al.*, 2011). “Gene association studies test whether an allele or genotype occurs more frequently within a group exhibiting a particular trait than those without the trait” (Fowler & Dawes, 2008, p. 584), this being the case-control design. Alternatively, family designs can be employed that “compare whether offspring exhibiting the trait receive a specific allele from their parents more often than would be expected by chance.” The statistical methods are those commonly found in most political science research, analysis of variance (ANOVA), and some form of regression.

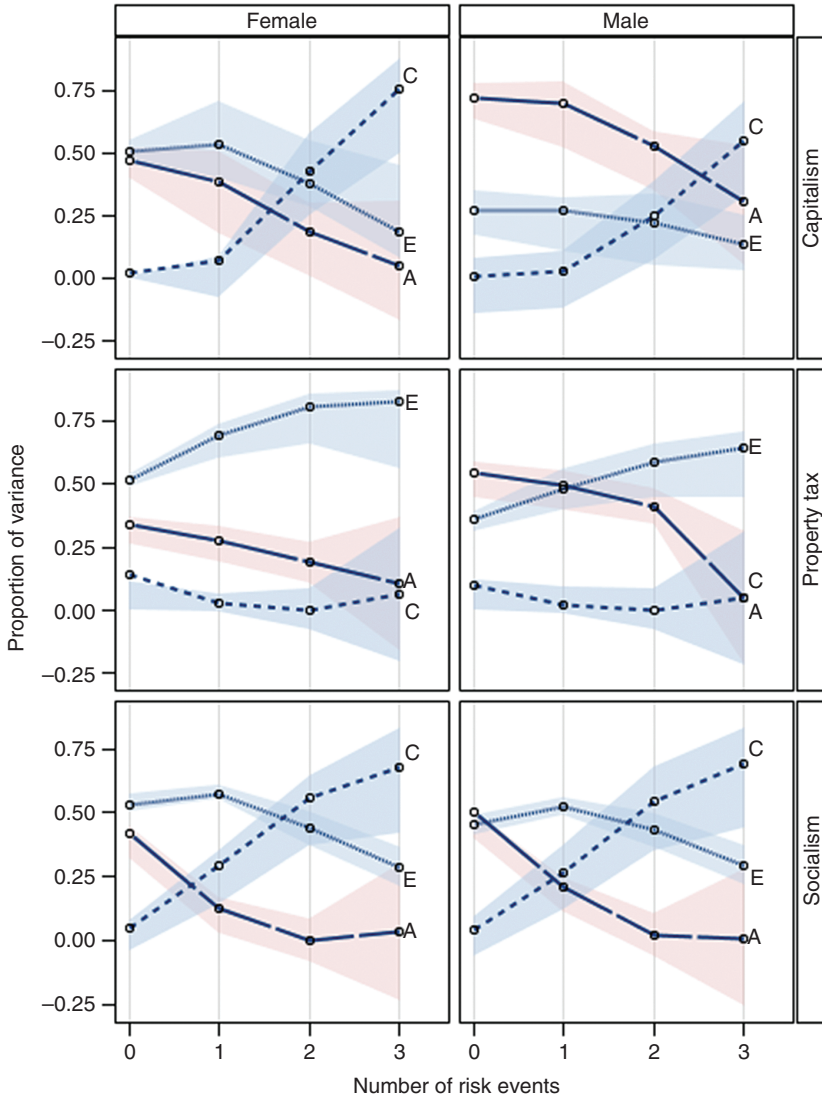


Figure 4 Change in Source of Variance on Capitalism, Property Tax, and Socialism When Exposed to One or More Financial Risk Events. Notes: Figure taken from Hatemi (2013).

This area of research is developing and numerous publications now exist which explore the relationship between genetic markers and political traits, including interactions with environmental conditions (Benjamin *et al.*, 2012; Dawes & Fowler, 2009; Fowler & Dawes, 2008; Fowler, Dawes, & Christakis, 2009; Fowler, Settle, & Christakis, 2011; Hatemi, Gillespie, *et al.*, 2011; Hatemi *et al.*, 2014; McDermott, Dawes, Prom-Wormley, Eaves, & Hatemi, 2013; McDermott, Tingley, Cowden, Frazzetto, & Johnson, 2009).

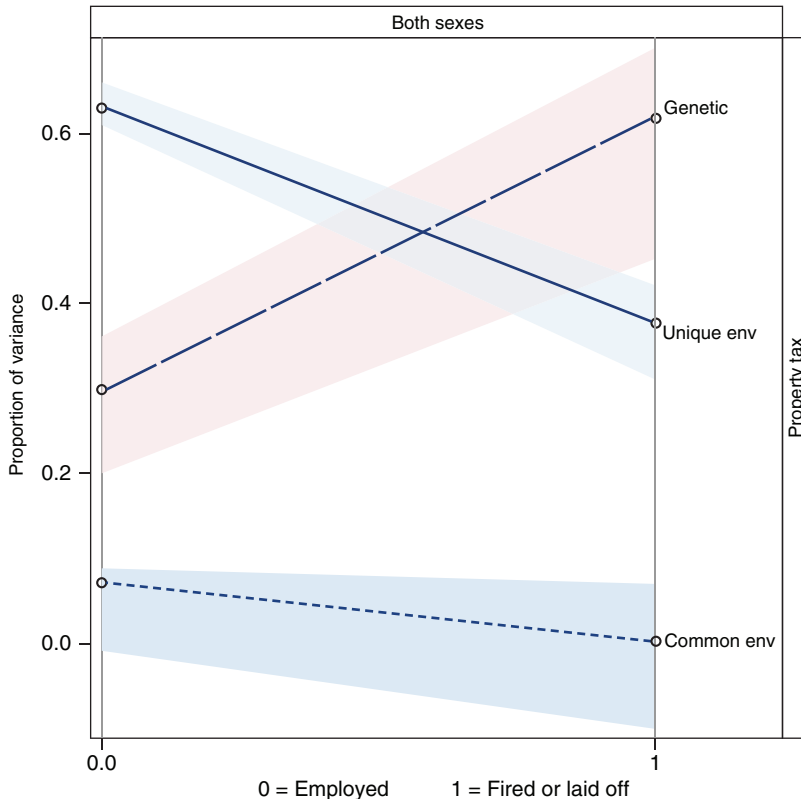


Figure 5 Changes in Source of Variance on Property Tax When Fired or Laid Off. Notes: Figure taken from Hatemi (2013).

For example, Fowler and Dawes (2008) build their selection of the MAOA and 5HTT (serotonin) as candidate genes associated with voter turnout because these two genes were previously linked to antisocial behavior. They find that the “high” allele of MAOA and the “long” allele of 5HTT are significantly associated with higher voter turnout, but only for those who frequently attend religious services. Similarly, focusing on the role of the dopamine D2 receptor in forming social attachments, Dawes and Fowler (2009) report that having an A2 allele in the DRD2 gene is significantly associated with higher probability of partisan attachment. However, yet again, the authors and Hatemi, Byrne, *et al.* (2012, p. 319) point out that “the proposed pathway suggested to influence voting behavior noted above, is certainly a function of the genotype; that is, certain genotypes appear to have a role in the greater or lesser release or uptake of hormones, but the regulation of these hormones are a function of gene expression. [...] However, it is critical to note that most candidate gene studies account for a very small amount of the variance, and most results fail to withstand efforts at replication.”

Avoiding the bias of preselecting particular genes, genome wide approaches (GWAs) present themselves as “more empirically rigorous method [that] scans the entire genome for a genetic marker or chromosomal region that is significantly related to the trait of interest” (Hatemi, Byrne, *et al.*, 2012, p. 317). These analyses can “implicate genes that we *did not* suspect were influencing a trait of interest and thus reveal novel pathways to the formation of political orientations” (Hatemi, Gillespie, *et al.*, 2011, p. 2). Hence, it is an exploratory and data-driven approach.

So far, GWAs have not identified a specific genetic marker related to political traits. Hatemi, Gillespie, *et al.*, 2011 conducted a genome wide linkage study and found three regions that significantly covary with and account for up to 13% of ideology. Many genes reside within the 90% or 95% confidence intervals of the identified peaks that were related to similar social traits. However, in two follow-up studies that employed a more rigorous genome wide association approach that focuses on specific variants, no specific markers were found to be associated with political ideology (Benjamin *et al.*, 2012; Hatemi *et al.*, 2014). This, however, was the expected conclusion because the effects of individual markers on such a complex traits are going to be extremely small, too small to identify because extremely large samples, possibly in the hundreds of thousands, are needed (Hatemi *et al.*, 2014, p. 22). Unfortunately, the largest sample available for GWA analyses on political traits numbers less than 14,000. Thus, for political traits, genome wide association studies are in a far less developed stage compared to models of latent influence, but technological and methodological expansion is certain to lead to further advancement.

KEY ISSUES FOR FUTURE RESEARCH

In this extremely abbreviated contribution, we have provided a brief review of genetic methodologies applied to political traits, using findings from the recent literature. We view the introduction of these BG approaches as a start, not a conclusion. Recent findings introduce new questions and continue BG methodological developments and availability of data accommodate the possibility of answering these questions. Research began with relatively simple models of heritability that raised both ontological and theoretical questions. Extended kinship models reinforced those findings and further detailed the underlying mechanisms behind the transmission of political attitudes. Subsequently, the interplay between physiological and environmental forces was incorporated into the scientific inquiry of political traits, and lately specific genetic markers, and the interaction between genes, and genes and environments are emerging. All these were necessary because prior findings posed new challenges and a more complicated picture of

political behavior emerged. We expect to see models that focus on gene expression and developmental and longitudinal designs in the near future (e.g., Hatemi *et al.*, 2009). Given that exclusively environmental theories of political behavior offered only partial answers, this more inclusive description of political behavior appears to resemble more closely the reality of contemporary politics.

Integrating models of genes and environments contributes to our broader conception of how individuals develop, select into and react to specific environments, and ultimately decide on crucial matters related to power sharing, social construction, and societal interactions. If the goal of research is to increase knowledge, the inclusion of BG approaches is indeed a substantive one. This integration, however, means that there is a lot of catching up to do. Some methodological concerns have been already addressed decades ago in other disciplines and accepted limitations have been treated as such. With the integration of research traditions, a detailed understanding of the underlying principles and limitations are necessary. One such example is the focus of models of heritability on within-population variance (i.e., individual differences) in contrast with the interest in population mean prediction in most SSMs. Providing an estimate of why people differ in a population is not equal to, and should not be equated with, why someone is a liberal or a conservative. When research is misunderstood, as is often the case with new material, interpretation of findings become erroneous, which inevitably has a spillover effect on the integration of the substantive implications and research approach.

Inclusion of a BG approach shifts research questions toward understanding mechanisms not simply manifestations. With increased attention, technology, data, and better measurement of traits, we can test our theories in a more rigorous manner. Forthcoming panel studies and experiments on genetically informative samples allow researchers to address issues of causality and to recover changes (or stability) in political traits in novel ways, including the driving forces behind change. Such models also allow for researchers to better focus on environmental stimuli, by controlling for genetic disposition. In the quest for understanding the complicated nature of individual political behavior, theoretical and methodological expertise from political science, genetics, psychology, sociology, and many other fields contribute to asking better questions and getting more accurate answers and ultimately increase our knowledge base in a rigorous scientific manner.

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