# 6. Genes and politics

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## INTRODUCTION

The role of genes in political attitudes and behaviors has become one of the liveliest and most heated debates in political science. Though our colleagues in psychology and other disciplines have long recognized that political orientations are influenced by genetics, just like other complex social behaviors, the exploration and theoretical development of the role of genes within our discipline is a fairly recent phenomenon. For many, the political world is a rational place where the marketplace of ideas leads individuals to deliberately select items from the menu of options as their issue attitudes, their ideology, and their partisanship. The menu and an individual's interpretation of their options result from cultural context, demographic positioning, life experiences, and purposive socialization. The idea that we may be genetically predisposed to prefer a certain set of policies or to process our environments in particular ways appears counter to decades of political science scholarship and centuries of political theory.

While some political scientists remain skeptical that genes have a meaningful role to play in understanding politics (for example, Bartels, 2013; Charney, 2008a, 2008b; Beckwith and Morris, 2008), there have been significant advances in the decade since Alford et al.'s (2005) seminal piece about genetic effects on political attitudes.[1] The ensuing research, which has relied primarily, but not exclusively, on studying twins raised together, has consistently shown that both genetic *and* environmental factors have a role to play in politics, both in the United States (for example, Funk et al., 2013) and abroad (for example, Bell et al., 2009; Hatemi et al., 2007). These results are corroborated by research using a variety of behavioral genetic methods – which rely on the known relatedness of individuals to infer the effect of genes on some trait – such as studies of twins raised together, twins raised apart (for example, Bouchard et al., 2003), and extended family designs that do not rely on twins (for example, Hatemi et al., 2010). The role of genes in politics has also been confirmed using molecular genetic methods, which rely on samples of actual genetic material to demonstrate the association of genes and some trait – such as candidate gene studies and genome-wide association studies (GWAS; for example, Benjamin et al., 2012b; Hatemi et al., 2011). In sum, research using a variety of genetic methods leaves little doubt that genetic factors play important roles in political attitudes and behaviors in interaction with environmental factors.

As genetic approaches have become more common in political science, a debate has arisen as to whether these approaches should constitute a new subfield – sometimes called "genopolitics" or "biopolitics" – or whether behavioral and molecular genetics are simply new tools in our methodological kit to explore political questions within existing subfields of political science. Because of the costly up-front investment in learning genetic theory and techniques, it may seem justified to treat genopolitics as a subfield. Other scholars suggest that behavioral and molecular genetics are just one more way to try to understand questions that have driven our field for centuries. If we segregate ourselves into a subfield,

then we will not engage the rest of the discipline, and our findings will have a much more limited impact on the key debates within political science.

Regardless of one's perception of where this area of study "fits" into political science, the examination of the role of genes in politics over the past decade has led to significant advancements in the study of political ideology and party identification, political behaviors, and other traits that are relevant to various aspects of political life. In this chapter, we will outline the major findings while explaining the pathways and theoretical links that scholars have posited between genes and politics. But first, we begin with an exposition of the twin method – the most common and traditional method of examining genetic and environmental influences on traits – to better guide the reader in understanding the assumptions and parameters underlying much of this area of research.

## THE TWIN METHOD

The twin study method has a long pedigree, stretching back at least to the mid-1920s and perhaps as far back as the work of Francis Galton in 1875 (Rende et al., 1990). Twin studies are used to determine the extent to which genes and upbringing influence a particular phenotype (that is, observed trait, attitude, or behavior) and leverage the differences in zygosity – or whether the twins were born from two separately fertilized eggs or one egg that split. The basic intuition of the design is that if a trait shows greater similarity among monozygotic (identical) twins (who share all of their genetic code) as compared to dizygotic (non-identical/fraternal) twins (who share, on average, half of the genetic code that varies among humans,[2] the same amount as typical siblings), this suggests that the trait has a heritable component. By contrast, if a trait is purely environmental, twins should be equally similar on average regardless of zygosity (that is, whether they are identical or fraternal twins), since all twins reared together share a common family environment with their co-twin (for an alternative review of twin methods see Medland and Hatemi, 2009; see Smith et al., 2012 for a detailed discussion of the assumptions underlying twin methodology).

However, while twin designs are a staple in behavioral genetics, this methodology is relatively new in political science. Some political scientists have been particularly vocal in challenging the validity of the classical twin design by questioning its underlying assumptions (Beckwith and Morris, 2008; Charney, 2008a, 2008b; Shultziner, 2013a, 2013b)[3], and similar critiques have also been raised in other disciplines (for example, Burt and Simons, 2015; Joseph, 2015). These critiques have been thoroughly addressed by others in the literature (for example, Alford et al., 2008a, 2008b; Barnes and Boutwell, 2015; Hannagan and Hatemi, 2008; Smith et al., 2012; Verhulst and Hatemi, 2013; Wright et al., 2015), but some of the more pertinent points are discussed below.

The classical twin design is often called an ACE model because it decomposes or divides the variance of a trait into three components: *A* for the additive genetic component of the variance, *C* for the common environment component (that is, family upbringing and other experiences shared by both twins), and *E* for the unique environment component (that is, experiences unique to each twin; this component also contains measurement error).[4] These components cannot be observed directly, but their relative size is inferred statistically based on the size of the correlations among identical twins and fraternal twins. Figure 6.1 portrays several typical patterns of twin correlations that may be observed in

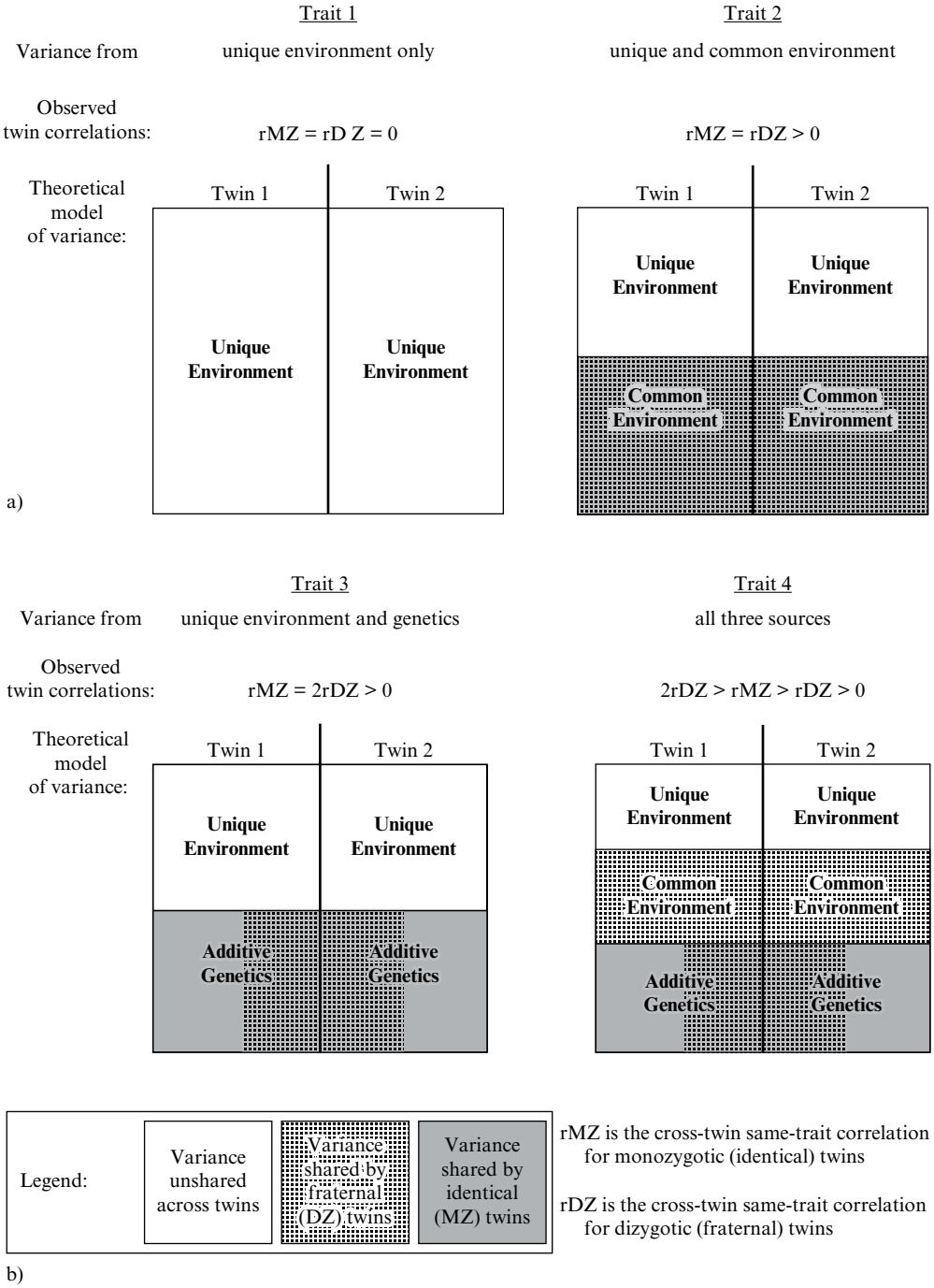


*Figure 6.1 Observed correlations and models of variance in the classical twin design*

real-world data and the inferences that can be drawn from each pattern of correlations. The boxes represent the theoretical sources or components of variance for each trait for a population of twins. In the simplest case, if identical and fraternal twins are uncorrelated with each other (as in Trait 1), then all of the variance in the trait is believed to be the result of unique environmental influences and measurement error (recall that *E* captures both of these terms; for brevity, we exclude measurement error from our discussion below but it is always a part of the unique environmental component). These unshared influences are represented in the figure as white space for each twin; these influences shape Trait 1 for each twin but are unshared across twins. If identical and fraternal twins are correlated with each other to the same degree (as in Trait 2), then the variance in the trait is believed to be the result of a combination of common environmental and unique environmental factors. The unshared, unique environmental influences are portrayed as white space as before, but now there is a region of overlapping variation, as well. This region is both shaded gray (indicating that it is shared among identical twins) and filled with dots (indicating that it is shared among fraternal twins) because common environmental factors are assumed to influence both identical and fraternal twins in the same way and, therefore, lead to the same amount of shared variance within twin pairs, on average, regardless of twin type. If identical twins have a correlation that is about double that over fraternal twins (as in Trait 3), then variance in the trait is believed to be the result of a combination of additive genetic and unique environmental factors. Note that the additive genetic region is shaded fully in gray (indicating that it is fully shared among identical twins, who have the same genetic code), but that it is only half filled with dots (indicating that the additive genetic variance is only half shared, on average, among fraternal twins, who share, on average, half of their segregating DNA). Finally, in the most complex case, if the identical twins' correlation is greater than the fraternal twins' correlation but less than double the fraternal twins' correlation, then this suggests that additive genetic, common environmental, and unique environmental factors all play a role in the trait. Note that in this case, the region of shared variance for identical twins encompasses the entirety of the common environmental and additive genetic regions, whereas for fraternal twins, the region of shared variance encompasses all of the common environmental region but only half of the additive genetic region.

It is important to recognize that when researchers use twin correlations or other genetic techniques to estimate the contributions of genetic and environmental factors to the variance for any trait, these estimates are population-specific. In a different sample, at a different time, in a different place, the proportions of variance that can be attributed to any one element may vary (for a brief and informative discussion on this point in the context of epidemiology, see Freese, 2006). This may be particularly true of political phenomena, which are constrained and defined by social contexts and institutions.

**Assumptions of Twin Studies**

Determining the components of variance of a trait is typically accomplished through a structural equation model (Neale and Cardon, 1992), though it is also possible to estimate sources of variation in a regression framework when considering only a single variable (DeFries and Fulker, 1985; Smith and Hatemi, 2013). Like all statistical models, the ACE model is based on various assumptions that, if violated, can result in biases in the

estimates of heritability and environmental influence that the model produces. For those skeptical that genes play a role in political attitudes and behaviors, violations that lead to overestimates of *A*, the genetic component in an ACE model, are particularly problematic. Recall that we cannot observe the size of the genetic component directly using behavioral genetic methods; it is inferred based on the relative correlation of identical, monozygotic (MZ) twins and fraternal, dizygotic (DZ) twins (see discussion of Figure 6.1 above). In general, an overestimate of the genetic component will occur when the trait correlation among MZ twins is inflated relative to the correlation among DZ twins for reasons other than the difference in genetic relatedness. Conversely, an underestimate of *A* will occur when the trait correlation among DZ twins is inflated relative to the correlation among MZ twins. There are three assumptions that, if violated, will lead to upwardly biased estimates of *A*, the heritability parameter.

The most commonly discussed violation pertains to the equal environments assumption (sometimes referred to in the literature as the EEA). In the ACE model, the common environment parameter, *C*, is assumed to have an equal influence on both identical (MZ) and fraternal (DZ) twins. This is because the common environment parameter is meant to capture the portion of the variance that is completely shared within twin pairs, regardless of their zygosity (that is, the shared environment of each twin pair). The challenge to this assumption states that MZ twins have a more similar environment than DZ twins, because, for example, parents treat identical twins more similarly than they treat fraternal twins. As a result, the MZ correlation on the trait of interest is inflated and some of the shared variance among MZ twins that is the result of the shared environment is misattributed to genetics. However, empirical work suggests that the EEA is not violated at least with regard to at least some political phenotypes (Smith et al., 2012). That is, parents socialize their children into their political beliefs equally, regardless of zygosity.

A second assumption whose violation can lead to overestimation of heritability is that there is no interaction between genetic variants at the same or different locations on the genome. Strictly speaking, the *A* component is an estimate of additive genetic effects (that is, thousands of small, independent effects resulting from variation in single nucleotides that comprise the genome), rather than non-additive effects. These non-additive effects can be either dominance effects, where alleles are interacting at a particular location on the genome (see note 4), or epistatic effects, which are the result of gene-by-gene interaction. When these effects are present, estimates of additive heritability in twin models (that is, *A*) are inflated (sometimes called "phantom heritability"; see Zuk et al., 2012 and 2014 for a detailed discussion of this issue and possible solutions). Although there is no way to overcome this limitation directly in a classical twin study, one albeit imperfect solution is to simply interpret the *A* estimate as an estimate of broad-sense heritability (that is, additive and non-additive genetic effects) rather than narrow-sense heritability (that is, only additive genetic effects). When the heritability estimate is significant, the proper interpretation in a classical twin design is that genes play a role in the trait being studied, but the estimate should be treated as one of broad-sense heritability since it may also be picking up non-additive effects that should be explored using other methods.[5] Thus, unlike the equal environments assumption, this second form of inflating *A*, when interpreted correctly, is less problematic for political scientists, as our traditional theories do not account for any genetic effects on political traits and behaviors, regardless of whether these genetic effects are additive, dominant, or epistatic.

A third complicating factor is the existence of gene–environment correlation. People with certain genotypes may select into environments that are conducive to their traits or that are more likely to generate certain traits. Consider the "IQ paradox," that IQ is highly heritable, but that environmental factors are known to have large influences on IQ from non-genetic research. Researchers have suggested that one solution is that people who are predisposed to have higher IQ may select into environments that foster higher IQ, thus creating a virtuous cycle (Dickens and Flynn, 2001a, 2001b). Analogously, individuals who are predisposed to be high in openness to new experiences may be exposed to a greater variety of life experiences, which may bolster their openness and also contribute to the development of liberal political attitudes (in accordance with a motivated social cognition view of ideology; Jost et al., 2003). In this sense, the genetic effects captured by heritability estimates may not be purely biological, but rather speak to a biological predisposition to select into particular types of environments. Violations of this assumption, then, are once again about the interpretation of *A*, which is inflated if we take a narrow view of it as additive genetic effects, but not if we treat it as an estimate of broad-sense heritability.

However, not all violations of ACE model assumptions lead to overestimates of even narrow-sense, additive-only heritability. There is an assumption of twin models which is routinely violated in studies of political phenotypes and can lead to an underestimation of *A* when violated: the assumption of no assortative mating. This assumption holds that people do not choose mates based on the trait or behavior in question (for example, a person is *not* more likely to mate with another person who shares his or her political attitudes). The effect of assortative mating is that DZ, fraternal, twins share more of their genes on average than they would if mating were completely random (MZ, identical, twins share 100 percent under any mating scenario by definition). The result of this increased relatedness among DZ twins is that, if the trait is affected by genetic factors, their trait-level correlation increases relative to MZ twins (again, because the genetic relatedness of fraternal twins for the genes that affect the trait is higher under assortative mating than it would be under random mating and the relatedness of MZ twins is unchanged). The result of an inflated fraternal-twin correlation is an overestimate of the common environment component (*C*) and an underestimate of the genetic component (*A*). Given that there is extremely strong assortative mating on political phenotypes (Alford et al., 2011; Klofstad et al., 2012, 2013; see also Hobbs et al., 2014), the result is that a classical twin model may underestimate the heritability of political phenotypes and overestimate the effect of common environmental factors, *C* (see Beauchamp 2011a on the effects of assortative mating in twin models).

Finally, it is also important to recognize that estimates of *A* are population-specific and may vary widely across populations as a function of differences in environmental factors. A classical twin design assumes that there is no gene-by-environment interaction. That is, genes may lead to the trait or behavior in question only under specific environmental parameters; this seems particularly likely for complex phenotypes like political attitudes and behaviors. For example, genetic effects on height are estimated to be much larger in nutritionally rich, affluent societies than in impoverished ones (for example, Silventoinen, 2003), with little variation in the size of the genetic component on body height among affluent countries even when mean levels differ significantly (Silventoinen et al., 2003). There is also greater variability in IQ in high socioeconomic status families and virtually

no effect of genetic factors in impoverished families (for example, Turkheimer et al., 2003). This is not to say that any of the estimates of heritability are wrong in one population or another. Rather, this assumption acknowledges the reality that the extent to which genetic factors can and do influence a trait is contingent on population-specific, environmental factors. It may truly be the case that when there is little variation in available nutrition in a population, genes account for a greater proportion of population variance, and that when there are vast differences in access to nutrition, genetic factors are overwhelmed by these environmental differences in accounting for variation in the population. The possibility of gene-by-environment interactions means that we must be cautious not to over-interpret the estimate of heritability and assume that just because a trait is heritable in one population (or subpopulation) it will be similarly heritable in all others. Rather, we should treat the classical twin design as what it is: a first cut at determining whether genes play a role in a trait or behavior that should be followed up with replications in different populations, theorizing about the types of environmental factors which might heighten or mute heritability, and the use of more sophisticated (and more costly) methods in future research, such as family studies or molecular genetic methods, which will be discussed in more detail below.

While violations of these five assumptions may push estimates in opposite directions (for example, a trait or behavior that is subject to epistatic effects and assortative mating will have its heritability estimate biased both up and down), there is no guarantee that these violations will balance out in any systematic way. However, since equal environments assumption violations do not appear to hold for political phenotypes, and since dominance effects, epistatic effects, gene-by-environment correlations, and gene-by-environment interactions are all at least partly genetic, one should simply interpret $A$ as an estimate of broad-sense heritability, or the sum of all genetic pathways in a particular environment. If the goal is simply to demonstrate that genes play a role in political traits and in linking traits together with each other and over time, subject to future study with extended family designs and genome-wide association designs, then the ACE model is sufficient for the task.

## OTHER METHODS FOR STUDYING GENES AND POLITICS

Although studies of twins raised together (that is, using the classical twin method) account for the bulk of the work that has considered the role of genes in politics, there are a number of other approaches that provide converging evidence for the importance of genetic factors in political life. Among these are other family designs, such as studies of twins raised apart (for example, Bouchard et al., 2003), of extended families which may include parents, siblings, or other relatives (for example, Hatemi et al., 2010), and of adoptees (for example, Cesarini et al., 2014). There are also various methods that examine unrelated individuals using molecular genetic techniques. Unlike behavioral genetic techniques, which infer that genes affect a trait based on the known relatedness of twins or other family members, molecular genetic techniques rely on collecting actual "wet" samples, measuring genetic information directly, and then using that genetic information to better understand the heritability (or lack of heritability) of a trait or behavior. Molecular genetic techniques include candidate gene studies that attempt to theoretically

identify genes which may influence a particular trait or behavior a priori and then test for that association (for example, Fowler and Dawes, 2008), genome-wide association studies that rely on analyses of large sections of the genome to identify particular polymorphisms associated with particular traits or behaviors without having to identify the polymorphisms of interest in advance (for example, Benjamin et al., 2012b), and as yet unpublished studies that estimate the total additive effect of genes using genome-wide complex trait analysis (see Yang et al., 2011 for an introduction to GCTA). While each of these family and molecular methods has its own assumptions, those assumptions vary widely across methods. Taken together, the results converge on a single conclusion: genes do play a role in political traits.

## GENES AND POLITICAL ATTITUDES

Behavioral geneticists have expressed sporadic interest in genetic effects on political and social attitudes for many years (for example, Eaves and Eysenck, 1974; Martin et al., 1986). But it is only in the past decade that political scientists themselves have begun to examine the role of genetic and biological processes in political phenomena. In that short time, numerous studies have shown that genes do play a role in both political attitudes (for example, Alford et al., 2005; Bell et al., 2009; Eaves and Hatemi, 2008; Hatemi et al., 2009a, 2009b; Hatemi et al., 2011a; Smith et al., 2012) and in political behaviors like voting (for example, Dawes and Fowler, 2009; Fowler et al., 2008; Fowler and Dawes, 2008; Hatemi et al., 2007). This literature has spawned extensive reviews in political science (Alford and Hibbing, 2008; Ebstein et al., 2010; Fowler and Schreiber, 2008; Funk, 2013; Hatemi et al., 2011b; Hatemi and McDermott, 2012a; Hibbing et al., 2014a; see also the edited volume by Hatemi and McDermott, 2011 and the book by Hibbing et al., 2013) with similar interest and reviews in other social sciences including economics (for example, Beauchamp et al., 2011b; Benjamin et al., 2012a), sociology (Freese, 2008, 2011; Freese and Shostak, 2009), and criminology (for example, Wright et al., 2015). Cross-sectional twin studies of political ideology have even become numerous enough to warrant a cross-national meta-analysis (Hatemi et al., 2014).

Twin studies of political ideology have relied on many different measures of ideology. These include multi-item scales like the Wilson–Patterson Attitude Inventory (Wilson and Patterson, 1968; for example, Alford et al., 2005), single-item measures (for example, Settle et al., 2009), latent variables extracted with factor analysis (for example, Bell et al., 2009), the Society Works Best index (Smith et al., 2011b; for example, Funk et al., 2013), and combinations of these measures (Funk et al., 2013). Each of these measurement strategies has found that genes play a role in the direction of political ideology, a result that was reaffirmed by meta-analysis (Hatemi et al., 2014).

The finding from twin studies that political ideology is heritable has also been confirmed using other methodologies. For example, research using molecular genetic measures from unrelated individuals has confirmed that genes play a role in political ideology by utilizing a genome-wide association methodology (Benjamin et al., 2012b; Hatemi et al., 2011a; 2014). There is also neuroscience evidence that there are structural brain differences between liberals and conservatives in the anterior cingulate cortex and right amygdala (Kanai et al., 2011). Combined with research into political attitudes using twins

raised apart (Bouchard et al., 2003) and extended family studies (Hatemi et al., 2010), there is convergent, multi-method evidence that genetic and biological factors do play a role in political attitudes.

In light of this, recent research has turned to identifying intermediate variables, or mediators, between genes and political ideology (for example, Smith et al., 2011b), such as personality traits (Verhulst et al., 2010) and cognitive ability (for example, Oskarsson et al., forthcoming). Instead of a mediating relationship, some researchers have suggested that personality and political ideology both arise independently from some of the same genetic influences in a process called pleiotropy (Hatemi and Verhulst, 2015; Verhulst et al., 2012a).

There are also genetic studies that focus on specific policy attitudes rather than ideology in general. Alford et al. (2005) disaggregated the Wilson–Patterson index and found heritability in all of the 28 individual policy areas that compose it (although the method they used did not allow for significance tests of these estimates). In a Canadian sample, Bell and colleagues (2009) examined the heritability of a number of factor-analytically derived issue domains and found heritability on religiosity/social conservatism, economic equality, competition/business, and ethnic/racial minorities but not on environmentalism or preference for an activist state on social issues. Verhulst et al. have variously distinguished social, economic, and military/defense attitudes (Verhulst et al., 2012b), religious, sex, out-group, and punishment attitudes (Verhulst et al., 2010), and social and religious ideology (Hatemi and Verhulst, 2015). Related research has shown that religiosity is heritable (for example, Eaves et al., 2008) and that genes link religiosity and political conservatism (Ludeke et al., 2013), but that this genetic link is especially strong between religiosity and social conservatism rather than economic conservatism (Friesen and Ksiazkiewicz, 2015).

There are also a number of cases in which attitudes in a specific domain have been shown to be heritable and to be related to a specific physiological mechanism, which may be related to the genetic effect. For example, there is a significant genetic component to support for abortion rights and gay rights (Eaves and Hatemi, 2008), and this set of attitudes has been linked physiological disgust-response (Smith et al., 2011a; Balzer and Jacobs, 2011) and neurological reactions to disgusting images (Ahn et al., 2014). Together, these results indicate that physiological and neurological disgust-response may mediate the genetic effect on these issue attitudes, although research has yet to address this possibility directly.

Foreign policy attitudes provide another example. This subset of political attitudes is heritable (Cranmer and Dawes, 2012) and has been linked to physiological startle response (Oxley et al., 2008). Perhaps relatedly, individual differences in fear dispositions have been linked by genetic mechanisms to negative out-group opinions, such as pro-segregation and anti-immigrant attitudes (Hatemi et al., 2013).

Political scientists have used neuroscience methods to demonstrate that egalitarianism and economic redistribution are linked to empathy (Dawes et al., 2012); they have also used physiological methods to show that egalitarianism and economic redistribution are linked to respondents' current blood glucose levels (Aarøe and Petersen, 2013; Petersen et al., 2014). Furthermore, although economic attitudes may be affected in the short term by major life events that increase financial risks, over time the genetic factors reassert themselves (Hatemi, 2013). Related research has shown that personality traits, some of which are face-validly related to economic and redistributive preferences, are also

heritable. These include preferences for giving and risk taking (Cesarini et al., 2009a), which may rely on somewhat different neural mechanisms in liberals and conservatives (Schreiber et al., 2013), and overconfidence in one's abilities (Cesarini et al., 2009b; Johnson and Fowler, 2011).

In sum, these results suggest that while there is value in studying genetic effects on aggregate political ideology, there are likely to be many pathways through which genetic effects are mediated and these mechanisms may vary across issue areas.

## GENES AND POLITICAL BEHAVIORS

Of course, not all biopolitical studies of political phenomena have focused on attitudes and beliefs. Besides political ideology, scholars have looked at the role of biological factors in vote choice. Fazekas and Littvay (2012) argue that genetic factors may account for dispositional differences between individuals in their voting decision strategy. In addition, physiological measures, like electrodermal activity in response to seeing a portrait of President Obama, predict independent variance in the strength of individuals' candidate preferences and support for the Affordable Care Act (Wagner et al., 2015). Insofar as these physiological measures are related to underlying genetic differences, they may reinforce the findings from twin studies of vote choice.

Voter turnout has been a debated topic of study in genopolitics. Turnout has been found to have a heritable component in studies using a twin methodology in the United States (Fowler et al., 2008), Australia (Hatemi et al., 2007), Canada (Bell et al., 2009), and Sweden (Cesarini et al., 2014). Genetic relationships have been found between voter turnout and an individual's sense of general efficacy (Littvay et al., 2011) and civic duty (Loewen and Dawes, 2012; Klemmensen et al., 2012a), as well as extraversion, personal control, and cognitive ability (Dawes et al., 2014). Interest in politics, a standard predictor of voter turnout, is heritable (Bell et al., 2009) and has a genetic correlation with external efficacy (Klemmensen et al., 2012b), which is also a predictor of turnout.

There is, however, a growing body of literature on identifying the specific genetic mechanisms underlying individual differences in turnout. Some have proposed that candidate, or specific, genes related to serotonin and dopamine regulation play a role in turnout (Dawes and Fowler, 2009; Fowler and Dawes, 2008), although others have raised doubts about these results. For example, Charney and English (2012: 2) declare in their response that genes "do not predict voter turnout," and that there are problems with all studies that claim to have found a particular gene variant for complex social behaviors "because the search for genes that could predict prevalent and devastating behavioral phenotypes such as schizophrenia and autism, not to mention global killers such as diabetes and hypertension, has to date been unsuccessful." This critique echoes the concerns of molecular geneticists who note that caution should be exercised regarding candidate gene studies until they are replicated in very large samples because of the danger of false positives for any particular single nucleotide polymorphism (Ioannidis et al., 2001; cf. Chabris et al., 2012). Nevertheless, subsequent research has replicated the associations between 5-HTTLPR, a serotonin transporter gene, and political participation and voter turnout (Deppe et al., 2013), as well as providing an explicit response to the critics alongside a second replication (Fowler and Dawes, 2013). Moreover, there is some evidence to

suggest that the effect of genetic factors in turnout and participation more broadly may be moderated by social factors such as friendship (Settle et al., 2010; see also Christakis and Fowler, 2014). Regardless of whether this particular candidate gene is ultimately found to be a robust predictor of voter turnout, future research should continue to identify genetic variants that are related to political phenomena and the environmental factors that moderate those effects.

## GENES AND OTHER POLITICALLY RELEVANT TRAITS

Finally, there are a number of other streams of genopolitics research not directly related to political attitudes or political participation, but that may nevertheless be of importance to political science research. Among the most important of these streams is genetic research that has looked at party identification, although with somewhat mixed results. Some researchers have found that the direction of party identification does not have a heritable component in the United States, but strength of partisanship does (Dawes and Fowler, 2009; Hatemi et al., 2009a; Settle et al., 2009). This contrasts with findings on ideology discussed above, some of which have explicitly tested the direction–intensity distinction and found that direction is heritable but intensity is not (Settle et al., 2009). However, in a Canadian sample, party identification and affect toward parties were found to have a genetic component (Bell et al., 2009). Moreover, recent research has suggested that whether genes affect the direction of party identification may vary within a country over time, increasing, for example, in the US context as a function of polarization in the political environment (Fazekas and Littvay, 2015). Thus, more research on the heritability of party identification is called for across diverse populations to determine how, when, and why genes play a role in this process.

Another stream of genetics research that may be of relevance to political scientists considers the role of genes in social networks. Recent work suggests that genes are related to features of a person's social network, including the number of people who consider the individual a friend, the degree to which an individual's friends are also friends with each other, and an individual's centrality in the network (Fowler et al., 2009). Genes also play a role in occupying a leadership position, although the specific genetic mechanisms for this process are unclear (De Neve et al., 2013). Moreover, friendship networks have been shown to be characterized by homophily, meaning that certain genetic variants are positively correlated among friends (Fowler et al., 2011).

Social trust is another phenomenon of relevance to political science, which has been shown to be heritable (Sturgis et al., 2010), as are individual differences in the perceived acceptability of dishonest actions (Loewen et al., 2013). The role of genes in social trust, as in other complex traits, depends on the population being studied and environmental factors in society. For example, Ojeda (forthcoming) found that the role of genes in social trust in the United States dramatically increased immediately following 9/11, an effect which gradually decayed over time. In attempting to identify possible mediators of the genetic effect on social trust, research has found genetic links to extraversion, personal control, and intelligence (Oskarsson et al., 2012). Physiologically, this may be driven by differences in oxytocin levels or oxytocin processing, as social trust has been experimentally manipulated by administering oxytocin in humans (Merolla et al., 2013). However,

no effect on social trust was found when considering nine polymorphisms in the oxytocin receptor gene (Apicella et al., 2010), suggesting that this physiological pathway requires further research.

Genetic factors have been shown to play a role in media consumption patterns (Kirzinger et al., 2012). This finding is particularly important for the studies of attitude stability and change because media consumption patterns will affect the kinds of information that a person is exposed to and may thereby affect the likelihood of attitude change during a political campaign. Moreover, physiological data has shown that liberals and conservatives differ in how they direct their attention (Dodd et al., 2011), with subsequent research showing that conservatives are more likely to attend to negative stimuli than liberals (Carraro et al., 2011; Dodd et al., 2012; Hibbing et al., 2014b). It has been hypothesized that these differences in negativity bias may be driven by the orientation toward change in general, rather than political conservatism specifically (Ludeke and DeYoung, 2014). Subsequent research has implicated attentional biases for emotional materials as an explanatory factor (Mills et al., 2014) and has integrated these findings into an evolutionary psychology framework (Petersen and Aarøe, 2014). Regardless of the source, negativity bias remains another meaningful difference between liberals and conservatives that appears to have a biological foundation.

Some researchers have proposed that biopolitics research, including behavioral genetics and neuroscience, can help us to better understand the role of gender in society (Hannagan, 2008). Behavioral genetic methods have been applied to developing better theories of the gender gap in political attitudes (Hatemi et al., 2009c) and the gender gap in political knowledge (Hannagan et al., 2014).

Finally, Hatemi and McDermott (2012b) have argued for using genetic methods to better understand and prevent political violence. By developing a clearer understanding of who is predisposed to participate in political violence and under what circumstances, it is possible to develop interventions to prevent violence from occurring. For example, subsequent research found that individual differences in the MAOA gene interacted with exposure to violence in youth to increase the likelihood of utilizing physical aggression in adulthood (McDermott et al., 2013).

In sum, the conclusion from this literature is clear: genes and biological processes play an important role in a wide variety of political traits and behaviors.

## THE FUTURE OF GENETIC RESEARCH IN POLITICAL SCIENCE

Increases in the number of conference panels, published articles in top political science journals, and scholars pursuing this area of research indicate that the study of genopolitics is gaining traction in our field and does not appear to be disappearing anytime soon. As discussed above, scholars have addressed many of the methodological criticisms raised regarding the assumptions of the classical twin design and the replication of candidate gene studies. However, what remains are concerns related to perceived biological determinism and the dangers of eugenics. With regard to determinism, this is a case where the pernicious effects of the false dichotomy between nature and nurture can be seen in political science. To our knowledge, no genetics scholar claims that the evidence supporting

the heritability of political traits implies that genes determine an individual's political beliefs or that the environment is inconsequential for political traits. In fact, behavioral and molecular genetic data clearly show that environments do account for a significant portion of the variance in political traits and that genetic effects are conditional on environmental factors. Thus, the objections regarding biological determinism are speaking past the research that has been and is being conducted by genopolitics researchers; those who raise these objections fundamentally misunderstand that genetic effects on complex traits are always conditional on environmental factors and that the research bears out this conclusion.

The possibility of genopolitics research being used to justify eugenic policies is another criticism that is sometimes raised as a reason to abandon the pursuit altogether. First, it is important to recognize that none of the scholars conducting empirical genetic research in political science have advocated for any genetic modification aimed at altering individuals' political predispositions (even if such modifications were technologically feasible, which they are not at present). The closest analog to this type of argument are the debates among bioethicists regarding "the ethical desirability of moral bioenhancement" (for a review, see Specker et al., 2014) and human genetic modification in general. These debates are of relevance to consternation about political eugenics, but they encompass bioethical dilemmas that cover a much wider range of issues than those of central concern to scholars of genopolitics. In discussing the possibility of human genetic modification, bioethicists are proactively engaging with issues that society will have to face as new technologies develop over the coming decades, regardless of whether political scientists pursue genopolitics research or not.

If instead the concern is that unscrupulous actors will abuse the findings of legitimate scientific research, then this is not an objection that is unique to genopolitics and is not one that should be used to bar scientific inquiry into the origins of political traits. Unease regarding the abuse of scientific research could just as easily be raised regarding research in practically all other areas of empirical political science, such as communication research that could be used to generate more effective propaganda, institutional research that could lead to more effective methods of control and oppression, and behavioral research that could result in voter suppression. In discussing the future of genopolitics research, we believe that the focus should be on how behavioral and molecular genetics research complements other areas of political science and how we can fine-tune our methods and replicate our findings – in other words, execute good science, as all scholars should.

Even with the rapidly growing genopolitics literature that uses increasingly diverse methodologies, perhaps the most pressing issue is that most of the genetic variation in political traits remains unaccounted for. Thus, future research should continue to identify new variables that account for unique genetic variance in political ideology and other political traits and may involve using better measures of existing theoretical pathways from genes to politics (for example, facets of the Big Five personality traits, rather than the broader factors) or attempting to identify novel theoretical pathways (such as cognitive style and executive functions; see Ksiazkiewicz et al., 2016).

Although the predominant view in the literature until recently was that personality traits precede political beliefs (for example, Mondak, 2010), some have challenged this view, arguing instead that personality and political beliefs co-emerge from common genetic roots using direction of causation models (for example, Verhulst et al., 2012a) and,

recently, longitudinal data in adolescent and adult samples (Hatemi and Verhulst, 2015). Genetically-informative studies can also provide insights into the role of environmental factors in political traits. Creating such datasets will likely require cooperation with existing longitudinal studies in genetically-informative populations (that is, twins, families, and unrelated individuals for whom genetic data is available). One strategy would be to survey the adult political traits of individuals for whom longitudinal personality data is available in adolescence. Another approach would be to cooperate with twin and family researchers to begin the measurement of political traits in adolescence and measure the development of these traits over time. These types of data could directly address the mediation versus pleiotropy controversy, not just for Big Five traits, but for different types of cognitive style and for executive functions.

We wish to emphasize that not all political traits are underpinned by the same predictors. The factors that give rise to political ideology, ideological certainty, strength of attitudes, interest in politics, participation in politics, partisanship, and sophistication are not necessarily the same. Moreover, the mechanisms which give rise to different types of political ideology also seem to differ. One fruitful pathway that should continue to be explored in the future is the use of psychophysiological methods to identify biological correlates of political traits, like startle sensitivity (for example, Oxley et al., 2008), disgust sensitivity (for example, Smith et al., 2011a), cortisol levels (for example, French et al., 2014), and negativity bias (Carraro et al., 2011; Hibbing et al., 2014b). One previously unconsidered pathway may be the study of individual differences in resting metabolic rate, as these may affect the subjective experience of hunger, which has been linked experimentally to redistributive attitudes (for example, Petersen et al., 2014).

As with political ideology, future research into the biological underpinnings of political participation may benefit from distinguishing different types of participation from each other. One relevant dimension is whether a participatory act has a social element, as participatory acts with and without social elements are predicted by distinct personality traits (for example, Mondak et al., 2010). Insofar as these personality traits have different genetic underpinnings, it may be fruitful to distinguish them in behavioral genetic and psychophysiological research. Moreover, better measures of personality traits, such as scales with coverage of the facets of the Big Five, may be necessary to fully elucidate the genetic pathways that lead to political traits and behaviors, as indicated by the stronger genetic links between political ideology and cognitive style (a narrower personality trait) than political ideology and openness to new experiences (one of the broader, Big Five factors).

Although the focus of our discussion has been behavioral genetic studies and occasionally psychophysiological studies, molecular genetic methods also present new (albeit significantly more costly) opportunities for genopolitics research. These include not only candidate genes studies or genome-wide association studies, which could be used to identify particular genetic variants that are associated with political traits, but also newer methods like genome-wide complex trait analysis, which could be used to corroborate the estimates of heritability obtained in twin studies of political traits. One challenge in pursuing molecular genetic methods is that in order to find replicable results, very large sample sizes resulting from international research consortia are often needed (for example, Rietveld et al., 2013, 2014).

In addition to finer-grained methods to measure genes, the study of biopolitics would benefit from a more sophisticated approach to environmental factors. Some research

has begun to show that gene expression in politics is affected by life events (Hatemi, 2013), major events in society (Ojeda, forthcoming), and cross-national differences (Klemmensen et al., 2012a, 2012b; Nørgaard et al., n.d.). Research that focuses on environmental factors in genetically-informative populations should be actively pursued in the future. Moreover, these same methodologies should be applied to understanding how environmental variation at the sub-national level and across demographic groups, such as gender, race, and class, may affect the pathways through which genes affect political traits. These approaches have the potential to bridge the gap between genopolitics and the study of political institutions by showing how institutional factors affect the expression of genes in a group, region, or nation.

To wit, the effect of genes on political ideology and the pathways through which that effect manifests itself will differ across populations, places, and times, just as the effect of groups, socialization, and personality change over time. For example, while authoritarianism correlates with support of the capitalist system and endorsement of conservative policies in the United States, in former Soviet countries this same personality trait correlates with support for state control of the economy (McFarland, 1998). Similar findings have shown that while need for cognitive closure correlates positively with social and economic conservatism in Western Europe, it correlates positively with social conservatism and negatively with economic conservatism in Eastern Europe (Kossowska and Van Hiel, 2003). Kossowska and Van Hiel (2003) argue that this is because the content of traditional social values in Eastern and Western Europe is much more similar than the content of traditional economic values. In the West, where market institutions are the status quo, change is represented by movement towards greater government intervention in the economy, and so need for cognitive closure correlates positively with the maintenance of traditional market-oriented institutions. By contrast, in Eastern Europe, which has a long history of centrally-planned economic activity, change is represented by movement towards a free market system, and so need for cognitive closure correlates positively with the maintenance of traditional centrally-planned institutions. Just as the effect of epistemic motives, like the need for cognitive closure, differs as a function of the discursive superstructure, so too may genetic effects differ as a function of social and political institutions (see Nørgaard et al., n.d. for evidence on this point in a cross-national comparison of the United States and Denmark).

Finally, epigenetics, the process by which genetic effects can be amplified or muted in response to environmental factors, presents perhaps the greatest challenge and the greatest opportunity to the field of genopolitics going forward (see Feil and Fraga, 2012 and Mazzio and Soliman, 2012 for reviews of epigenetics). Over the next decade, political scientists will need to actively engage with this literature in order to develop a better understanding of how environmental influences, like political institutions, political cultures, socialization, friendships, the experience of systemic discrimination, and so on, influence the expression of genes with relevance to political traits.

In sum, there is a bright future for the field of genopolitics, with a plethora of new methodologies and technologies that are only now being applied to political phenomena for the first time. When informed by insights generated from decades of rigorous political science research, these new approaches have the potential to dramatically broaden the horizons of the discipline and contribute to our understanding of the nature of political life.

## NOTES

1. Although behavioral genetics scholars had shown that genes play a role in political attitudes in several studies prior to this piece (for example, Eaves and Eysenck, 1974; Martin et al., 1986), the publication of this piece in the *American Political Science Review* was a watershed moment that set off a series of high-profile publications in the area of genes and politics.
2. Specifically, half of their segregating DNA.
3. These same scholars have recently critiqued other techniques used in contemporary political genetics research, such as candidate gene studies (Charney and English, 2012). These critiques are addressed below.
4. There is also a fourth term that is implicit in this model, *D*, which accounts for non-additive genetic effects that result from dominance. However, inclusion of this term makes the model unidentified when using data only on MZ and DZ twins. Thus, *D* is usually fixed at zero in political science applications, resulting in an ACE model. When considering a single base pair on the genome where there is variability between people, where we label one variant *a* and the other variant *A*, an individual could have any one of three genotypes: *aa*, *aA*, or *AA*. A non-additive effect occurs when the effect of moving from *aa* to *aA* is not equivalent to the effect of moving from *aA* to *AA*. Thus, a large non-additive component (*D*) in a twin model indicates that non-additive effects are present at many of the base pairs that affect the trait of interest.
5. This is why some behavioral genetic researchers choose to label the *A* component *H*, for heritability, instead.

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