Are Political Orientations Genetically Transmitted?

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e test the possibility that political attitudes and behaviors are the result of both environmental and genetic factors. Employing standard methodological approaches in behavioral genetics—specifically, comparisons of the differential correlations of the attitudes of monozygotic twins and dizygotic twins—we analyze data drawn from a large sample of twins in the United States, supplemented with findings from twins in Australia. The results indicate that genetics plays an important role in shaping political attitudes and ideologies but a more modest role in forming party identification; as such, they call for finer distinctions in theorizing about the sources of political attitudes. We conclude by urging political scientists to incorporate genetic influences, specifically interactions between genetic heritability and social environment, into models of political attitude formation.

Why do people think and act politically in the manner they do? Despite the foundational nature of this question, answers are unfortunately incomplete and unnecessarily tentative, largely because political scientists do not take seriously the possibility of nonenvironmental influences. The suggestion that people could be born with political predispositions strikes many as far-fetched, odd, even perverse. However, researchers in other disciplines—notably behavioral genetics—have uncovered a substantial heritable component for many social attitudes and behaviors and it seems unlikely that political attitudes and behaviors are completely immune from such forces. In this article, we combine relevant findings in behavioral genetics with our own analysis of data on a large sample of twins to test the hypothesis that, contrary to the assumptions embedded in political science research, political attitudes have genetic as well as environmental causes.

Testing this hypothesis is important for two reasons. First and most broadly, as behavioral scientists we need to analyze all possible shapers of behavior, not just a select few. Second, a more complete understanding of the sources of attitudes and behaviors will help us to sort through existing puzzles of considerable interest to political scientists. One example is political ideology. Why is a reasonably standard left–right spectrum so widely applicable cross-culturally and over time? The universal left–right elements of belief systems around the world and over the decades is difficult for behavioralists to explain. But if there is a genetic component to political ideologies, if constraints on belief systems come not just from intellectualization or indoctrination but from something deeper, the concept of ideology takes on greater meaning and the commonality of ideology becomes easier to understand.

ATTITUDE FORMATION

Debates concerning the source of political attitudes revolve primarily around the question of whether early childhood factors have lasting relevance or whether these factors tend to be overwhelmed by more proximate events. Survey responses to political items presumably reflect attitudes and are thought to be a combination of longstanding “predispositions” and more recent “off-the-top-of-the-head” considerations (Zaller 1992, chaps 1–3; also see Converse 1964). Alternatively, an “on-line” pattern of processing could allow new incidents to ratchet affect one way or another from previously existing summary locations (see Lodge, McGraw, and Stroh 1989). Regardless, proximate forces include recent conversations and experiences, question-wording, priming from previous questions, and a variety of similar factors. Predispositions, on the other hand, are thought to be a “distillation of a person’s lifetime experiences, including childhood socialization and direct involvement with the raw ingredients of policy issues” (Zaller 1992, 23). Great interest exists in determining the relative clout of the early as opposed to the late environment but no interest has been displayed in determining the relative clout of environmental as opposed to genetic variables.

A parallel conclusion applies to research on individual attitudes rather than survey responses generally. For example, the consensus among those who study tolerance is that the extent to which individuals are tolerant hinges on a combination of “antecedent conditions and contemporary information” (Marcus et al. 1995). Antecedent conditions, in turn, are believed to

1 Evidence consistent with an evolutionary theory of political behavior is found in Brewer 2000, Hibbing and Alford 2004, and Orbell et al. 2004.

2 To his credit, Zaller (1992) goes on to acknowledge a possible role for “inherited” traits in shaping predispositions (23).
be shaped by “personal circumstances” such as “family, neighborhood, region . . . and early group experiences” (Marcus et al. 1995, 5; for more on the importance of long established proclivities, or antecedent conditions, see Stouffer 1955). Typically, no role for genetically-induced tendencies is considered (for an exception, see Monroe 2004, chap 6).

More broadly, the literature on political socialization has long revolved around the question of the effects of early as opposed to late environmental forces. Early political socialization researchers (e.g., Easton and Dennis 1969, Greenstein 1960, Jennings and Niemi 1968, and Searing, Schwartz, and Lind 1973) and the authors of The American Voter (Campbell et al. 1960) presented arguments and evidence supporting the primacy of early events. Later researchers, however, questioned the value of early childhood socialization and provided evidence that judgments about more recent conditions and occurrences can dramatically alter preferences we might have held as children and adolescents (see, e.g., Fiorina 1980; for good summaries of the debate over the relative importance of early and late environmental events, see Cook 1985; Merelman 1986, and Sears 1989). In the last 50–60 years, the emphasis in the literature has gone from personality studies (Adorno et al. 1950; Eysenck 1954; Laswell 1930), to ideological and childhood socialization studies, to the effects of media frames, perceptions of current conditions, and other types of contemporary information. In fact, for the past couple of decades research on political socialization has been suffering through a “bear market” (Cook 1985), and studies of personality, while experiencing a remarkable comeback in psychology (for an introduction, see Wiggins and Trapnell 1997), have been largely absent from political science since McCloskey’s (1958) work in the 1950s on the conservative personality. Thus, political science debates concerning the source of political attitudes and behaviors have been over timing, over whether attitudes and behaviors are primarily shaped early in life or by more proximate occurrences. Conspicuously absent is consideration of the possibility that certain attitudes and behaviors may be at least partially attributable to genetic factors.

**MODERN BEHAVIORAL GENETICS**

But what is the physical process by which a genetic allele could shape a political attitude? If there is any connection at all, is it not that the effect is so small that it can be safely ignored? And even if this is not the case, in light of potentially troubling normative implications such as biological determinism, is it not best to ignore relationships between genes and social behavior? It is difficult for many outside the biological sciences to understand how it is even possible for genes to influence behavior, so a brief discussion is in order. Genes provide instructions for the production of proteins, which are built and identified by a specific combination of amino acids (which in turn are constructed from complex organic molecules). As such, each protein has a chemical sequence that then interacts with other chemicals in the body, sometimes reacting directly with these other chemicals but often serving as enzymes that facilitate but are not themselves altered by chemical reactions. If a gene coding for a particular enzyme is absent, the chemical reaction it is meant to enhance will occur with much less efficiency. For example, a gene for the enzyme tryptophan hydroxylase-2 (Tph2) facilitates production of the neurotransmitter serotonin in the brain, but a certain form of this gene (which varies from the standard form by a single amino acid) produces about 80% less serotonin and people with this mutant allele appear to be significantly more likely to suffer from unipolar depression (Zhang et al. 2005).

Still, the connection is rarely so simple that a given genetic allele can be seen as causing a certain behavior. More typically, findings in modern behavioral genetics reveal the effect of genes to be interactive rather than direct, let alone determinative. To provide one illustration, in humans there is a gene on chromosome 17 involved with serotonin uptake (5-HTT). As is often the case with genes, 5-HTT has a long allele and a short allele. Mice have a parallel gene, and in that species the short form had previously been connected to listless, depressive behavior. Scientists were eager to determine if such a correlation between the short form of 5-HTT and depression was present in humans. In a long-term study of the health records of nearly 1,000 New Zealanders whose 5-HTT alleles were known, it was found that major episodes of depressive behavior were not much more prevalent among those with the short form. But then the researchers combined genetics and the environment; specifically, they interacted each subject’s 5-HTT allele with the number of high-stress events (romantic calamities, bankruptcies, deaths of loved ones, etc.) experienced in that individual’s life. They found that those who had a high number of such events and who had the short form of 5-HTT were significantly more likely to display behaviors associated with depression compared to either those experiencing few high stress events or those with the long form who suffered through a comparably large number of high-stress events (see Caspi et al. 2003).

In this particular case, genotype did not make people behave a certain way; rather, it influenced the extent to which their behavior was contingent on the environment—and this pattern likely will apply to all sorts of other human activities. Whether the behavior of interest is depression, cooperation, fear response, or susceptibility to drug addiction, some people are more sensitive than others to particular features of their environment, and genetics, far from determining behavior, influences its sensitivity. Genetics makes the mood of some people far more dependent on the extent to which their lives have been beset with difficulties and it likely makes some people’s political attitudes far more contextually dependent than others. In other words, the connection between genes and attitudes may not involve specific attitudes as much as the flexibility of those attitudes (Is abortion always wrong, or does it depend?). The issue is not nature versus nurture but...
the manner in which nature interacts with nurture (see Marcus 2004 and Ridley 2003).

**MONOZYGOTIC AND DIZYGOTIC TWINS**

The process of identifying in the laboratory the precise genes responsible for given human behaviors (especially those behaviors that do not have corollaries in lab-friendly animals such as mice) is extremely challenging. Fortunately, even without identifying the genes responsible, it is possible to compile information on the matter of most concern to social scientists: the extent to which attitudes and behaviors have a genetic component. The relevant procedures center on comparisons of monozygotic (MZ; frequently but erroneously called identical) twins and dizygotic (DZ; fraternal) twins.

MZ twins develop from a single egg, fertilized by a single sperm, and share an identical genetic inheritance. DZ twins develop from two separate eggs, fertilized by two separate sperm, and are in effect simply two siblings that happen to be born simultaneously. As such, DZ twins share the same average of 50% of genetic material as do any two biological siblings. It is this fixed ratio (two to one) of genetic similarity between MZ and DZ twins, and the contrasting average equivalence of environment influence, that provides most of the power of twin designs. It is important to appreciate that the assumption of environmental equivalence is one of equivalence across types of twins, not across pairs of twins or across twins within a given pair. For example, there is undoubtedly at least some variability in parental socialization across siblings, even those of identical age, but across multiple twin pairs the assumption is that this variability is essentially equal for the MZ and the DZ pairs.

This assertion that the effect of genetics is measurably distinct for MZ and DZ twins, while the effect of the environment is either equivalent or at least randomly distributed around equivalence, is crucial to everything that follows from twin research. It is important therefore to raise and consider the criticisms of this fundamental assumption. The arguments come in two essential varieties. The first is that MZ twins, genetics aside, experience a more similar environment because they are treated more similarly than are DZ twins. This would seem particularly telling for childhood socialization, where, for example, parents might show less of a tendency to treat MZ twins as individuals compared to DZ twins. The second is that MZ twins, genetics aside, interact with each other more throughout life than do DZ twins. This would seem to be of particular importance for adult socialization, where closer adult contact between MZ twins might lead us to expect a greater degree of environmentally induced similarity than we would see for the more distant DZ twins.

Both caveats have been subject to sustained and varied investigation and neither has been found to hold up under empirical scrutiny. The argument of more similar treatment fails on several fronts. Parents frequently miscategorize their twins (DZ twins are often believed by their parents to be MZ twins) and the differential correlation persists in these instances of miscategorization. In other words, the degree of correspondence between MZ twins surpasses that of DZ twins even in the large subpopulation of twins thought by their parents to be MZ twins (Bouchard and McGue 2003; Bouchard et al. 1990; Plomin 1990). The contention that MZ twins have closer or more frequent contact than DZ twins turns out to be at best irrelevant. The correlation between the frequency of contact between twins and the similarity between twins on all attitudinal and behavioral variables tested, including conservatism, is slight and actually negative (Martin et al. 1986). In other words, twins in greater contact with their cotwins are not more likely to share the same attitudes and behaviors, so even if MZ twins have more contact than DZ twins, this contact is not the cause of any elevated correlations. But the most powerful refutation of both of these criticisms comes in recent studies utilizing MZ and DZ twins raised apart. These studies uniformly validate MZ and DZ differences found in earlier studies of twins raised together. Arguments about the relative degree of shared environmental effects between MZ and DZ twins simply offer no credible explanation if the twins in question have been raised apart (Bouchard 1998; Bouchard et al. 1990). In effect, this naturally occurring, if uncommon, condition provides precisely the sort of laboratory control that we would want in an experimental setting.3

Other evidence against the exclusive environmental argument is that the empirical results suggest MZ twins reared together are often less likely to share behavioral traits with their twins than are MZ twins reared apart, presumably because of extra efforts to establish distinct identities when the twins live together. In addition, as adult MZ twins living apart age, they tend to become, not less, similar (Bouchard and McGue 2003), a finding that is difficult to reconcile with the belief that only the environment matters. Interestingly, this precise effect is predicted in an early landmark criticism of behaviorism and the conditioned response research on animal behavior that formed its empirical core. Over time, substantial anomalies began to accumulate in this research pointing toward a primacy for some nonenvironmental behaviors. Breland and Breland (1961) summarized this tendency with the phrase “learned behavior drifts toward instinctive behavior” (684).

Given the genetic differences and environmental similarities of the two types of twins, for any trait that is partly heritable the tendency for MZ twins to share that characteristic should be stronger than the tendency for DZ twins to share that characteristic. In contrast, characteristics that arise purely from the environment, whether shared by the twins, as would typically be the case for parental socialization, or not

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3 To explain this finding, opponents would need to argue that adoption agencies are more likely to place MZ twins in similar homes than they are to place DZ twins in similar homes. In fact, information on twin zygosity is typically unavailable to those making placement decisions, and even if it were available, it seems highly unlikely that it would factor into their decisions.
shared by the twins, as would be the case for many adult experiences, should not generate any significantly different patterns when we contrast MZ and DZ twins (see Eaves, Eysenck, and Martin 1989 and Plomin et al. 2001 for a thorough discussion of the relevant statistical techniques).

The procedures involved with the twin methodology are standard fare in behavioral genetics but are not familiar to most political scientists, so it is appropriate that we explain the basic terminology, theory, and technique in some detail. Influences on an individual trait, whether it is a political attitude or a physical characteristic, are typically divided into two broad groups—heredity (H) and environment (E). The total variation in a trait can thus be represented as the sum H + E. Heredity is the impact of genetic inheritance on trait variation. In the case of a physical characteristic such as adult height, this would be the proportion of the total variation in height across individuals due to the variation across individuals in the multiple genes that control ultimate physical height. For any one individual, the source of this genetic influence is relatively well defined, as on average 50% of our genes come from our mother and 50% come from our father. This leads to the fact that biological children of tall parents are more likely to be tall than are the biological children of short parents, though even for a relatively straightforward additive physical trait like height, the relationship is far from determinative.

“Environment” is all of the nongenetic external factors that influence trait variation across a population. These influences range broadly from the earliest biological environment of the womb, to the physical environment of a childhood house, to the social environment of the adult workplace. In the case of adult height, some of the obvious environmental factors are prenatal nutrition, the adequacy of childhood and adolescent diet, and exposure to chemical agents that can inhibit growth.

Environmental influences can be further divided into two subcategories: the shared environment and the unshared, or unique, environment. The shared environment is all of the shared external influences that we would typically think of as leading to trait similarity between individuals. Siblings, for example, might share similar childhood environments, including similar parental interactions, a similar physical environment, and similar nutrition. If the siblings happen to be twins, they would also share a more similar prenatal environment.4 In the case of adult height, a shared environmental factor, such as a regional diet limited in protein and specific nutrients, could lead to similarity in height across the entire population of a region.

The unshared environment is all of the distinctive external influences that we would typically think of as leading to trait dissimilarity across individuals. While much of the early childhood environment, for example, is similar across siblings, much is nonetheless variable. Siblings differ in diet, disease exposure, peer influences, and a host of other unique experiences. Even twins, whose childhood environment is made increasingly similar by virtue of their identical age, are exposed to substantial unique external influences. With the shift to adult life, the share of unique influences on siblings increases sharply, as peer, workplace, family, and physical settings typically diverge.

In the classic political science studies of socialization (see, especially, Jennings and Niemi 1968, 1991 and Tedin 1974), the focus has been on the correlation between the attitudes of parents and their children. In terms of the three sources of trait variability outlined above, as informative as it is, this design does not allow for an unambiguous estimation of any of the three categories. The correlation between a parent and a child arises from a combination of shared genes, shared environment, and parental socialization (an indirect form of shared environment in which the parent’s attitudes provide a path from the parent’s environment to the child’s environment), all of which are pressures toward similarity in parent–child attitudes. The failure of this parent–child correspondence to reach +1.0 presumably reflects the pressure toward dissimilarity coming from the unshared environment, but since the genetic similarity of a parent–offspring pair is only .5, there is as much genetic dissimilarity as there is similarity. Thus, trait dissimilarity, like trait similarity, is an underdetermined mixture of genetic and environmental influences. Our inability to tease apart genetic heritability and environment, whether shared or unshared, in these parent–child studies is a direct result of the fact that there is no measured variation in genetic similarity across the data set of parent–child pairs (i.e., all biological offspring share the same average of 50% of the variable genetic code with each parent).

This inability of standard parent–child observations to distinguish genetic heritability from parental socialization (or other features of the shared environment) is something that has long been understood, but largely ignored in modern social science. Fortunately, twins provide a powerful “natural experiment” by introducing known genetic variation into analyses of the sources of trait variability. By shifting the focus from the similarity between parents and offspring to the similarity between two siblings, we can take advantage of the fact that some siblings vary in well-known ways in the degree of their genetic correlation.

POLITICS AND GENETICS: PREVIOUS FINDINGS AND OUR EXPECTATIONS

Comparisons of the correlations of MZ and DZ twins on a wide variety of variables have been conducted, with intriguing results. Using appropriate modeling techniques including controls for parental traits and assortative mating, it is possible to partition the explanatory powers of heredity, shared environment, and
nonshared environment on any given variable. These techniques have been valuable for epidemiological traits, intelligence, personality, social attitudes such as those connected to religion, psychological interests, and behaviors such as risk-taking propensities (for a thorough review, see Bouchard and McGue 2003). Of most interest to us are the findings pertaining to social attitudes and behaviors. At first, researchers were so confident that social attitudes were not heritable that they employed such items as controls. Quickly they discovered that other controls would have to be found because most social attitudes consistently displayed a surprising measure of heritability (see, e.g., Crelia and Tesser 1996, Scarr and Weinberg 1981, and Tesser 1993).

Political attitudes were never a central focus in this research stream but many of the patterns found in other social attitudes should be present for political attitudes as well, and this assumption guided the formulation of our expectations. Since the social attitudes tested to date have demonstrated a strong heritable component, frequently stronger than attitude covariance attributable to shared environment, we predict that political attitudes will also be heavily heritable. Heritability estimates calculated by previous researchers for attitudes associated with psychological conservatism are quite high, while the relevant models typically show little or no effect for shared environment (the remainder is likely the result of nonshared environmental factors). Notably, these findings come from studies of twins in settings as disparate as Australia, Virginia, and Minnesota, and the findings of the Minnesota study, utilizing twins reared apart, conform well to the other studies of twins raised together (for a summary, see Bouchard and McGue 2003). Careful studies of adopted children confirm the finding that genetics matter more than parentally created environment in influencing social attitudes and behaviors, personality traits, and intelligence.

We further predict that attitudes on political issues tracking most closely to central personality traits should be the most heritable since personality traits are generally heritable and since the heritability of social attitudes is likely derivative of the heritability of various personality traits (see Bouchard and Loehlin 2001 and Eaves, Eysenck, and Martin 1989). For example, one of psychology’s “Big 5” personality traits is general “openness” and it seems likely degree of openness is relevant to the political arena as well. Liberals and conservatives, on average, differ in their openness to atheism, homosexuality, communism, immigration, and countercultural activities. These differences may be entirely due to enculturation, but then again, they may not be, and we will never know without testing for the effects of genetics.

Based on behavioral geneticists’ study of religion, it seems that group identification is something that is heavily influenced by the environment, especially shared environment, and is mostly unconnected to genetics. Children of Methodists are likely to be Methodists not because there is a gene for Methodism or even a personality particularly oriented toward Methodism, but because of parental socialization. Thus, even as attitudes connected to religiosity and religious beliefs and activities (e.g., Sabbath observance, church authority, belief in heaven, religious fundamentalism, frequency of attendance) were found to be shaped more by genetic inheritance than by parental views on those issues (for details, see Bouchard et al. 1999, Eaves, Martin, and Heath 1990, Maes et al. 1999, and Martin et al. 1999), identification with a particular religious group was shaped more by socialization and almost not at all by genetics. We expect to find a similar pattern with political party identification. Children are eager to belong to the groups their parents belong to and parents are frequently eager to encourage children in this regard. Assuming these identifications have some stickiness into early adulthood, our core expectation is that party identification will be influenced more by parental socialization (shared environment) than by genetic inheritance but that this pattern will be reversed for political attitudes with inheritance playing a role at least as large as the shared environment. By predicting a large influence for genetic inheritance, we depart from typical behavioralist expectations anticipating that political attitudes will be predominantly influenced by environmental factors, rendering genetic inheritance largely, if not completely, inconsequential.

**DATA AND METHODS**

Since twin studies have not been conducted by political scientists, political attitudes have been at best a sidelined, and properly refined measures of political variables have not been constructed and employed (the heritability of political behavior has not been analyzed at all). Nonetheless, some previously employed variables in twin studies have political relevance. For example, the heritability of conservatism is frequently assessed (see, e.g., Bouchard et al. 1990, Eaves, Eysenck, and Martin, 1989, and Martin et al. 1986), and even though conservatism is viewed by the scholars who do twin studies more as a psychological trait than a political ideology, measures of it include political items.

Of most relevance here is the Wilson-Patterson (W-P) Attitude Inventory. This inventory is administered by presenting subjects with a short stimulus.
phrase such as death penalty or royalty and eliciting a simple agree, disagree, or uncertain response. The broadest version of the W–P inventory includes 50 items, 25 of which contribute positively to the conservatism score and 25 of which contribute negatively to the conservatism score. While some of the items relate to a heavily social conception of conservatism—for example, pajama parties, nudist camps, computer music, and horoscopes—others have a much more direct political content—for example, disarmament, socialism, patriotism, and death penalty. Studies typically utilize reduced sets of W–P items or modify individual items to better suit the country in which the items are being administered. For political science this presents two frustrations. The list of politically relevant items is tantalizing but limited and unfocused, and the results are often presented only for the entire combined scale, making it difficult to assess the contribution of the directly political items to the overall index of heritability.

We were granted access to the data for the W–P items in the United States and were able to conduct comparable, though more limited, twin correlation analyses from published results of an Australian study. The U.S. study included information on thousands of twin pairs in Virginia, supplemented with twin pairs recruited through the cooperation of AARP. A subset of these twins and their relatives has been asked questions regarding their social attitudes, including numerous items from the W–P inventory.

A brief explication of twin methodology should help readers make independent sense of the tables. The standard techniques in behavioral genetics are based on correlation analysis (in the case of limited response items like the W–P inventory, the actual measure is the polychoric correlation coefficient, a technique that is appropriate when individual subjects are using a limited set of categories to express location on what is in fact a continuous trait). The correlations are computed separately for male/male and female/female twin pairs to provide an appropriate comparison, since all MZ twins are same-sex pairs, while DZ twins are a mix of same-sex and opposite-sex pairs (in other words, female/male DZ twin pairs are excluded from the analysis). Without this control, the presence of any male/female differences would spuriously deflate the correlations for DZ pairs relative to the same-sex MZ pairs.

Heritability is typically estimated by subtracting the correlation for DZ pairs from the correlation for MZ pairs and then doubling the resulting difference. At one extreme, if the correlations are the same for MZ and DZ pairs, suggesting that genetic similarity plays no role in similarity for that particular trait, then the result will be an estimate of heritability of zero. At the other extreme, a purely genetic additive trait should produce a correlation of .5 for DZ pairs and 1.0 for MZ pairs, resulting in an estimate of heritability of 1.0 (1.0 − .5 = .5, and 2 × .5 = 1.0). In a similar way, we can estimate the influence of shared environment, as opposed to shared genetic material, by doubling the correlation for DZ pairs and then subtracting the correlation for MZ pairs. Again, a purely genetic additive trait should produce a correlation of .5 for DZ pairs and 1.0 for MZ pairs, resulting in an estimate of the impact of shared environment of zero (2 × .5 = 1.0, and 1.0 − 1.0 = 0). At the other extreme, if the correlations are the same for MZ and DZ pairs, suggesting that genetic similarity plays no role in similarity for that particular trait, then the result will be an estimate of the impact of shared environment that is equal to the MZ or DZ correlation (e.g., if MZ = DZ = .4, then 2 × .4 = .8, and .8 − .4 = .4). Whatever is left over is taken to be attributable to the unshared environment.

THE HERITABILITY OF POLITICAL ATTITUDES

Table 1 contains the results of a standard polychoric correlation analysis for the 28 W–P items available in the Virginia 30K data set and for a select set of additional items to provide some sense of perspective for the level of these correlations. Even the quickest glance at the results in Table 1 is enough to set aside the traditional view that genes do not play any role in explaining political attitudes. All 28 of the MZ correlations are larger than their corresponding DZ correlations, and in every case the difference is statistically significant at the .01 level. Far from typically being at or near zero, none of the 28 heritability estimates falls in the single digit range, and more than half of the 28 items have heritability estimates of .3 or more. Heritability ranges from a high of .41 to a low of .18, all suggesting that the influence of heredity on political attitudes is very real, and given the diverse range of items included here, this genetic influence is also pervasive. So the view that heritability of social and political attitudes will be nonzero but small relative to shared environment is also called into question. We see from Table 1 that the
TABLE 1. Genetic and Environmental Influences on Political Attitudes: The 28 Individual Wilson–Patterson Items

<table>
<thead>
<tr>
<th>Attitude Item</th>
<th>MZ</th>
<th>DZ</th>
<th>Heritability, $z$ for (MZ – DZ)</th>
<th>Shared Environment, $z$ for (2 × DZ) – MZ</th>
<th>Unshared Environment, $z$ for (1 – MZ)</th>
<th>$z$ for (MZ–DZ) Difference$^a$</th>
</tr>
</thead>
<tbody>
<tr>
<td>School Prayer</td>
<td>0.66</td>
<td>0.46</td>
<td>0.41</td>
<td>0.34</td>
<td>0.72</td>
<td>9.83</td>
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<td>Property Tax</td>
<td>0.47</td>
<td>0.27</td>
<td>0.41</td>
<td>0.06</td>
<td>0.53</td>
<td>7.66</td>
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<td>Moral Majority</td>
<td>0.42</td>
<td>0.22</td>
<td>0.40</td>
<td>0.03</td>
<td>0.58</td>
<td>7.16</td>
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<td>Capitalism</td>
<td>0.53</td>
<td>0.34</td>
<td>0.39</td>
<td>0.14</td>
<td>0.47</td>
<td>7.85</td>
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<td>Astrology</td>
<td>0.48</td>
<td>0.28</td>
<td>0.39</td>
<td>0.09</td>
<td>0.52</td>
<td>7.39</td>
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<td>The Draft</td>
<td>0.41</td>
<td>0.21</td>
<td>0.38</td>
<td>0.02</td>
<td>0.59</td>
<td>6.94</td>
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<td>Pacifism</td>
<td>0.34</td>
<td>0.15</td>
<td>0.38</td>
<td>–0.03</td>
<td>0.69</td>
<td>6.43</td>
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<td>0.37</td>
<td>0.07</td>
<td>0.66</td>
<td>6.89</td>
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<td>0.30</td>
<td>0.36</td>
<td>0.12</td>
<td>0.52</td>
<td>6.91</td>
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<td>0.25</td>
<td>0.36</td>
<td>0.07</td>
<td>0.57</td>
<td>6.53</td>
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<td>Foreign Aid</td>
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<td>0.23</td>
<td>0.35</td>
<td>0.06</td>
<td>0.59</td>
<td>6.42</td>
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<tr>
<td>X-Rated Movies</td>
<td>0.63</td>
<td>0.46</td>
<td>0.35</td>
<td>0.28</td>
<td>0.57</td>
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<td>Immigration</td>
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<td>0.33</td>
<td>0.12</td>
<td>0.55</td>
<td>6.20</td>
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<td>Women's Liberation</td>
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<td>0.33</td>
<td>0.13</td>
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<td>6.83</td>
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<tr>
<td>Censorship</td>
<td>0.40</td>
<td>0.29</td>
<td>0.30</td>
<td>0.10</td>
<td>0.60</td>
<td>5.36</td>
</tr>
<tr>
<td>Living Together</td>
<td>0.67</td>
<td>0.52</td>
<td>0.30</td>
<td>0.37</td>
<td>0.33</td>
<td>7.54</td>
</tr>
<tr>
<td>Military Drill</td>
<td>0.38</td>
<td>0.24</td>
<td>0.29</td>
<td>0.09</td>
<td>0.62</td>
<td>5.24</td>
</tr>
<tr>
<td>Gay Rights</td>
<td>0.50</td>
<td>0.46</td>
<td>0.28</td>
<td>0.32</td>
<td>0.40</td>
<td>6.26</td>
</tr>
<tr>
<td>Segregation</td>
<td>0.38</td>
<td>0.24</td>
<td>0.27</td>
<td>0.11</td>
<td>0.62</td>
<td>4.83</td>
</tr>
<tr>
<td>Busing</td>
<td>0.43</td>
<td>0.30</td>
<td>0.26</td>
<td>0.16</td>
<td>0.57</td>
<td>4.92</td>
</tr>
<tr>
<td>Nuclear Power</td>
<td>0.42</td>
<td>0.29</td>
<td>0.26</td>
<td>0.16</td>
<td>0.58</td>
<td>4.84</td>
</tr>
<tr>
<td>Democrats</td>
<td>0.47</td>
<td>0.34</td>
<td>0.26</td>
<td>0.21</td>
<td>0.53</td>
<td>4.96</td>
</tr>
<tr>
<td>Divorce</td>
<td>0.47</td>
<td>0.34</td>
<td>0.26</td>
<td>0.21</td>
<td>0.53</td>
<td>4.99</td>
</tr>
<tr>
<td>Abortion</td>
<td>0.64</td>
<td>0.52</td>
<td>0.25</td>
<td>0.39</td>
<td>0.63</td>
<td>6.23</td>
</tr>
<tr>
<td>Modern Art</td>
<td>0.43</td>
<td>0.30</td>
<td>0.25</td>
<td>0.18</td>
<td>0.57</td>
<td>4.69</td>
</tr>
<tr>
<td>Federal Housing</td>
<td>0.36</td>
<td>0.26</td>
<td>0.20</td>
<td>0.16</td>
<td>0.64</td>
<td>3.61</td>
</tr>
<tr>
<td>Liberals</td>
<td>0.44</td>
<td>0.35</td>
<td>0.18</td>
<td>0.26</td>
<td>0.56</td>
<td>3.40</td>
</tr>
<tr>
<td>28-item mean</td>
<td>0.47</td>
<td>0.31</td>
<td>0.32</td>
<td>0.16</td>
<td>0.53</td>
<td></td>
</tr>
</tbody>
</table>

Source: Access to the data provided by Eaves et al., principal investigators, Virginia 30K twin study (see note 7).

$^a$ The MZ–DZ correlation difference is statistically significant for all of the table items at the 0.01 level or above.

The impact of shared environment exceeds that of heredity for only four of the 28 items, and the mean estimate of heritability for the 28 W–P items is .32, compared to a mean estimate of shared environmental influence of .16.

The second-to-last column in Table 1 reports the estimates for the proportion of the variation in an attitude that is attributable to the unshared environment. As described above this is essentially a residual variance category, reflecting such factors as random choice as well as external influences such as the unique experience of each individual, including those from childhood, and later influences in life that have been termed “adult socialization” in the political science literature.

Across the 28 W–P items the estimate of the impact of unshared environment varies from about one-third (for School Prayer) up to about two-thirds (for Pacifism) of the overall variation. The average impact of the unshared environment for these items is .53, or roughly half of the overall variation. The summary picture for this set of political attitudes, then, is that shared influences (genetic and environmental) account for about half of the variation in these political reactions, with unique individual and environmental factors accounting for the remainder. Within the half that is accounted for by shared influences, genetic influences, in contradiction to behavioralist expectations, are roughly twice as influential as environmental influences.

While the individual items provide interesting variation, the purpose of the W–P inventory is to provide an overall index of conservatism. We compute a simple index by assigning a value of +1 to any “conservative” response (i.e., a “yes” to an item like Death Penalty or a “no” to an item like Women’s Liberation) and −1 to any “liberal” response (i.e., a “no” to an item like Death Penalty or a “yes” to an item like Women’s Liberation). Items where the respondent chose a non-committal (?) response are coded as zero. When these individual scores are summed across the 28 items they yield an index that varies from a potential low of −28 (indicating a set of uniformly “liberal” responses) to a high of +28 (indicating a set of uniformly “conservative” responses). The actual index scores for the twins in the study range from −26 to +26, with the median response falling between +2 and +3. Given the far more continuous nature of this overall index, we can now utilize the more traditional Pearson’s correlation coefficient. The results for the overall index
are presented in Table 2 and clearly support a powerful role for heredity in influencing conservatism, at least as measured by the W–P inventory. The estimate for heritability is .43, higher than for any of the individual items. The estimate for shared environment is .22, falling within the upper range of the individual items, while the estimate for unshared environment is only .35, falling very near the bottom of the range for individual items. The overall picture is again a very strong role for heredity and a less powerful, but clear role for shared environment. What is different for the overall index is that the role of shared influences (genetic and environmental) account for almost two-thirds of the variation in the index (compared to about one-half for the individual items), with unique individual and environmental factors accounting for only about one-third of the variation. This decline in the role of unique individual and environmental factors seems sensible, as we are moving from individual and highly specific items that could involve a host of unique experiential, associational, and informational perturbations to an index where those idiosyncratic features of individual items have the opportunity to cancel each other out.

The W–P items can also be used to construct a rough index of political opinionation by taking advantage of the frequency of ? responses. The number of times that a respondent chose a yes or no response over a neutral ? response was summed to produce an index that varies from zero to 28, with a 28 indicating that the respondent was willing to express a directional opinion on any of the 28 items. The median for this index is 21 yes or no response choices of 28 possible. The results for the overall index clearly support a powerful role for heredity in influencing political opinionation, at least as it is captured by the admitted rough gauge of the frequency of nonneutral responses to the W–P inventory items. The estimate for heritability is .36 and the estimate for shared environment is only .02. The estimate for unshared environment is high, at .61, falling near the top of the range for individual items. To the extent that there is a family effect on political opinionation, it would appear to be entirely a genetic one, with the remaining roughly two-thirds of the variation being due to nonshared factors.

Two items from the survey that are not a part of the W–P inventory are included in Table 2. Party affiliation is the most clearly political of the items in the broader questionnaire, and it is useful here on its own, as well as in contrast to the attitudinal items. Party identification is distinct among U.S. political attitudes both in our conception of it as an identification, and hence as something at least potentially distinct from simple item evaluation, and in its established tendency to correlate well between parent and child (see Jennings and Niemi 1968). This distinctiveness is apparent in Table 2. As we expected, the pattern for party identification is nearly the exact reverse of that for the average attitude item. Heritability for party affiliation is relatively low (r = .14), while shared environment is much stronger (r = .41). Note also that not one of the 28 W–P items has an average heritability that is as low as that for party affiliation, and likewise, not one of the 28 items has an average coefficient for the impact of shared environment that is as high as that for party affiliation. Clearly, party identification is, at least for the United States, a different sort of beast than reactions to issue items.

In this regard it is particularly interesting that the two major parties also appear in the W–P battery, but here they are objects of affect rather than labels of possible identification, and the “pro” or “con” reactions to the parties that these items pick up do not exhibit the same patterns of genetic and environmental influence that we see for party affiliation. In fact, if we average the polychoric correlation for the “Democrats” item with the correlations for the “Republicans” item and compute the resulting estimates we get a heritability
estimate of .31 and a shared environment estimate of .17, almost exactly the same as the mean results for all 28 attitude items. It would appear that affect toward the major parties is largely a matter of genetic predisposition but that, just as the political socialization literature has concluded all along, party identification itself is primarily the result of parental socialization. This pattern is intriguing in and of itself but it also should give pause to those who would dismiss the findings on attitude items as the product of some methodological quirk of twin studies. If estimates of heritability are somehow artificially inflated, why does this alleged contamination not occur for party identification?

Table 2 also reports the results for a summary indicator of educational attainment from the survey. We include it here partly because it reflects an actual behavior, if only a self-reported one, and partly because it carries the role of genetics more directly into the world of actual and meaningful social variation. Educational attainment is also useful as an example of a behavior that is traditionally thought to be heavily influenced by shared environment, particularly by parental example, expectations, and resources. This traditional view is supported by the shared environment estimate of .46, a figure higher than any of the estimates for the 28 attitude items and even somewhat higher than the estimate for party ID. What may surprise readers is that as important as shared environment is to educational attainment, heredity, at .40, is almost as important. Taken together, family effects are almost the entire story for variation in education attainment. The estimate for the impact of the unshared environment is only .14, a value markedly lower than any other in the table.

ASSORTATIVE MATING

Assortative mating is a particular concern here. As detailed above, the assumption that DZ twins, like any other pair of biological siblings, share on average 50% of the variable genetic code is crucial to the estimation of heritability. This contrasts with MZ twins, where the shared proportion is 100%, and the DZ level forms the baseline for separating genetics from the shared environment. What may not be immediately apparent is that the assumption that purely genetic traits in DZ twins will on average correlate at .50 is itself built on the assumption that their biological parents will on average correlate at .00 for the same traits. In other words, the estimate of .50 is itself built on the assumption that their biological parents will on average correlate at .00 for the same traits. In other words, DZ twins of these parents will be as genetically alike on this one trait as MZ twins are on this trait. Across a study population, the higher the proportion of spouses that share identical genetics for a trait, the closer the DZ correlation will be to the MZ correlation. Since heritability of a trait is estimated as \( 2 \times (MZ - DZ) \), the increased similarity of DZ and MZ pairs will lead to an underestimation of heritability for this genetic trait.

This is important for our assessment of the heritability of political attitudes. If there is a tendency for people to choose mates with similar positions on political issues, then the estimates of heritability in Tables 1 and 2 are biased. Fortunately for us, the direction of the bias is uniform and conservative. Any measurable tendency toward assortative mating on political orientation will push up the DZ twin correlation while leaving the MZ correlation unaffected, and this reduction in the MZ–DZ gap will have the related effect of lowering estimates of heritability. Note also that any increase in similarity of DZ twins will inflate the estimate of the importance of shared environment, as the estimation formula of \( 2 \times DZ - MZ \) makes clear.

The immediate empirical question is how much of a role assortative mating plays in political issue positions. A quick answer can be found by looking at the interspouse polychoric correlations for the individuals included in the Virginia 30K study. The average interspouse polychoric correlation for the 28 items is .41 and the individual correlations range from a low of .26 for Censorship to a high of .64 for School Prayer. While some of this interspouse similarity could plausibly be attributed to persuasion effects taking place after mate choice rather than to assortative mating, the levels of similarity are probably too high to dismiss assortative mating entirely. This is confirmed by a preliminary look at the impact of controlling for assortative mating on these 28 attitude items. The Virginia 30K study includes data for parents of twins in the study, including parents’ individual responses to the same W–P items that the twins responded to. The usable sample size does drop substantially when we restrict our analysis to only twin pairs with completed W–P results for both parents (there are a total of 304 pairs of male/male or female/female twins with complete twin and parent W–P data, compared to approximately 4,400 pairs in the twin only analysis in Table 1). This effectively limits us to an assortative mating analysis that focuses on the overall index score, rather than looking at each item in the inventory individually.

The approach we used is to compute the partial correlation for twin similarity in the overall index for the 28 W–P items, controlling for (partialing out) the influence of the degree of parental similarity on the overall index. The implication for relative twin agreement is simple; if parental agreement results from assortative mating, then the resulting increase in genetic similarity will increase DZ twin correlations (the more alike genetically the parents are on a trait, the more alike siblings will be on a trait). Controlling for parental similarity will therefore reduce the size of the DZ twin correlations. However, parental agreement resulting from assortative mating and the resulting increase in genetic
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similarity will not increase MZ twin correlations (MZ twins are already genetically identical, regardless of parental similarity or dissimilarity). Therefore, controlling for parental similarity should have no effect on the size of the MZ twin correlations. In contrast, if parental agreement results from persuasion or from a shared environment for the couple, then the impact of parental agreement has no genetic implications and operates on their offspring solely through its influence on the offspring’s shared environment. This should produce relatively higher correlations of equal magnitude for both MZ and DZ twins and, therefore, lead to roughly comparable reductions in both the MZ and the DZ correlations when we partial out the effect of parental agreement.

The results for a partial correlation analysis controlling for parental agreement are reported in Table 2, on the row just below the results for the overall index. For MZ twins the issue of whether their parents agree or disagree on a particular item makes little difference (.65 without control versus .64 after partialing out the effect of parental agreement). In contrast, the correlation between DZ twins decreases modestly when the impact of parental agreement is removed (.43 without control versus .37 after partialing out the effect of parental agreement). Further, the tendency of assortative mating to deflate estimates of heritability while inflating estimates of the impact of shared environment is clear. Without controls, the estimate of heritability for the overall index is .43 and the average estimate of the impact of shared environment is .22. When the impact of parental agreement is partialled out, the average estimate of heritability rises to .53, and the average estimate of the impact of shared environment drops to .11. Note that the traditional socialization account of attitude formation is not at odds with this last finding. If the issue positions of parents are in conflict, then we would hardly expect this shared conflicted setting to yield sibling agreement.8

**COMPARATIVE POLITICAL GENETICS: EVIDENCE FROM AUSTRALIA**

Even with a data set as large as the Virginia 30K, questions may arise over the extent to which conclusions are bound by time and geography. As a result, it is helpful to note results from a quite different context and a slightly different time period. Table 3 presents a comparison of the key summary results in Table 1 from the Virginia 30K study to comparable results in the Australian data described before (Truett et al. 1994; see also Lake et al. 2000). While the Australian study

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8 The same sort of control for parental agreement that was applied to the W–P inventory was applied to the party affiliation analysis. Because this is only a single item, the results are much less reliable than those averaged across the 28 items. However, despite the fact that assortative mating clearly takes place with regard to party ID (only 24 of the 543 parent pairs had opposite party affiliations), the general pattern of party ID being due more to shared environment than to heredity holds up. Using a very broad definition of disagreement (i.e., anything short of exact agreement on a five-point scale), the shared environment estimate weakens modestly but remains high, at almost twice the heritability estimate in the subset of twin pairs with parents in some degree of disagreement on party affiliation.

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### Table 3. Comparison of Australian and U.S. Estimates of Genetic and Environmental Influences on Political Attitudes

<table>
<thead>
<tr>
<th>Attitude Item</th>
<th>Virginia 30K Data</th>
<th>Australian Data</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Heritability, (2 \times (MZ – DZ))</td>
<td>Shared Environment, (2 \times (DZ – MZ))</td>
</tr>
<tr>
<td>Astronomy</td>
<td>0.49 0.33 0.41 0.01 0.14 0.06</td>
<td>0.30 0.32 0.31 0.07 0.18 0.13</td>
</tr>
<tr>
<td>Pacifism</td>
<td>0.46 0.34 0.40 –0.09 –0.03 –0.06</td>
<td>0.26 0.48 0.37 0.09 –0.08 0.01</td>
</tr>
<tr>
<td>Censorship</td>
<td>0.55 0.20 0.37 –0.12 0.17 0.02</td>
<td>0.30 0.48 0.39 0.11 –0.03 0.04</td>
</tr>
<tr>
<td>Socialism</td>
<td>0.33 0.36 0.35 0.05 0.08 0.07</td>
<td>0.24 0.24 0.14 0.38 0.24 0.31</td>
</tr>
<tr>
<td>Military Drill</td>
<td>0.42 0.24 0.33 –0.02 0.12 0.05</td>
<td>0.32 0.42 0.37 0.20 0.08 0.14</td>
</tr>
<tr>
<td>Immigration</td>
<td>0.29 0.35 0.32 0.16 0.10 0.13</td>
<td>0.50 0.20 0.07 0.50 0.23 0.37</td>
</tr>
<tr>
<td>Death Penalty</td>
<td>0.27 0.35 0.31 0.27 0.19 0.23</td>
<td>0.42 0.54 0.48 0.10 –0.03 0.04</td>
</tr>
<tr>
<td>Women’s Liberation</td>
<td>0.23 0.35 0.29 0.08 0.17 0.13</td>
<td>0.22 0.32 0.27 0.15 0.16 0.16</td>
</tr>
<tr>
<td>Segregation</td>
<td>0.34 0.23 0.29 0.04 0.14 0.09</td>
<td>0.44 0.38 0.41 0.09 0.08 0.09</td>
</tr>
<tr>
<td>Modern Art</td>
<td>0.31 0.21 0.26 0.11 0.22 0.16</td>
<td>0.40 0.22 0.31 –0.02 0.26 0.12</td>
</tr>
<tr>
<td>Abortion</td>
<td>0.26 0.24 0.25 0.29 0.44 0.36</td>
<td>0.10 0.42 0.26 0.50 0.25 0.38</td>
</tr>
<tr>
<td>Divorce</td>
<td>0.20 0.28 0.24 0.22 0.21 0.21</td>
<td>0.16 0.44 0.30 0.31 0.10 0.21</td>
</tr>
<tr>
<td>Mean</td>
<td>0.35 0.29 0.32 0.08 0.16 0.12</td>
<td>0.24 0.37 0.31 0.21 0.12 0.16</td>
</tr>
</tbody>
</table>

Source: Access to the original U.S. data provided by Eaves et al., principal investigators, Virginia 30K twin study. Australian data computed from Martin et al. 1986, Table 1, p. 4365.

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used a larger set of W–P items (50 in all, compared to 28 in the U.S. study), the items were a mix of political and social items, and only six items appeared in exactly the same form in both studies. An additional six items were similar enough, in our judgment, to merit comparison, and they are included in Table 2 with the Australian wording italicized.

The broad picture from Table 3, and its comparison to Tables 1 and 2, is one of remarkable similarity. The mean heritability for the 12 item subset of the Virginia 30K data is .32 for the full 28 items in Table 1 and .31 for the 12-item subset of the Australian data. The mean estimate for the effect of shared environment for the 12 item subset of the Virginia 30K data is .12 compared to .16 for the full 28 items in Table 1 and .16 for the 12-item subset of the Australian data. Thus the general pattern of a relatively greater role for heredity compared to shared environment detailed above in the discussion of the U.S. data in Tables 1 and 2 also applies to the Australian data in Table 3. While most of the individual items also have broadly comparable results in the two countries, a few, specifically “socialism” and “immigration” (“nonwhite immigration” in the Australian study), are noticeably different. In both cases the U.S. pattern of substantially higher relative heritability is reversed in the Australian data, where we see evidence of relatively higher shared environmental effects. Whether these are meaningful reflections of differences in how these items relate to deeper political orientations is not clear, but they are in any case the exceptions rather than the rule.

THE GENETICS OF POLITICAL IDEOLOGY

The possibility that attitudes and behaviors are influenced by genetic variables is an emotionally charged topic so it is important that readers understand the claims being made. Partitioning the origins of human traits, whether they be physiological or behavioral, into the discrete, quantifiable components of genetic inheritance, shared environment, and unshared environment should not be taken to imply that these components work separately. Rather these numbers only provide a rough indication of the influence of three categories of independent variables that are intimately intertwined. (Moreover, they are estimates of the ability of independent variables to account for variance in the dependent variables not for the variables themselves.) As mentioned earlier, gene–culture interaction is the key to understanding the source of political attitudes and behaviors, just as it is the key to understanding most physical and behavioral aspects of the human condition. Genes do not work in isolation and instead generally influence the extent to which organisms are responsive to particular environmental conditions (see Boyd and Richerson 1985 and Masters 1993).

And this conditioning influence of genetics on complex social behaviors is not the product of a single gene but rather numerous genes that, to make matters more complicated, appear to combine in configural as opposed to additive ways. The same set of multiple genes may influence behavior in different ways depending on the order in which they express themselves and the manner in which they interact with other genes. Recent discoveries also suggest that biological markers of phenotypic manifestations include the manner in which DNA is packed in the nucleus, particularly the physical location of genes relative to other genes and to the histones that help to give DNA its structure. An accurate understanding of gene expression appears to require knowledge not just of the sequence of nucleotides (e.g., ATCAGG) that constitutes the gene itself but also of the context in which each gene resides, thus forming an interesting parallel to the way we must try to understand the organisms (e.g., human beings) genes help to construct (for a good summary, see Kosack and Grouidine 2004; also see Lykken 1999).

Individual genes for behaviors do not exist and no one denies that humans have the capacity to act against genetic predispositions. But predictably dissimilar correlations of social and political attitudes among people with greater and lesser shared genotypes suggest that behaviors are often shaped by forces of which the actors themselves are not consciously aware, a point that is made with some force by Bargh and Chartrand (1999), Marcus (2002), Marcus, Neuman, and MacKuen (2000), McDermott (2004), and Wegner (2002). It is not biological determinism to posit the existence of complex collections of genes that increase the probability that certain people will display heightened or deadened response patterns to given environmental cues. And it is not antibehavioralism to suggest that true explanations of the source of political attitudes and behaviors will be found when we combine our currently detailed understanding of environmental forces with a recognition that genetic variables subtly but importantly condition human responses to environmental stimuli.

IMPLICATIONS FOR POLITICS

It is important to note that none of the data or arguments presented in this paper indicates that extant empirical knowledge about political socialization is useless. In fact, it strongly reinforces many of the most salient findings in that research stream. We know from that research, for example, that if both parents share a political identification, there is a high degree of likelihood that their offspring will have that same political identification (Jennings and Niemi 1968; Tedin 1974). Our “twin study” results confirm this finding. One of the peculiar findings in the political socialization literature even makes more sense when a role for genetic inheritance in conceded. Scholars have occasionally puzzled over the fact that family arrangements and
styles of operation have little if any impact on the extent to which there is a match between parental and offspring political attitudes on a wide variety of items (see Jennings and Niemi 1968, 180–83). Fathers do not have more influence over sons, and mothers do not have more influence over daughters; fathers are not generally more influential; the distribution of power within the family is irrelevant to parent–child correlations (i.e., neither highly autocratic, highly permissive, nor middling arrangements affect the extent to which attitudes are correlated); the degree to which children and parents feel close to each other does not matter; the frequency with which the family discusses politics does not much affect correspondence between offspring and parent views (though, as we would have predicted since it is based on active socialization, party identification is more sensitive to family arrangements); and the extent to which politics is important to the parents is also irrelevant. Scholars grounded in traditional behavioralism have difficulty accounting for these “perplexing configurations” (Jennings and Niemi 1968, 183), but recognizing that the correlations between the views of parents and children derive more from genetics than familial socialization makes it much less surprising that the strength of these correlations is not reliant on family arrangements (for an example of political science work that does posit a role for genetics, see Peterson 1983).

Still, the substantive findings we present here offer a direct challenge to common assumptions and interpretations that political attitudes and behavioral tendencies are shaped primarily or even exclusively by environmental, especially familial, factors. Setting aside the important special case of party identification, we find that political attitudes are influenced much more heavily by genetics than by parental socialization. For the overall index of political conservatism, genetic accounts for approximately half of the variance in ideology, while shared environment including parental influence accounts for only 11%. And in the case of the variance in people’s tendencies to possess political opinions at all, regardless of their ideological direction, genetics explains one-third of the variance, and shared environment is completely inconsequential.

What are the implications of these findings for political science? Acknowledging a role for heritability in politics affects our understanding of, first, political issues, second, political learning, and, third, political cleavages. Inherited attitudes seem to be demonstrably different than acquired attitudes. Tesser (1993) provides evidence that attitudes higher in heritability are manifested more quickly, are more resistant to change, and increase the likelihood that people will be attracted to those who share those particular attitudes. It has long been known that certain political issues seem “hard” to people, and others seem “easy,” presumably because some issues trigger “gut responses” while others do not (Carmine and Stimson 1980, 79), but no explanation has yet been offered for why given issues do or do not elicit gut responses. Why do social, more than economic, issues tend to hit people in the gut, even though both constitute ongoing and equally complex societal concerns? In light of the new findings, one distinct possibility is that easy “gut” issues tend to be those that are more heritable.

To the extent that political ideologies are inherited and not learned, they become more difficult to manipulate. Conservative parents who try to make their children conservative by carefully controlling their children’s environments are probably overestimating the importance of those environments. Offspring of such parents are likely to end up being conservative but less because of the environment created by the parents than the genes passed along by the parents. A political match between parents and children should not be taken to be the result of a socialization process—that is, the active postnatal transmission of views—just as political mismatches between parent and child should not be taken as evidence against a role for genetics. Parent–child mismatches are distinctly possible given the uncertainties of meiosis (the random selection of just 50% of each parent’s DNA) and the possibility for occasional errors in the transcription and translation of genes (mutations). These mismatches are likely to be the primary cause of the fact that some children rebel against the views of their parents but most do not—just a pattern that environmental factors have never explained satisfactorily.

Finally, we go into somewhat greater detail to illustrate the manner in which results such as ours can be of use in understanding the divisions characterizing virtually all politics and, certainly, the United States in the early twenty-first century. Remember, genes influence people’s outlooks and personalities, and it is these broad features that then predispose individuals toward suites of specific attitudes. This interpretation likely explains the otherwise puzzling consistency in ideological divisions that is present across space and time. The package of attitudes held, for example, by conservatives in the modern United States is remarkably similar to that held by conservatives in other cultures and at earlier times in American history (on the durability of the liberal–conservative spectrum in the United States, see Poole and Rosenthal 1997). Environmental determinists have no convincing explanation for the pervasiveness of this division but genetics does.

If, as our results suggest, there is a genetic basis for the varying political views people hold, and if, as seems probable, genetic transmission frequently affects clusters of political attitudes, we are likely to observe broad but distinct political phenotypes. The number of these phenotypes may vary, but for purposes of illustration we discuss two probable orientations. One is characterized by a relatively strong suspicion of out-groups (e.g., immigrants), a yearning for in-group unity and strong leadership, especially if there is an out-group threat (“Do not question the President while we are at war with terrorists”), a desire for clear, unbending moral and behavioral codes (strict constructionists), a fondness for swift and severe punishment for violations of this code (the death penalty), a fondness for systematization (procedural due process), a willingness to tolerate inequality (opposition to redistributive policies),
and an inherently pessimistic view of human nature (life is “nasty, brutish, and short”).

The other phenotype is characterized by relatively tolerant attitudes toward out-groups, a desire to take a more context-dependent rather than rule-based approach to proper behavior (substantive due process), an inherently optimistic view of human nature (people should be given the benefit of the doubt), a distaste for preset punishments (mitigating circumstances), a preference for group togetherness but not necessarily unity (“We can all get along even though we are quite different”), suspicion of hierarchy, certainty, and strong leadership (flip-flopping is not a character flaw), an aversion to inequality (e.g., support for a graduated income tax), and greater general empathic tendencies (rehabilitate, don’t punish).

Common political usage would call the first phenotype conservative and the second liberal, but we seek phrases that are less connected to political ideologies and that indicate that these two phenotypes run to the very orientation of people to society, leadership, knowledge, group life, and the human condition. Thus, we label the first “absolutist” and the second “contextualist.” This fundamental dimension offers a credible precursor to basic cleavages manifested in a broad range of human social activity: politics (conservatives/liberals), religion (fundamentalists/secular humanists), law (procedural/substantive due process), education (phonics/whole language), art (traditional form-based realism/modern free-form impressionism), sports (football/frisbee), medicine (traditional AMA/wholistic), morality (enduring standards/situational ethics), and scientific inquiry (formal/empirical). In our view, all of these vexing perennial dichotomies are related cultural expressions of a deep-seated genetic divide in human behavioral predispositions and capabilities. We certainly are not asserting that everyone holds one of these two orientations. Even if the individual genes involved with absolutism or contextualism tend to move together, this does not mean they always do. Some individuals may carry, say, an absolutist’s aversion to out-groups but a contextualist’s rejection of a universalistic behavioral code. Moreover, genes not included in these central packages, perhaps those related to extraversion, ambition, and intelligence, often muddy the waters.

More importantly, let us not forget that a heritable component of 50% for political ideology and probably somewhat higher for the absolutist-contextualist dimension still leaves plenty of opportunity for the environment to alter attitudes and behaviors—and even orientation. An individual with a contextualist genotype who has been repeatedly victimized by out-group members, or who has simply spent a great deal of time listening to persuasive absolutists, may adopt attitudes that run against type. Thus, even if a political system started with two pure genotypes, it would soon display a fascinating array of expressed orientations and beliefs, intensity levels, and degrees of involvement even as the system would continue to revolve around the central division between absolutists and contextualists.

Such an account is speculative at this point but is fully consistent with the findings presented here, with previous research on the durability of political ideologies, and with recent events in the United States. Accounts of the 2004 election, for example, that do not invoke this fundamental difference in orientation have fallen flat. Issues did not determine vote choice for the many citizens who expressed disagreement with existing economic policies and/or the war in Iraq yet still voted for the incumbent president, George W. Bush. Indeed, if the focus remains on issues, the resultant description of the American public is grossly at odds with reality. Morris Fiorina’s (2005) creative analysis of survey responses indicates that Americans can be placed in the middle on many important issues, but if this is true, then what explains the vitriol and intensity of feeling displayed by so many ordinary Americans in 2004?

Issues do not explain Americans’ politics. Many Americans admit that they do not follow or understand the issues (Hibbing and Theiss-Morse 2002), and to the extent they do, they support whatever their preferred politician and party seems to support (Page and Jones 1979). In the 1990s, a Democratic president (Bill Clinton) transformed welfare to workfare; then in the 2000s, his Republican successor (George W. Bush) greatly expanded federal involvement in both education and the provision of prescription drugs for senior citizens. If the enactors of these policies were reversed, the groups of citizens displaying support for the policies also would have reversed. Similarly, if a Republican president had committed adultery with a young intern or if a Democratic president had dramatically worsened the deficit and taken the country to war in a far-off land on the basis of undeniably incorrect beliefs about the opponents’ nuclear and chemical weapons capabilities, the positions of most voters on the acceptability of these conditions would be completely reversed. Issue positions generally reflect divisions; they do not create them.

Instead, the most accurate account of voting behavior in 2004 moves beyond issues to the basic, partially genotypic orientations described above. This sort of broad orientation is not far removed from what most commentators are trying to capture by reference to a “moral” division in the electorate, but without tying it to specific moral issues such as gay rights. The chasm inspiring so much hostility between citizens of the United States in the early twenty-first century did not divide supporters and opponents of privatizing Social Security; it did not even divide supporters and opponents of gun control. Rather, as has typically been the case, it divided absolutists and contextualists.

And the prospects for eliminating this divide are not promising. Since mate choice appears to be heavily tilted toward those with similar social and political attitudes, no genetic melting pot exists for these traits. Thus, the evidence presented here on assortative mating should be quite sobering to those in search of unity and togetherness. If anything, the heritability of orientation in combination with assortative mating may exacerbate the current divide.
But admitting that genetics influences political attitudes could actually help to mute societal divisions. Currently, absolutists and contextualists simply do not connect, and the result is frustration. To contextualists, absolutists appear simplistic and selfish; to absolutists, contextualists appear naive and indecisive. Each side talks past, and is authentically miffed by, the other. Recognizing that our political antagonists probably have a different genetic predisposition to people, life, human nature, and politics may serve to ease frustrations and, eventually, to improve communications across the chasm. If absolutists spent more time trying to think like contextualists and contextualists trying to think like absolutists, understanding would be increased and debates could become more constructive. As frustrating as it may be to debate with someone who holds such different orientations, value exists in recognizing that intrapsychic is not the result of willful bullheadedness but, rather, genetically driven differences in orientation.

The exciting next step is to understand the reason such distinct orientations have evolved and lasted. Evolutionary psychologists tend to assume that all enduring traits are adaptive (for a dissenting view, see Gould 2000) since natural selection drives out variation and makes adaptive traits ever more common. In this organism-based interpretation, whichever orientation—absolutism or contextualism—is evolutionarily superior should soon come to numerically dominate the other. This is possible but unlikely. An alternative group-based interpretation sees variation itself as adaptive (see Alford and Hibbing 2004 and Sober and Wilson 1998). The benefits of genetic variation are most easily observed in the ability of differential immune systems to prevent a group of organisms from being completely wiped out by a single pathogen, but it is easy to imagine how sociopolitical variation could also create more viable groups. In fact, computer simulations give support to the hypothesis that divergent individual-level social behaviors, such as cooperation and defection, are beneficial at the group level (Hammond 2000). As loathe as contextualists and absolutists are to admit it, the presence of the other orientation may make a society stronger.

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Genetic Variation in Political Participation

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The decision to vote has puzzled scholars for decades. Theoretical models predict little or no variation in participation in large population elections and empirical models have typically accounted for only a relatively small portion of individual-level variance in turnout behavior. However, these models have not considered the hypothesis that part of the variation in voting behavior can be attributed to genetic effects. Matching public voter turnout records in Los Angeles to a twin registry, we study the heritability of political behavior in monozygotic and dizygotic twins. The results show that a significant proportion of the variation in voting turnout can be accounted for by genes. We also replicate these results with data from the National Longitudinal Study of Adolescent Health and show that they extend to a broad class of acts of political participation. These are the first findings to suggest that humans exhibit genetic variation in their tendency to participate in political activities.

Why do people vote? The classic paradox of turnout has puzzled theorists for years (Aldrich 1993; Downs 1957; Feddersen and Sandroni 2006; Riker and Ordeshook 1968). When one person votes, everyone with the same preferences benefits from the increased likelihood that their preferred outcome will result. Yet those who do vote must bear the cost of time and effort required to learn about election alternatives and go to the polls. In large populations, the probability that a single vote will change the outcome of an election is miniscule (Gelman, King, and Boscardin 1998), meaning that even very small costs to the individual typically outweigh the expected benefits he or she would receive from voting. As a result, classic game theoretic models that assume individuals are self-interested and fully optimizing in their behavior show that the equilibrium amount of voter turnout approaches zero as the population becomes large (Palfrey and Rosenthal 1985). Yet in spite of this theoretical result, millions of people do vote, suggesting that something other than self-interest or optimizing behavior drives their decision (Bendor, Diermeier, and Ting 2003; Feddersen and Sandroni 2006; Fowler 2006b). In addition, the fact that millions of people abstain suggests that there may be inherent variation in the human tendency to participate in politics. Empirical models of turnout and political participation have tried to explain this variation using numerous covariates inspired by a vast literature (Plutzer 2002; Timpone 1998; Verba, Schlozman, and Brady 1995), including demographic factors like age (Strate et al. 1989), gender (Schlozman et al. 1995), race (Verba, Schlozman, and Brady 1993), marital status (Stoker and Jennings 1995), education (Leighley and Nagler 1992a), income (Leighley and Nagler 1992b), occupational prestige (Nie, Powell, and Prewitt 1969a; Nie, Powell, and Prewitt 1969b), and home ownership (Highton and Wollinger 2001); attitudinal and behavioral factors like interest in the campaign (Verba, Schlozman, and Brady 1995), access to political information (DiMaggio, Hargittai, and Neuman 2001), general political knowledge (Galston 2001), strength of partisanship (Huckfeldt and Sprague 1992), feelings of civic duty (Blais and Young 1999), internal and external efficacy (Finkel 1985), political trust (Hetherington 1999), church attendance (Cassel 1999), personal skill acquisition (Brady, Verba, and Schlozman 1995), humanitarianism (Jankowski 2007), altruism (Fowler 2006a), and patience (Fowler and Kam 2006); social factors like interpersonal communication (McLeod, Scheufele, and Moy 1999), social identification (Fowler and Kam 2007), group consciousness (Miller, Gurin, and Gurin 1981), socialization (Cho 1999), the status of neighbors (Huckfeldt 1979), political disagreement (Mutz 2002), and social capital (Lake and Huckfeldt 1998); and institutional factors (Jackman and Miller 1995) like closeness of the election (Shachar and Nalebuff 1999), contact from political organizations.
In spite of Merelman’s exhortation, genetic studies of participation were not forthcoming. Scholars continued to focus on personality factors underlying participation like efficacy (Finkel 1985) and self-esteem (Sears 1987) without mentioning the fact that these factors may themselves be heritable. A few scholars have consistently argued on general principle that genes must play a role in political behaviors like participation (Carmen 2004; Masters 1990; Somit and Peterson 1998) but they have not empirically tested their genetic hypotheses. As a result, the current state of scientific knowledge on the heritability of political involvement is limited.

In this article we conduct three tests of the hypothesis that part of the variation in political participation can be attributed to genetic factors. The results of all three of our tests suggest that individual genetic differences make up a large and significant portion of the variation in political participation, even taking socialization and other environmental factors into account. Our results show participation is heritable and suggest that political science as a discipline should be thinking more about biological sources of variation in political behavior. In particular, we argue that these results open the door to an untapped realm of causal theories and empirical tests that will help us to improve our understanding of one of the most basic acts of citizenship and democratic government.

TWIN STUDIES

In order to estimate the heritability of voting behavior, we study the turnout patterns of (identical) monozygotic (MZ) twins who were conceived from a single fertilized egg and (non-identical) dizygotic (DZ) twins who were conceived from two separate eggs. MZ twins share 100% of their genes, while DZ twins share only 50% on average. Thus, if voting behavior is heritable, MZ twins should exhibit more concordance (both twins vote or both twins abstain) than DZ twins. Moreover, if we assume that MZ twins and DZ twins share comparable environments (more on this assumption below), then we can use these concordances to estimate explicitly the proportion of the overall variance attributed to genetic, shared environmental, and unshared environmental factors. Very few differences have been found between twins and non-twins (Kendler et al. 1995), therefore we expect the results for twins to be generalizable to a non-twin population.

The twin study design has been shown to be an extremely powerful tool for identifying the relative degree to which genetic and environmental factors influence an observed outcome (Evans, Gillespie, and Martin 2002; Neale and Cardon 1992). The basic twin model assumes that the variance in observed behavior...
known relationships between three observed statistics the degree to which they contribute to variance is a observed covariates are needed in the model because covariance is inferred through their effects on the covariances environment are not measured directly but their influence the so-called ACE model. The role of genotype and co-twins (C), and unshared environmental (E). This is behavioral factors which are shared or common to environment influence on variation in the phenotype that create an unshared rather than a common environment. 

\[
\begin{align*}
\sigma_P^2 &= \sigma_A^2 + \sigma_C^2 + \sigma_E^2 \\
COV_{MZ} &= \sigma_A^2 + \sigma_C^2 \\
COV_{DZ} &= \frac{1}{2}\sigma_A^2 + \sigma_C^2,
\end{align*}
\]

where \(\sigma_P^2\) is the observed phenotypic variance (the same for MZ and DZ twins), \(COV_{MZ}\) and \(COV_{DZ}\) are the observed covariances between MZ and DZ co-twins, and \(\sigma_A^2, \sigma_C^2, \sigma_E^2\) are the variance components for genes, common environment, and unshared environment, respectively. These relationships yield three equations and three unknowns, so it is possible to infer the unobserved portions of variance attributable to each factor.

Since the variance components are not directly observable, the ACE model’s assumption of additivity cannot be tested and more complicated relationships are possible. For example, it is possible that genes interact with the environment (GxE) or with other genes (GxG) to yield variation in behavior, or at a higher level phenotypes interact with the environment (PxE) (Turkheimer and Waldron 2000). We limit our analysis to the ACE model but point out that if a strong effect for genes is found in the additive model, then genes are also likely to play a role in more complex specifications.

Finally, it is important to clarify the difference between the common environment (C) and the unshared environment (E) in the twin model. Common environment includes the family environment in which both twins were raised, as well as any other factor to which both twins were equally exposed. In contrast, the unshared environment includes idiosyncratic influences that are experienced individually. It is possible to have unshared environmental exposure as a child (twins may have different friends with different political beliefs) and to have shared environments as an adult (twins may see the same election results). Thus, the distinction between common and unshared environment does not correspond directly to family—nonfamily or adult-child differences in factors that influence a given behavior. Moreover, there may be a similarity in the objective environment but twins may have idiosyncratic experiences that influence their effective environment that create an unshared rather than a common environmental influence on variation in the phenotype (Turkheimer and Waldron 2000).

**THE COMPARABLE ENVIRONMENTS ASSUMPTION**

Some scholars have objected to the assumption that MZ and DZ environments are comparable, arguing that the identical nature of MZ twins cause them to be more strongly affiliated and more influenced by one another than their non-identical DZ counterparts. If so, then greater concordance in MZ twins might merely reflect the fact that their shared environments cause them to become more similar than DZ twins. However, studies of twins raised together have been validated by studies of twins reared apart (Bouchard 1998), suggesting that the shared environment does not exert enhanced influence on MZ twins. Moreover, personality and cognitive differences between MZ and DZ twins persist even among twins whose zygosity has been miscategorized by their parents (Bouchard and McGue 2003), indicating that being mistakenly treated as an identical twin by one’s parents is not sufficient to generate the difference in concordance. And, although MZ twins are sometimes in more frequent contact with each other than DZ twins, it appears that twin similarity (e.g., in attitudes and personality) may cause greater contact rather than vice versa (Posner, Baker, and Martin 1996). Finally, contrary to the expectation that the influence of the unshared environment would tend to decrease concordance over time once twins reach adulthood, MZ twins living apart tend to become more similar with age (Bouchard and McGue 2003).

**TURNOUT IN THE SOUTHERN CALIFORNIA TWIN REGISTRY**

To assess the heritability of turnout behavior, we obtained electronic voter registration records for 3.8 million voters from Los Angeles County with complete vote histories for eight elections (three primary, two statewide, and three general) from 2000 to 2005 and matched them to the Southern California Twin Registry (Baker et al. 2006), a list of MZ and DZ twins who live in the Los Angeles area. A principal advantage of this approach is the use of field evidence based on third-party observations of actual voter behavior rather than self-reports. This type of data is rarely used in twin studies and is an especially important source for evaluating political participation since a significant number of individuals who did not vote typically report that they did (Karp and Brockington 2005).

About 30% of the adult population in Los Angeles County is not registered to vote, so we cannot include them in our sample. We cannot merely assume that all unregistered twins chose not to vote—for example, it is possible that they died or moved out of the county and registered elsewhere. However, focusing on registered individuals allows us to exclude those who might generate false concordance because they are ineligible to vote due to foreign citizenship status—this is a particular concern in Los Angeles County where 22% of the total population are foreign citizens (2000 U.S. Census). It also allows us to avoid false concordance generated by individuals with cognitive or literacy deficits who are
not capable of voting since these individuals probably do not register.

Twin registry and voter registration records were matched by surname, first name, birthdate, place of birth, and zip code. Full matches were automatically included in our data. Partial matches on three or more of these attributes were manually checked and included in the data if the failure to match fully was determined to be the result of a typographical error. We restricted our search to same-sex twin pairs because MZ twins are always same sex and DZ twins are not. Including opposite-sex twin pairs would complicate the analysis because we would have to assess whether differences in concordance between MZ and DZ twins are the result of closer social affiliation between same-sex pairs.

Out of 878 same-sex twins (535 MZ, 343 DZ) on the registry who live in Los Angeles County, this procedure yielded vote histories for 396 twins—168 MZ twins and 102 DZ twins in matched pairs, and 79 MZ and 47 DZ “singletons” where we found one twin in the pair but not the other.1 A Mann Whitney U test suggests that the difference in the success rate for matching between MZ twins (48.6%) and DZ twins (43.4%) was not significant (p = 0.14).

Although we did not have access to information about the twins’ socioeconomic status for the entire sample, we were able to use their addresses to look up estimated home values and square feet on the home appraisal web site zillow.com.2 We also examined data from previous studies in which subsets of the matched twins had participated through the Southern California Twin Registry.3 Although not available for the entire sample, these prior data are used to evaluate possible differences between MZ and DZ pairs that might explain their voting behavior.

To test the comparable environments assumption for our sample, we performed a series of tests on the mean difference between MZ and DZ twins for a number of variables (see Tables 1 and 2). High p-values in Mann Whitney U tests suggest that differences between types of twins are not significant for rates of turnout (p = 0.79), Democratic Party membership (p = 0.84), Republican Party membership (p = 0.83), third party membership (p = 0.88), age (p = 0.25), house value (p = 0.49), house square footage (p = 0.86), and lot square footage (p = 0.15). Furthermore, t-tests of data obtained from previous studies of subsets of these twins revealed no differences in their education level (p = 0.72) or personality, including extraversion (p = 0.38) and neuroticism (p = 0.92). Thus, the similarity of the MZ and DZ twin samples suggests that differences in concordance cannot be explained by mean differences in political participation, political affiliation, personality, education, or other socioeconomic factors. We also note that in our sample MZ twins are not more likely than DZ twins to live at the same address (p = 0.69) or in the same postal code (p = 0.84). Thus, greater concordance in MZ twins is probably not due to higher frequency of contact.

It is important to note that we do not need to compare similarities between co-twins to test the comparable environments assumption. For example, if we show that MZ twins are more similar than DZ twins for income, then it means income attainment might be heritable but it has no bearing on whether or not MZ and DZ twins come from essentially similar environments. Conversely, we might find that MZ and DZ co-twins are equally similar on income, but this would not imply their environments were the same. The fact that MZ and DZ twins are drawn from households that have similarly distributed income suggests that there is nothing unique about MZ twins that could cause a spurious difference in the similarity of turnout via a difference in income. For example, if MZ twins were much richer than DZ twins, they might be more similar since wealthier households vote more. Since they are not richer, we can reject this possible explanation for why we find a difference in the similarity of turnout between MZ and DZ twins.

One might worry about the house values in Table 1, because they suggest that the subject pool is drawn from the more affluent part of the population (the average single family house at this time in Los Angeles county sold for about $600,000). However, this would only be important if it had a systematic effect on turnout. Table 2 shows that DZ twins and MZ twins do not exhibit systematically different rates of turnout in the different elections. Table 2 also shows that turnout rates for all twins were somewhat higher than those for the population, but this should not bias estimates of the magnitude of the difference in concordance between MZ and DZ twins. This is because the variances are not systematically different from the population (the twin variance is higher than the population variance in two elections and lower in three). If the twin variance were much lower than the population variance, this would compress the difference in the MZ and DZ twin concordances, causing us to underestimate heritability.4 Conversely, if the twin sample variance were much higher than the population variance we would overestimate the effect of heritability. The lack of a systematic difference in the sample and population variances suggests that mean differences will not generate bias in the estimates.

INITIAL RESULTS AND THE BAYESIAN ACE MODEL

Figure 1 shows two two-dimensional density plots of the number of elections in which each twin chose to vote (MZ twins on the left, DZ twins on the right).

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1 The statistical power of twin studies is maximized when DZ twins exceed the number of MZ twins by a factor of 3 or 4 to 1 so not only is our total number of twins small but the ratio is not optimal. However, this affects efficiency and not bias.

2 Thanks to John Zaller for this suggestion.

3 See Baker et al. (2006) for a description of studies conducted using the Southern California Twin data.

4 For example, in the extreme case where all twins are perfectly concordant and the turnout rate = 100%, the variances shrink to 0, the concordances for both MZ and DZ twins grow to 1, and the difference between the concordances would also shrink to 0, suggesting 0 heritability.
TABLE 1. Summary Statistics, by Zygosity, Los Angeles Sample

<table>
<thead>
<tr>
<th></th>
<th>MZ Twins</th>
<th>DZ Twins</th>
<th>Difference of Means Test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Standard Error</td>
<td>Mean</td>
</tr>
<tr>
<td>Voter file data</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Turnout Rate, All Elections</td>
<td>0.57</td>
<td>0.03</td>
<td>0.58</td>
</tr>
<tr>
<td>Democrat</td>
<td>0.51</td>
<td>0.05</td>
<td>0.52</td>
</tr>
<tr>
<td>Republican</td>
<td>0.24</td>
<td>0.04</td>
<td>0.25</td>
</tr>
<tr>
<td>Third Party</td>
<td>0.05</td>
<td>0.02</td>
<td>0.05</td>
</tr>
<tr>
<td>Age</td>
<td>36.8</td>
<td>2.5</td>
<td>33.6</td>
</tr>
<tr>
<td>Same Address</td>
<td>0.47</td>
<td>0.07</td>
<td>0.52</td>
</tr>
<tr>
<td>Same Postal Code</td>
<td>0.54</td>
<td>0.07</td>
<td>0.64</td>
</tr>
<tr>
<td>Zillow.com data</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Was House in Zillow?</td>
<td>0.71</td>
<td>0.04</td>
<td>0.71</td>
</tr>
<tr>
<td>House Value</td>
<td>821,729</td>
<td>40,577</td>
<td>784,421</td>
</tr>
<tr>
<td>House Square Feet</td>
<td>2148</td>
<td>111</td>
<td>2106</td>
</tr>
<tr>
<td>Lot Square Feet</td>
<td>8062</td>
<td>392</td>
<td>9117</td>
</tr>
<tr>
<td>SCTP data</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education Highest Grade</td>
<td>15.48</td>
<td>0.36</td>
<td>15.25</td>
</tr>
<tr>
<td>Extraversion</td>
<td>0.66</td>
<td>0.04</td>
<td>0.71</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>0.43</td>
<td>0.03</td>
<td>0.43</td>
</tr>
</tbody>
</table>

Note: These data show that we could find no significant differences in the MZ and DZ twin samples, suggesting that they are drawn from comparable environments. Data are derived from three sources: 1) the Los Angeles County voter registration and vote history files for matched twins, 2) housing characteristics for 71% of the matched twins found on zillow.com on October 25, 2006; and 3) education and personality information for 15% of the matched twins (this subsample is limited to those who participated in previous studies in which education and personality questions were asked). We utilized Mann–Whitney U tests to analyze differences in means in the voter registration and zillow data and t tests for the SCTP data.

TABLE 2. Comparison of Mean Turnout and Variance in Turnout in Twin Sample and General Population in Los Angeles County, by Election

<table>
<thead>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>All twins</td>
<td>0.54</td>
<td>0.76</td>
<td>0.36</td>
<td>0.57</td>
<td>0.68</td>
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<td>(N = 396)</td>
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<td>(0.23)</td>
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<td>(0.22)</td>
<td>(0.25)</td>
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<td>MZ twins</td>
<td>0.55</td>
<td>0.75</td>
<td>0.36</td>
<td>0.58</td>
<td>0.71</td>
<td>0.42</td>
<td>0.86</td>
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<td>(N = 247)</td>
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<td>(0.23)</td>
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<td>0.53</td>
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<td>0.37</td>
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<td>(N = 149)</td>
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<td>(0.23)</td>
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<td>Population</td>
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<td>0.68</td>
<td>0.26</td>
<td>0.45</td>
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<tr>
<td>(N = 22)</td>
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<td>(0.25)</td>
<td>(0.25)</td>
<td>(0.24)</td>
<td>(0.17)</td>
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</tr>
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Note: Variances are shown in parentheses.

color of each square indicates the number of observations at each point, so for example, there is a strong mode for MZ twins where each twin voted exactly twice (the point 2,2 contains about 7% of the MZ sample). With this representation we lose resolution because it is possible for both twins to vote in the same number of elections without voting at the same time (e.g. twin 1 might vote in two primaries and twin 2 might vote in two general elections). Nonetheless, patterns start to emerge. There appear to be more observations on the main diagonal for MZ twins, and DZ twins appear to be more likely to have large differences in the frequency they vote. We can analyze the pattern of voting statistically by examining the number of times each twin pair differs (one votes and one abstains). A simple t test of the absolute difference in co-twin voting behavior suggests that MZ twins are significantly more similar than DZ twins (p = 0.045, mean number of times co-twins made different decisions for MZ = 1.45, DZ = 2.00).

In the behavior genetics literature a simple comparison of polychoric correlations is frequently used as a first test of the rate of twin concordance in behavior (for a detailed explanation of this method, see Alford, Funk, and Hibbing 2005). In our pooled observations, the correlation in turnout was significantly higher (p = 0.006) between the MZ twins (0.71) than the DZ twins (0.50). Another simple and direct way to see if zygosity influences co-twin similarity is DeFries-Fulker regression (DeFries and Fulker 1985).

5 Because the concordance in DZ twins is greater than half the concordance in MZ twins, the common environment may play a role in voting. As a result it is appropriate to model twin-only data with an ACE model instead of the alternative ADE model that assumes the common environment plays no role.
In this method, the dependent variable is each twin’s behavior and the independent variables are zygosity, the co-twin’s behavior, and an interaction of the two. If the interaction term is significant, it means that MZ twins are statistically more likely to exhibit the same characteristics than DZ twins. We use a general estimating equation (GEE) to correct for multiple observations on the same twin pair and find that the interaction coefficient is indeed significant (Wald statistic = 4.38, \( p = 0.036 \)).

However, these measures are only a crude guide since they treat every choice as the same and they make no provision for the unique information contained in each election. For example, suppose everyone voted in the first election but only half voted in the second — the first election would not be very informative about the individual tendency to vote since there was no variation, but the second would be very informative. To take advantage of the differing discriminatory power of each election, we employ a generalized latent variable model, otherwise known as a two-parameter item response model (Clinton, Jackman, and Rivers 2004).

We assume there is a single latent propensity to vote underlying all eight observed turnout decisions.\(^6\) We also assume that both genetic and environmental effects operate through a common pathway (Eaves et al. 2005). The model can be specified as a generalized linear mixed-effects model where subject \( j \) is a member of family \( i \) choosing to vote (\( T_{ijk} = 1 \)) or abstain (\( T_{ijk} = 0 \)) in election \( k \). We assume the probability that an individual will vote in election \( k \) (a binary choice) is

\[
\Pr(T_{ijk} = 1) = \Psi(\delta_{k} \tau_{ij} - \alpha_{k}),
\]

where the \( \Psi \) function that links the latent tendency to vote to a probability is a logit:

\[
\Psi(x) = \frac{1}{1 + \exp(-x)}.
\]

In this model \( \tau \) is a normally distributed continuous variable that corresponds to the individual’s latent propensity to vote, \( \delta \) is the discriminating power of each election, and \( \alpha \) is the threshold identifying the point at which the likelihood of voting is greater than abstaining in each election (also known as the “difficulty parameter” in item-response models). The parameter \( \delta \) is analogous to loadings in a simple factor model, which allow each election to have a different weight in the underlying tendency to vote (Eaves et al. 2005).

In order for this model to be identified we fix the total variance of the latent trait (\( \tau \)) to one.

Next, we assume that the latent tendency to vote is influenced by additive genetic factors, shared environment, and unshared environment. These three factors completely account for the three different kinds of variance that it is possible for us to diagnose in a model of identical twins. We model this assumption using three random effects variables for MZ twins:

\[
\tau^{MZ}_{q} = A_{i} + C_{i} + E_{ij},
\]

where \( A_{i} \) is the family genetic factor, \( C_{i} \) is the family shared environment factor, and \( E_{ij} \) is the individually-experienced unshared environment factor. For DZ twins the tendency to vote is modeled using four random effects variables:

\[
\tau^{DZ}_{q} = A_{1i} + A_{2ij} + C_{i} + E_{ij},
\]

where \( A_{1i} \) is the family genetic factor shared by both twins, \( A_{2ij} \) is the individually-inherited genetic factor that is unique to each twin, and \( C_{i} \) and \( E_{ij} \) are the same as for MZ twins.

It is important to reiterate that there are no observed covariates in any of the models. In particular, none of the measured environmental variables we examined in Table 1 are included. Everything on the right hand side involves latent variables whose effects are estimated solely from the observed participation decisions. Adding covariates to the right hand side would not affect the variance decomposition because they would merely reduce the magnitude of the most-closely

\(^6\) A Cronbach test (\( \alpha = 0.78 \)) reveals that these eight elections are reliable measures of a single scalar latent value for the propensity to vote.
related component. For example, suppose that neighborhood context influences political participation among twins who live apart as adults. If so, we might include a factor in the model like average neighborhood income. If we inserted this variable as an additive factor that directly influences the individual’s turnout propensity, it might reduce the magnitude of the unshared environmental variance since it would partially account for some of it. However, we would have to add the variance explained by mean neighborhood income to the unshared environmental variance to estimate its total influence. Thus, in essence, the latent factors indicate the total additive influence of all possible genetic and environmental variables that could be included the model.

Traditionally, the typical approach to estimate the components of variance has been structural equation modeling (SEM), however Bayesian methods are increasingly being viewed as a superior modeling approach (Burton et al. 1999). For our modeling task there are two main advantages to using a Bayesian model. First, discrete phenotypes (like the dichotomous decision to vote or abstain) present computational challenges for SEM software packages because the likelihoods contain high-dimensional integrals that cannot be evaluated in closed form and thus must be evaluated numerically (van den Berg, Beem, and Boomsma 2006). As a result, scholars have begun to use Markov Chain Monte Carlo (MCMC) algorithms. These algorithms evaluate the integrals using random draws rather than evaluating them analytically. In particular, simulation studies suggest that MCMC methods perform better than SEM for models like ours. For example, Kuhnert and Do (2003) show that a Bayesian binary response model identifies the correct model more often than a comparable SEM model in cases where the simulated heritability is low or medium (both performed equally well in cases of high heritability).

Another advantage of the Bayesian approach is that credible intervals for the variance component estimates do not rely on large-sample theory that may not be appropriate for twin studies with small sample sizes (Chen, Manatunga, and Williams 1998). In an extensive simulation study, Burton et al. (1999) showed that a Bayesian binary response model identifies the correct model more often than a comparable SEM model in cases where the simulated heritability is low or medium (both performed equally well in cases of high heritability).

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Replicating the methods used in this literature, we assume that our unobserved random effects are normally distributed: $A \sim N(0, \sigma^2_A)$, $A_1 \sim N(0, \sigma^2_A/2)$, $A_2 \sim N(0, \sigma^2_A/2)$, $C \sim N(0, \sigma^2_C)$, and $E \sim N(0, \sigma^2_E)$. Notice that the variance of $A_1$, the family genetic effect for DZ twins, is fixed to be half the variance of $A$, the family genetic effect for MZ twins, reflecting the fact that DZ twins on average share half as many genes as MZ twins. Moreover, DZ twins are also influenced by individually-specific genes $A_2$ that are drawn from the same distribution as the shared genes since on average half their genes are shared and half are not. These assumptions about the genetic variance help to distinguish shared genes from the unshared environment variable $C$ that is assumed to have the same variance for both MZ and DZ twin families, and the residual unshared environment variable $E$ from which a unique draw is made for each individual.

If we tried to estimate all three components of variance simultaneously the model would not be identified, so we fix the variance of the unshared environment $\sigma^2_E = 1$ and then use the estimates of $\sigma^2_A$ and $\sigma^2_C$ to derive the proportion of variance generated by each factor. This procedure generates estimates for the influence of heritability $h^2 = \sigma^2_A/(\sigma^2_A + \sigma^2_C + \sigma^2_E)$, common environment $c^2 = \sigma^2_C/(\sigma^2_A + \sigma^2_C + \sigma^2_E)$, and the unshared environment $e^2 = \sigma^2_E/(\sigma^2_A + \sigma^2_C + \sigma^2_E)$. Since the underlying components are not constrained, the estimated proportions can range anywhere between 0 (the component has no effect on variance) and 1 (the component is solely responsible for all observed variance).

In some cases, the estimate for $e^2$ will be close to 0, so we can test the hypothesis that the common environment matters by dropping it from the ACE model, creating an AE model (alternatively we could drop A to create a CE model). If the AE model fits better than the ACE model, then it suggests a weak or insignificant role for the common environment. Procedurally, the difference between the ACE and AE model is that the random effect for the common environment is not estimated and $\sigma^2_C = 0$. To compare the fit of ACE and AE models we used the deviance information criterion (DIC), a Bayesian method for model comparison analogous to the Akaike Information Criterion (AIC) in maximum likelihood estimation. Models with smaller DIC are considered to have the best out of sample predictive power (Gelman et al. 2004). The DIC penalizes models for deviance (Dbar), which captures model fit, and the effective number of parameters (pD), which captures model complexity.

In our MCMC procedure we use vague prior distributions to ensure they do not drive model results. For $\mu$ we use a mean-zero normal distribution with variance 1,000,000 and for the precision parameters associated with $\sigma^2_A$ and $\sigma^2_C$ we use a pareto distribution with shape parameter equal to 1 and location parameter equal to 0.01. In addition, we use convergence diagnostics to be sure we have reached the stationary posterior distribution.\footnote{Recent studies have successfully applied Bayesian methods to genetic models using binary data (Kuhnert and Do 2003; van den Berg, Beem, and Boomsma 2006), survival analysis (Do et al. 2000), nonlinear developmental change and GxE interaction (Eaves and Erkanli 2003), item response theory (Eaves et al. 2005), longitudinal models (Burton et al. 2005), and multivariate models for ordinal data (van den Berg et al. 2006).}

\footnotetext[9]{We experimented with priors using different distributions. We tried a gamma with shape parameters 0.001, 0.01, and 0.1 and scale parameters 1,000, 100, and 10, respectively. We also tried uniform priors (0,10), (0,20), and (0,100) on $\sigma_A$ and $\sigma_C$ but found they had essentially no effect on the final estimates.}

\footnotetext[10]{To ensure that the models converged to what we believe to be their target posterior distribution, we began sampling from the joint posterior distribution after convergence was established using the}
How do the assumptions we make in this model compare to assumptions political scientists typically make in their models? Consider a simple logit model of turnout. Like the ACE model presented here, a logit model (1) implicitly assumes that there are independent normal data generating processes that (2) influence a latent variable that is (3) transformed via a logistic function into a probability, which (4) itself is also a latent, unobserved variable. The ACE model is somewhat more complex, but not more so than state-of-the-art Bayesian item-response models (Clinton, Jackman, Rivers 2004) which also include assumptions about prior distributions and multilevel latent factors.

RESULTS

The results of the ACE model suggest that 53% of the variance in turnout behavior can be accounted for by additive genetic effects ($h^2$). The 95% credible interval (C.I.) for the estimate is (10%, 89%), indicating that we can reject the hypothesis that genes do not contribute to variation in turnout. The ACE model also suggests that the environment is important, with the shared environment ($c^2$) accounting for about 35% of the variance (C.I. 2%, 73%) and the unshared environment ($e^2$) accounting for 12% (C.I. 3%, 26%). Figure 2 shows the 95% credible area of the joint estimates. Notice that the contribution of the common environment is close to zero. This suggests that an AE model, where the common environment variable is assumed to be zero, may be more appropriate. Indeed, measures of model fit indicate that an AE model is superior to the ACE model (see Appendix A-1). Although one may be concerned that our analysis lacks power because our sample of 396 subjects is small, multiple observations per individual improve the precision of the estimates, and the credible intervals in the posterior indicate that there is an extremely low probability ($p < 0.0001$) that voting behavior is not heritable.

To ensure that our model is consistent with the data, we use it to generate replicated values of the dependent variable from the predictive distribution for each simulated parameter in the model and compare these replicated values with the observed dependent variable (Gelman et al. 2004). Variance in the common factor underlies the individual election turnout variables. The Mplus software provides maximum likelihood estimates in genetic models for observed categorical variables (Prescott 2004). Variance in the common factor was explained primarily by genetic factors ($A = 67\%$; S.E. = 38%), with non-significant effects of shared environment ($C = 27\%$; S.E. = 34%) and non-shared environment ($E = 7\%$; S.E. = 8%). All alternatives (Bayesian and non-Bayesian) suggested that turnout behavior is heritable, with mean $h^2$ consistently estimated to be greater than 50%. We also explored whether heritability estimates differed by types of elections (primary vs. general, close vs. not close), however the power for our sample was too low to detect any significant difference.11

Note: Ternary plot shows the posterior Bayesian distribution of estimated components of total variance in an ACE model of voter turnout among subjects in the Southern California Twin Registry (SCTR). Mean heritability ($h^2$) is estimated to be 53%. Colors indicate credible areas calculated by using 10,000 posterior draws to estimate a three-dimensional kernel density. The dark areas indicate the highest density regions with the most credible estimates, while the light areas contain 95% of the draws (i.e., the probability that the true coefficients lie outside the colored regions is $p = 0.05$).

FIGURE 2. Heritability of Voter Turnout in Los Angeles

We estimated separate election-type-specific heritability parameters within the same model. This is equivalent to including a dichotomous interaction term on the A parameter. When we compared the four closest elections to the other four elections, the difference in heritability was insignificant ($-13\%$, 95% C.I. $-71\%$, 50%). Similarly, the difference in heritability between the three primaries and the three general elections was also insignificant ($+16\%$, 95% C.I. $-46\%$, 70%).

Brooks and Gelman (1998) statistic (values of less than 1.1 on each parameter indicate convergence). For the Los Angeles voting models the “burn-in” period was 500,000 iterations and for the Adolescent Health voting and political participation models it was 1 million iterations. The Los Angeles and Adolescent Health models respectively were thinned by 100 and 200 for the posterior sample.
One potential objection to our model is that by including an election-specific fixed effect (the “difficulty” parameter in the model), we automatically remove “institutional variation” from the model (e.g. the procedural differences between primary and general elections which may influence turnout). To determine the extent to which including fixed effects for each election in the model may be artificially deflating the amount of variance to be explained, we also generated results from a model in which the difficulty parameters were removed and turnout was purely a function of the latent propensity to vote and election factor loadings. This robustness check ensures that institutional variation is included in total variance. The results of this model indicate heritability of 51% (C.I. 9%, 89%).

Another potential objection to our model is in fixing the genetic variance in DZ twins to be half the value of MZ twins, which is tantamount to assuming that DZ twins share exactly 50% of their genes—in reality, there is some variance from pair to pair in the amount shared resulting from the small number of recombinations on each chromosome that are possible. The empirical distribution has been estimated to be approximately normal with a mean of 50% and a variance of 0.13% (Visscher et al. 2006). When we incorporate this distribution in the Bayesian model instead of assuming an exact figure of 50%, the heritability estimate and confidence intervals are nearly identical (53%, C.I. 10%, 89%).

We reiterate that an important assumption of classical twin studies is that MZ and DZ twins share comparable social environments. Therefore, greater similarity of the phenotype in MZ twins compared to DZ twins indicates the degree to which genes influence the phenotype. If this assumption is violated, it is possible that the estimated genetic effect is inflated. In our study, violation of the “equal environments” assumption likely would have produced significant differences between MZ and DZ twins in the distribution of turnout, party affiliation, education, and socioeconomic status. Because the distributions of these variables do not appear to differ for the two types of twins, any possible overestimation of the genetic effect is likely to be small.

Another factor to consider is assortative mating. One assumption of the ACE model is that the distribution of parent genotypes is independent. If political participation is heritable and if people who participate in politics tend to have children with other politically-active individuals, then this will increase the concordance in participatory behavior in their children. However, the extent of this assortment is to increase the degree of concordance in offspring, making it harder to detect differences in MZ and DZ twins. For example, perfect assortment and perfect genetic transmission would yield a concordance of 1.0 for both MZ and DZ twins, and this lack of difference in the concordance would suggest that heritability plays no role. As a result, the more assortative mating there is, the more it biases downward the estimate of heritability. Thus, if the possibility exists that people choose mates based in part on their disposition to participate in politics, then the ACE model estimates will be conservative—heritability will actually be underestimated.

INDEPENDENT REPLICATION IN THE ADD HEALTH STUDY

Given the narrow geographic region of our study, we decided to conduct an independent replication of the results using data from a nationally representative sample. The National Longitudinal Study of Adolescent Health (Add Health) is a study that, among other topics, explores the causes of health-related behavior of adolescents in grades 7 through 12 and their outcomes in young adulthood. Three waves of the Add Health study have been completed: Wave I was conducted in 1994-1995, Wave II in 1996, and Wave III in 2001–2002. In Wave I of the Add Health study, researchers created a genetically informative sample of sibling pairs based on a screening of a sample of 90,118 adolescents. These pairs include all adolescents that were identified as twin pairs, half-siblings, or unrelated siblings raised together. Twins and half biological siblings were sampled with certainty. The Wave I sibling-pairs sample has been found to be similar in demographic composition to the full Add Health sample (Jacobson and Rowe 1998). Nearly 80% of the sibling-pairs sample participants in Wave I also participated in Wave III (Haberstick et al. 2005) and the demographic characteristics of the sibling-pairs sample did not change significantly over the course of the three waves (Hopfer et al. 2005). The total number of twins who participated in Wave III was 1,082 (442 MZ and 640 DZ), with 806 twins (442 MZ and 364 DZ) in same sex pairs.

The Add Health data has been used in a wide variety of twin studies (Harris et al. 2006). As a result, there have been several analyses of the comparable environments assumption for MZ and DZ twins. One of these studies claimed that the environments were not comparable (Horwitz, Videon, and Schmitz 2003), but other scholars have pointed to serious deficiencies in this work (Freese and Powell 2003). In consonance with most studies of the Add Health twin data, we conduct our own assessment of equal environments in Table 3 and find no significant differences in MZ and DZ environments for several socioeconomic and politically relevant variables.

In Wave III of the Add Health study, respondents provided information about their recent political activity that will permit analysis of both voting and other kinds of participation. This includes one question about voting: “Did you vote in the most recent presidential

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12 Women make up 49% of the study’s participants, Hispanics 12.2%, Blacks 16.0%, Asians 3.3%, and Native Americans 2.2%. Participants in Add Health also represented all regions of the country: the Northeast made up 17% of the sample; the South 27%, the Midwest 19%, and the West 17%.

13 Detailed information about the Add Health study can be found at www.cpc.unc.edu/projects/addhealth.

14 For example, Horwitz et al. (2003) showed that including observed social variables in a twin model causes the p-value on the genetic component for males trying alcohol to change from being just below 0.05 to just above it. Freese and Powell (2003) note that this is unsurprising since adding variables to a regression can have a substantial effect on efficiency. Even worse, they point out that Horwitz et al. (2003) do not acknowledge that their own fit statistics indicate the models with and without social variables are statistically indistinguishable, suggesting that the model with additional variables should be rejected!
election?” It also includes five questions about other kinds of political participation: “Which of the following types of organizations have you been involved with in your volunteer or community service work in the last 12 months?” (“political clubs or organizations”) “Which of the following things have you done during the last 12 months?” (“contributed money to a political party or candidate”; “contacted a government official regarding political or community issues”; “run for a public office”; “run for a non-public office”; “attended a political rally or march”). Due to low incidence, we pooled the two “run for office” questions to create a variable indicating whether the subject ran for any office, public or nonpublic. We performed a factor analysis of these five variables that suggested they all relate to an underlying tendency to participate in politics. A Cronbach test of internal consistency (α = 0.61) reveals that it is reasonable to include these variables in a model in which a single scalar latent value for participation is being estimated (see Verba, Schlozman, and Brady 1995, who report a similar α for a scale of participation that includes these items).

It is important to note that there are several differences between the Los Angeles sample and the Add Health sample. First, Add Health is nationally representative, suggesting that the results are more likely to generalize to the population outside Los Angeles. Second, Add Health includes subjects who were eligible but not registered to vote. This is important because the act of registration itself may be an important part of the decision to vote. Third, Add Health relies on self-reported turnout instead of official records meaning it is more susceptible to overreporting than the Los Angeles sample. Fourth, Add Health is restricted to young adults in their late teens and twenties (all eligible to vote)—thus, while it increases generalizability with respect to geography, socioeconomic composition, and local political conditions, it decreases generalizability with respect to age. Finally, Add Health includes data on turnout for just a single election compared to eight in the Los Angeles data. As a result, the greater efficiency of a larger sample may be partially offset by fewer observations per individual.

There are also some small differences in the modeling of the Add Health data. The Add Health voting model is based on a single election, k = (1), therefore subject j is a member of family i choosing to vote (Tij = 1) or abstain (Tij = 0) in the election. As in the Los Angeles voting model, the observed phenotypes are dichotomous variables and we assume τ is a continuous variable that maps to the individual’s latent propensity to vote via a logit function. In fact, the only difference is that we restrict δ1 = 1 to identify the model since there is only a single election for subjects in the Add Health data.

The only difference between the Add Health model of political participation and the Los Angeles voting model is that the dichotomous outcome variables in the former indicate whether subjects participated in various acts of participation rather than whether or not they voted in various elections. The latent tendency to participate in political activities in the Add Health sample is modeled in the same manner as the latent tendency to vote in the Los Angeles sample.

The results of both replications using the Add Health study show that participatory behavior is heritable. Figure 3 shows that about 72% of the variance in turnout behavior can be attributed to genes (95% C.I. 32%, 93%). The shared environment accounts for 20% of the variance (95% C.I. 1%, 57%), but an AE model without common environment actually fits the data better than the ACE model (see Appendix). Figure 4 shows that genetic effects account for 60% (C.I. 11%, 91%) of the variance in political participation with the shared environment having little effect (18%, C.I. 1%, 54%). Once again, an AE model without shared environment fits better, suggesting that most variance can be attributed to genetic and unshared environmental factors. In summary, both Add Health replications yield estimates of heritability that are similar in magnitude to the 53% estimate for heritability in the Los Angeles sample, suggesting the heritability of political participation is robust.15

15 We also re-ran the Los Angeles voting model, AddHealth voting model, and AddHealth political participation model with separate heritability, common, and shared environment components for males and females. This was done to ensure pooling males and females is appropriate. The DIC for the Los Angeles gender-specific voter model was higher than for the pooled model indicating the pooled model fits the data better. The DIC for the AddHealth gender-specific model is lower than the pooled model, however the male and female heritability estimates are nearly identical (0.69 for males and 0.66 for females). Finally, the DIC for the AddHealth political participation index is higher for the gender-specific model.
FIGURE 3. Heritability of Voter Turnout in Add Health

Note: Ternary plot shows the posterior Bayesian distribution of estimated components of total variance in an ACE model of voter turnout among subjects in the National Longitudinal Study of Adolescent Health (Add Health). Mean heritability ($h^2$) is estimated to be 72%. Colors indicate credible areas calculated by using 10,000 posterior draws to estimate a three-dimensional kernel density. The dark areas indicate the highest density regions with the most credible estimates, while the light areas contain 95% of the draws (i.e., the probability that the true coefficients lie outside the colored regions is $p = 0.05$).

FIGURE 4. Heritability of Political Participation in Add Health

Note: Ternary plot shows the posterior Bayesian distribution of estimated components of total variance in an ACE model of political participation among subjects in the National Longitudinal Study of Adolescent Health (Add Health). Mean heritability ($h^2$) is estimated to be 60%. Colors indicate credible areas calculated by using 10,000 posterior draws to estimate a three-dimensional kernel density. The dark areas indicate the highest density regions with the most credible estimates, while the light areas contain 95% of the draws (i.e., the probability that the true coefficients lie outside the colored regions is $p = 0.05$).

It is important not to confuse these estimates with those from other models in the turnout literature. They are not comparable. For example, we referred to another study (Plutzer 2002) earlier in which environmental factors account for only 31% of the variance in turnout, but many of the variables in that model might well include genetic effects (for example, parental turnout might in part be a proxy for genetic association). It is also possible that there are as-yet undiscovered or unmeasured environmental factors that will improve the fit of that model.

Nor can we state with certainty that genetic effects are somehow more important than environmental effects. Although we estimate that genetic variation accounts for more than 50% of the variance in participation in all three tests, these estimates are based on a simple additive genetic model that undoubtedly masks richer and more complex gene-environment interactions. We therefore strongly discourage readers from perceiving these results as a horse race between genes and environment. In fact, our results suggest that both genes and environment matter, and our job now is to look closer at both to understand better how nature and nurture work together to create the political phenomena we observe in the world.

DISCUSSION

The fact that we have found that genetic variation in voting, and political participation in general, should not be surprising given the large number of behaviors that have already been found to be heritable (Bouchard and McGue 2003; Turkheimer 1998). However, our goal is not simply to show that political behavior can be added to this long list of behaviors. Instead, we suggest that our findings are the first step in a research agenda with the goal of uncovering biological sources of participatory behavior, a finding that would have important implications for political science in general and studies of voting behavior in particular.

Political scientists have typically not focused on the role of genetic and biological factors in political behavior (Alford, Funk, and Hibbing 2005), which has potentially biased our interpretations of several important phenomena. For example, if political participation is heritable, it would help to explain why models based primarily on environmental variables fit poorly to observed behavior (Matsusaka and Palda 1999). It would also conform to two well-known features of voting. First, parental turnout behavior has been shown to be one of the strongest predictors of turnout behavior in young adults (Plutzer 2002). Although this has previously been interpreted as the result of social influence, the findings here suggest it may be mostly due to heritability since the shared environment appears to play only a small (if any) role. Second, turnout behavior has been shown to be habitual—the majority of people either always vote or always abstain (Fowler 2006b; Gerber, Green, and Shachar 2003; Green and Shachar 2000; Miller and Shanks 1996; Plutzer 2002; Verba and Nie 1972). Scholars previously interpreted this as the result of reinforcement learning, but given the small
effect of environmental variation it might also be largely due to inherent genetic variability.

While the results here suggest a significant role for genes, they are completely silent on the specific mechanism that links genes to participation. Therefore, the next step in this line of research must move beyond estimates of heritability and attempt to identify why genes matter so much. There are many possible mechanisms one could imagine, but here we speculate on a few.

The theoretical literature on voting has centered on rational, self-interested models (Aldrich 1993; Downs 1957; Riker and Ordeshook 1968) that have great difficulty explaining high turnout in large populations. One popular extension to these models is to assume that some individuals experience an extra benefit from voting (the “D” term as Riker and Ordeshook called it) that has nothing to do with the outcome. Instead, this benefit comes from the satisfaction of fulfilling a civic duty or of contributing to the democratic process. In other words, these models posit that there is inherent heterogeneity in the desire to vote. While many scholars believe this argument is plausible (notably Aldrich (1993, p. 266) argues “most of the action is probably in the intrinsic values of voting per se”), not a single one has suggested that this heterogeneity may have genetic origins. Thus, our results suggest that a fruitful avenue for future research is to study whether or not variation in feelings of civic duty intermediate the relationship between genes and political participation.

A more recent extension to the rational model posits that voters get utility for behaving “ethically” as a way of coordinating high participation equilibria between competing groups (Sandroni and Feddersen 2006). This argument is also plausible, but since it is based on equilibrium analysis, it is agnostic about the origin of the preference for ethical behavior. The evidence here suggests that genetics may play a role. The ethical voting model works equally well in small groups and large populations, so it is possible that the ethical mechanisms underlying equilibrium evolved genetically in small-scale settings in early human societies and then continued to have an influence as humans became involved in the larger-scale behavior of recent history.

Another possibility is that variation in voting and participation are related to variation in prosocial behavior. A wide range of studies have already shown a strong genetic basis for prosocial personality and behavior (McGue, Bacon, and Lykken 1993; Rushton et al. 1986; Scourfield et al. 2004; Cesarini et al. 2008). This literature suggests that innate dispositions play a significant role in an individual’s willingness to participate in social activities or to engage in acts that primarily benefit others. Meanwhile, observational studies (Edlin, Gelman, and Kaplan 2007; Jankowski 2002; Jankowski 2007) and laboratory experiments (Fowler 2006a; Fowler and Kam 2007) suggest that prosocial attitudes and behavior are important factors for explaining voter turnout and political participation. Thus, genes may influence voting and political participation because they influence a generalized tendency to engage in social behavior.

The frontier before us is vast. Future work should explore the interaction effects of genes and environment on participation. These studies will help us to learn what the causal mechanisms are that link genes which have taken millions of years to evolve to large-scale political behavior which is an extremely recent phenomenon on the scale of human evolution. Evidence of political behavior in chimpanzees (de Waal 1998 [1982]), capuchins (Brosnan, Freeman, and de Waal 2006), and early human societies (Boehm 1999) suggests that it may have, in part, adapted genetically to small-scale interactions, but it is an open question whether or not these small-scale adaptations influence large-scale political participation. The obvious place to start is with factors for participation that have already been identified like cognition and efficacy, which also have a genetic basis (McGue and Bouchard 1998). It is also possible that genes influence political participation via their effect on personality traits that have not yet been linked to it, like their effect on assertiveness or competitiveness. Thus an important area of research will study the extent to which the link between genes and participation can be explained by genetic variation in inherent personality attributes.

Future research should also begin the work of identifying genes that are implicated in political behavior. It is extremely unlikely that such efforts will uncover a “voting gene”, however, the results presented here suggest that there is some (possibly large) set of genes whose expression—in combination with environmental factors—regulates political participation. Finding out which genes they are and what physical function they have will improve our understanding of the biological processes that underlie these complex social behaviors and may also shed light on their evolutionary origin (Fitzpatrick et al. 2005).

Finally, we offer a note of caution. Heritability studies have shown that genes account for some of the variance in a very large set of human behavior, including activities like television watching that are extremely recent in human history and not (yet) relevant to genetic evolution. In particular, Turkheimer (1998) argues that these results have been well known in other disciplines for a very long time, but expectations that they would lead to the discovery of specific “deeper” biological explanations of human behavior have largely been disappointed. There are simply too many genes and too many causal steps between genes and behavior to expect that genetic analysis will ever lead to improved understanding. Moreover, high heritability for a phenotype does not guarantee that it will be possible to identify specific genes that contribute to it. For example, in cancer genetics the least heritable cancers have been the most amenable to molecular genetic analysis, because they are rare and caused by single genes of large effect. Highly heritable cancers are more common and highly polygenic and it is therefore harder to identify genes for them (Risch 2001).16

However, the recent revolution in genotyping presents possibilities that were not available to

16 Thanks to Eric Turkheimer for bringing this idea to our attention.
behavior geneticists when they first uncovered evidence of the heritability of complex social behaviors. Scholars have already begun discovering specific genes associated with political behavior, which may be the first few pieces in the puzzle to understanding the biology that underlies it. For example, two studies (Fowler and Dawes 2008; Dawes and Fowler 2008) recently identified variants of three genes that are positively correlated with voter turnout. The genes they studied are known to influence social behavior via the dopaminergic and serotonergic systems, suggesting that voting may, in fact, be a prosocial act. Moreover, the association between one of these genes and turnout appears to be mediated by partisanship (Dawes and Fowler 2008). Thus, the realization that participation is heritable has already helped to generate additional evidence that may be applied to existing theories of turnout, partisanship, and prosocial behavior, and it also yielded new theories about the effect of the serotonin and dopamine system on participation. Therefore, although it may not surprise behavior geneticists that participation is heritable, it seems premature to argue that heritability studies will not bear fruit in political science. These studies provide the first step needed to excite the imaginations of a discipline not used to thinking about the role of biology in human behavior.

APPENDIX

### TABLE A1. Summary of Model Results

<table>
<thead>
<tr>
<th></th>
<th>Heritability</th>
<th>Common Environment</th>
<th>Unshared Environment</th>
<th>Deviance Information Criterion (DIC)</th>
<th>Dbar</th>
<th>pD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$h^2$</td>
<td>$c^2$</td>
<td>$e^2$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Los Angeles Turnout</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACE</td>
<td>0.53</td>
<td>0.35</td>
<td>0.12</td>
<td>2643.4</td>
<td>2351.6</td>
<td>291.9</td>
</tr>
<tr>
<td>(0.10, 0.89)</td>
<td>(0.02, 0.73)</td>
<td>(0.03, 0.26)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AE</td>
<td>0.86</td>
<td>0.14</td>
<td></td>
<td>2639.2</td>
<td>2347.9</td>
<td>291.3</td>
</tr>
<tr>
<td>(0.71, 0.95)</td>
<td>(0.05, 0.29)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Add Health Turnout</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACE</td>
<td>0.72</td>
<td>0.20</td>
<td>0.09</td>
<td>852.8</td>
<td>532.6</td>
<td>320.2</td>
</tr>
<tr>
<td>(0.32, 0.93)</td>
<td>(0.01, 0.57)</td>
<td>(0.05, 0.15)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AE</td>
<td>0.91</td>
<td>0.09</td>
<td></td>
<td>850.5</td>
<td>528.3</td>
<td>322.3</td>
</tr>
<tr>
<td>(0.85, 0.95)</td>
<td>(0.05, 0.15)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Add Health Political Participation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACE</td>
<td>0.60</td>
<td>0.18</td>
<td>0.23</td>
<td>615.3</td>
<td>490.8</td>
<td>124.5</td>
</tr>
<tr>
<td>(0.11, 0.91)</td>
<td>(0.01, 0.54)</td>
<td>(0.04, 0.59)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AE</td>
<td>0.70</td>
<td>0.30</td>
<td></td>
<td>605.5</td>
<td>489.8</td>
<td>115.7</td>
</tr>
<tr>
<td>(0.31, 0.93)</td>
<td>(0.07, 0.69)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Note:** These results show that we consistently found that a large proportion of variance in turnout and political participation behavior is due to heritability and that the best fitting models are those that assume a role for heritability and the unshared environment (but not the common environment). The first column describes each model. ACE models estimate a parameter for genetic ($A$), common environment ($C$), and unshared environment ($E$); AE models assume the common environment has no effect. Columns 2, 3, and 4 show the mean estimated proportion of total variance attributed to heritability ($h^2$), common environment ($c^2$), and unshared environment ($e^2$), with 95% credible intervals indicated in parentheses below each estimate. Model fit is assessed using the deviance information criterion (DIC), which penalizes models for deviance (Dbar), capturing model fit, and the effective number of parameters (pD), capturing model complexity. The results show that the AE model generates the best fit for all three samples. The empirical means, 95% credible intervals, and DICs reported for the Los Angeles voting models are based on 10,000 draws from the posterior distribution.

### TABLE A2. Posterior Predictive Checks

<table>
<thead>
<tr>
<th>Discrepancy Measure:</th>
<th>Realized Value</th>
<th>Predicted Value (Predicted)</th>
<th>95% CI</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>% Voting in</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No elections</td>
<td>9.1</td>
<td>7.7</td>
<td>[5.3, 10.4]</td>
<td>0.17</td>
</tr>
<tr>
<td>One election</td>
<td>12.4</td>
<td>13.5</td>
<td>[10.6, 16.7]</td>
<td>0.74</td>
</tr>
<tr>
<td>Two elections</td>
<td>12.4</td>
<td>13.5</td>
<td>[10.4, 16.7]</td>
<td>0.72</td>
</tr>
<tr>
<td>Three elections</td>
<td>11.1</td>
<td>11.1</td>
<td>[8.3, 14.1]</td>
<td>0.46</td>
</tr>
<tr>
<td>Four elections</td>
<td>13.4</td>
<td>11.5</td>
<td>[8.6, 14.4]</td>
<td>0.09</td>
</tr>
<tr>
<td>Five elections</td>
<td>8.8</td>
<td>10.9</td>
<td>[8.1, 13.9]</td>
<td>0.90</td>
</tr>
<tr>
<td>Six elections</td>
<td>11.9</td>
<td>10.7</td>
<td>[7.8, 13.6]</td>
<td>0.23</td>
</tr>
<tr>
<td>Seven elections</td>
<td>11.4</td>
<td>11.3</td>
<td>[8.3, 14.4]</td>
<td>0.43</td>
</tr>
<tr>
<td>Eight elections</td>
<td>9.6</td>
<td>9.8</td>
<td>[7.1, 12.6]</td>
<td>0.60</td>
</tr>
</tbody>
</table>
REFERENCES


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The Heritability of Partisan Attachment

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One of the strongest regularities in the empirical political science literature is the well-known correlation in parent and child partisan behavior. Until recently, this phenomenon was thought to result solely from parental socialization, but new evidence on genetic sources of behavior suggests it might also be due to heritability. In this article, the authors hypothesize that genes contribute to variation in a general tendency toward strength of partisanship. Using data collected at the Twins Days Festival, the authors compare the similarity of partisan strength in identical twins who share all of their genes to the similarity of partisan strength in nonidentical twins who share only half of their genes. The results show that heritability accounts for almost half of the variance in strength of partisan attachment, suggesting we should pay closer attention to the role of biology in the expression of important political behaviors.

Keywords: political psychology; political methodology; public opinion and political participation

The study of partisanship occupies a vast part of the political behavior literature because of the complexity of what it means, how it forms, and what it predicts (e.g., Campbell et al. 1960; Fiorina 1981; Niemi and Jennings 1990; Popkin 1991; Gerber and Green 1998). Partisanship is typically evaluated along two dimensions—the strength of reported partisan attachment and the direction of that attachment. While there is much divergence of opinion on the nature and measurement of partisanship, scholars have almost exclusively focused their attention on the socialization process and environmental determinants of the origin, direction, and intensity of partisanship. However, recent work has demonstrated that heredity plays a role in closely related political behaviors, such as political attitudes (Alford, Funk, and Hibbing 2005; Hatemi et al. 2007; Tesser 1993), political orientations (Alford, Funk, and Hibbing 2005; Settle et al. 2008), voting behavior (Fowler, Baker, and Dawes 2008; Fowler and Dawes 2008; Dawes and Fowler 2008), and trust (Cesarini et al. 2008). The developing consensus that genes play an important role in political behavior leads us to believe that heritability could also help to explain one of the remaining questions in the partisanship literature: what contributes to the underlying strength of a person’s partisan identity?

What Is Partisanship?

Party identification was originally conceived as an affective attachment resulting from the process of socialization (Campbell et al. 1960), stemming from childhood and reflecting the influences of the immediate social milieu and the family (Hyman 1959; Greenstein 1965). Subsequent work built on this social psychological view argued that identification with a particular party is based on images of that party as a social group (Gerber and Green 1998; Green, Palmquist, and Schickler 2002; Fowler and Kam 2007). The authors of The American Voter (Campbell et al. 1960) essentially viewed strength of partisanship as a fixed factor that could be used to predict political behavior, but they could only speculate as to why or how it was fixed.

Since the 1970s, a debate in the literature has contested whether partisanship is affective, nearly immutable, and emotionally based or whether it is better conceived as instrumental, changeable, and responsive to current conditions and attitudes toward contemporary political events. As opposed to the social psychological interpretations of partisanship, instrumental theories view partisan attachment as an information shortcut that is continually updated and
adjusted based on rational evaluation (Fiorina 1981; Popkin 1991). For example, Achen (1992) argued that voters utilize a Bayesian process, prospectively judging parties based on their observations of the party’s past performance and information received from a campaign. Some research indicates that voters receive “noisy” signals about party performance, originating at either the individual level or system level of the information environment, or both. If, due to high levels of individual-level noise, voters cannot determine party differences, they may be less likely to form party attachments (Huber, Kernell, and Leoni 2005).

Instrumentalists have challenged the use of partisanship as an independent variable, instead asserting that it is not as stable as it was originally conceived (Fiorina 1981; Franklin and Jackson 1983). For example, presidential approval, consumer sentiment, specific political events, and attitudes toward particular administrations affect levels of partisanship in the population at large (Mackuen, Erikson, and Stimson 1989; Brody and Rothenberg 1988; Converse and Markus 1979; Meier 1975; Page and Jones 1979). However, recent experimental manipulations show that simulating the effects of short-term political forces does not have an effect on party choice, reinforcing traditional notions of partisanship (Cowden and McDermott 2000).

Beyond the debate over what partisanship is, scholars have offered a series of interpretations about what causes it to vary, both in strength and in direction. Most scholarship suggests that the seeds of partisanship are planted early in life and that strength of partisanship develops and changes over the life course. One of the few relatively consistent findings in the literature is that partisanship strengthens over the course of a lifetime, although interpretations for this vary. The authors of The American Voter argued that the strength of partisan attachment increases with age as the result of an individual becoming more active within the community and associating with social groups, some of which have partisan ties (Campbell et al. 1960). While Converse (1969, 1972, 1976, 1979) suggested that partisanship is formed early in life and reinforced by experiences interpreted through a partisan lens, Abramson (1976, 1979a, 1979b) argued that generational effects and period effects play a more significant role in shaping partisanship strength over time. Further studies have shown that parental partisanship has an influence on children that is strongest at age eighteen but continues into adulthood and that issues play an increasingly important role in partisanship formation over time (Niemi and Jennings 1991).

Given the importance of the question of stability of partisanship and the well-known correlation between parent and child partisan behavior, it is somewhat surprising that this literature has ignored heritability as a factor in partisan attachment. As we have indicated, there are many examples of careful empirical studies of theories of partisan attachment, but almost all have focused exclusively on environmental explanations. If a substantial portion of our tendency to attach to parties is passed from parent to child via genetic predispositions, it could help explain stability of partisanship over time since, barring recombination or mutation, genetic factors are fixed over the course of our lifetimes. Moreover, since children share half of each parent’s genes, it might help to explain within-family correlations in partisanship.

Recent work has shown that genetic factors account for a significant proportion of variation in social attitudes (Martin et al. 1986; Tesser 1993) and political attitudes related to the direction of partisan identification (Alford, Funk, and Hibbing 2005; Hatemi et al. 2007; Settle et al. 2008). The strength of political opinion, defined as the percentage of non-neutral responses on a survey of political opinions, has also been demonstrated to have a genetic component (Alford, Funk, and Hibbing 2005). Likewise, genetic factors are also important for political behaviors like voting (Fowler, Baker, and Dawes 2008; Fowler and Dawes 2008; Dawes and Fowler 2008) that are known to be influenced by the tendency to attach to a given party.

However, while heritability plays a role in many political behaviors, we do not expect that it will play a role in all political behaviors (Alford and Hibbing 2008), and we have reason to expect a difference in the heritability of strength versus direction of partisanship. As measured in both our sample and in Hatemi (2007) and Hatemi et al. (2007), identifying oneself as a “strong” partisan is a reflection of the propensity toward group attachment. Research on the heritability of religion has found that while religiosity is strongly heritable, denominational affiliation is not (Eaves 1977; Martin et al. 1986; Bouchard and McGue 2003; Koenig et al. 2005; Bouchard et al. 1999; Beer, Arnold, and Loehlin 1998; Bouchard et al. 2004). We conjecture that the relationship between strength of partisan attachment and party identification is analogous to the relationship between religiosity and denominational affiliation (Hatemi et al. 2007; Jennings, Stoker, and Bower 2001). That is, the intensity of one’s attachment to a group may be shaped by genetic predispositions, but the selection...
of the group to which one attaches is largely shaped by parental and environmental exposures. We expect a different pattern for ideology, which is best viewed as an orientation toward the social world and its organization (Jost 2006; Alford and Hibbing 2008). Ideological orientations may be heritable because of their social psychological role (Jost et al. 2003; Jost 2006; Alford and Hibbing 2008), but the intensity of these beliefs may not have a strong heritable component since the underlying psychological factors associated with liberalism (like openness) and conservatism (like conscientiousness) are directional.

In this article, we hypothesize that genes contribute to variation in a general tendency toward strength of partisanship. Using data collected at the Twins Days Festival in 2006 and 2007, we compare the similarity of partisan strength in identical twins who share all of their genes to the similarity of partisan strength in nonidentical twins who share only half of the genes that vary between human beings, on average. The results indicate that strength of partisan attachment is heritable, and they suggest that we should pay closer attention to the role of biology in the expression of important political behaviors. Although these results do not identify specific causal mechanisms underlying these genetic predispositions, they do indicate that we should be searching for biological and genetic sources of partisan behavior.

**Twin Studies**

The technique currently best suited to study the contribution of heredity to political behavior is the twin study, a method used for decades by psychologists but used only recently in political science. The twin study model cannot tell us which genes contribute to a particular behavior or the mechanism by which genes and the environment interact to produce certain phenotypes, but it is useful for testing the existence of a genetic component and establishing the relative importance of the environment for a specific behavioral outcome.

By partitioning phenotypic variance into its constituent components, we can estimate the role of heredity versus environment, or nature and nurture. The “relative importance” of heredity, or the proportion of the total phenotypic variance due to genes transmitted by parents to their offspring, is called the heritability of the character (Falconer and Mackay 1996). We can estimate the degree to which partisanship strength is heritable by studying the reported strength of partisanship of (identical) monozygotic (MZ) twins, conceived from a single egg; and (non-identical) dizygotic (DZ) twins, conceived from two separate eggs. MZ twins share 100 percent of their genes, while DZ twins share only 50 percent on average. Thus, if the decision to vote is based in part on genetic characteristics, MZ twins should exhibit more behavioral concordance than DZ twins. Based on the assumption that MZ twins and DZ twins share comparable environments, we can use these concordances to estimate explicitly the relative influence of genetic, shared environmental, and unshared environmental factors.

The twin study design has been shown to be an extremely powerful tool for identifying the relative degree to which genetic and environmental factors influence an observed outcome (Evans, Gillespie, and Martin 2002; Neale and Cardon 1992). The reasoning behind this model relies on the principle that all independent variables explaining political behavior can be put into three mutually exclusive categories: additive genetic factors (A), shared or common environmental factors (C), and unshared environmental factors (E). We describe this model in the appendix.

It is important to clarify the difference between the common environment and the unshared environment in the twin model. Common environment includes the family environment in which both twins were raised as well as any other factor to which both twins were equally exposed. In contrast, the unshared environment includes idiosyncratic influences that are experienced individually. It is possible to have unshared environmental exposure as a child (twins may have different friends with different political beliefs) and to have shared environments as an adult (twins may see the same election results). Thus, the distinction between common and unshared environment does not correspond directly to family–nonfamily or adult–child differences in factors that influence a given behavior. Moreover, there may be a similarity in the objective environment, but twins may have idiosyncratic experiences that influence their effective environment that create an unshared rather than a common environmental influence on variation in the phenotype (Turkheimer 2000). For example, twins may watch the same campaign speech but process it differently, with different effects on their feelings towards the competing parties.

Some scholars argue that the identical nature of MZ twins causes them to be more strongly affiliated and more influenced by one another than their non-identical DZ counterparts, which would indicate that...
greater concordance in MZ twins might merely reflect the fact that their shared environments cause them to become more similar than DZ twins. This situation would violate the assumption that MZ and DZ environments are comparable. However, studies of twins raised together have been validated by studies of twins reared apart (Bouchard 1998), suggesting that the shared environment does not exert enhanced influence on MZ twins. Furthermore, even among twins whose zygosity has been misclassified by their parents, personality and cognitive differences between MZ and DZ twins persist (Bouchard and McGue 2003), indicating that being mistakenly treated as an identical twin by one’s parents is not sufficient to generate the difference in concordance. Although MZ twins may sometimes be in more frequent contact with each other than DZ twins, it appears that twin similarity (e.g., in attitudes and personality) may be the cause of greater contact rather than an effect (Posner et al. 1996). Finally, contrary to the prediction that the influence of the unshared environment would tend to reduce concordance over time once twins reach adulthood, MZ twins living apart tend to become more similar with age (Bouchard and McGue 2003).

The ACE model measures the total variance in a given phenotype, in this case partisanship strength, and then estimates the relative contributions of genetic and environmental influences separately to the total observed variance of that phenotype. The roles of genotype and environment are not measured directly, but their influence is inferred through their effects on the covariances of twin siblings (Neale and Cardon 1992). While the ACE model does not indicate the specific causal mechanisms that interact with genes and/or mediate the relationship between genes and strength of partisanship, it is a useful tool to establish whether genes play a role and, thus, whether they merit further study to explain aspects of political behavior. The ACE specification is the simplest model of genes, shared environment, and unshared environment; more complicated interactions certainly occur in nature, but a strong effect for genes in the additive ACE model indicates that genes are also likely to play a role in more complex specifications as well.

**Twins Days Festival Data Collection**

The data used in this study were gathered from a sample of twins in attendance at the 2006 and 2007 Twins Days festival in Twinsburg, Ohio. This festival attracts about 2,000 pairs of twins each year, who, in addition to participating in social events, have the option to volunteer for a number of research studies. The sample consists of 353 pairs of same-sex adult twins, aged eighteen or older (mean age of thirty-six), in attendance at the annual festival in August 2006 and 2007. A condition for participation was that both twins in a pair were able to complete the survey. In total, 706 individuals participated (75 DZ and 278 MZ same-sex pairs of twins). Zygosity was determined by self-report, which has been used previously for studies of Twins Days participants (Ashenfelter and Krueger 1994). In particular, one study of 86 Twins Days subjects showed self-report to be 100 percent reliable compared to a genotypic assessment of zygosity (Wise et al. forthcoming). Subjects who had participated in 2006 were excluded from taking part in 2007.

Participants were asked a series of demographic questions and a question about their partisan affiliation, using the traditional 7-point partisanship scale. This scale was then “folded over” to produce a 4-point, directionless scale of partisan attachment. This is a variation of the standard National Election Studies (NES)/Michigan party identification scale, the most frequently used scale of partisanship in the literature (Weisberg 1999). This scale has been found to tap into respondents’ general partisan tendencies and may be less sensitive to transitive or election-specific partisan preferences than other measures (Whiteley 1988; Green and Palmquist 1990; W. Miller 1991; Abramson and Ostrom 1991, 1992, 1994a, 1994b; W. Miller and Shanks 1996). Moreover, this measure has been used frequently as an explanatory variable in models of political participation (Timpone 1998; Verba, Schlozman, and Brady 1995). Because we are most interested in the long-term aspects of partisan strength, the wording of this question makes it most appropriate for our purposes.

Table 1 shows summary statistics for study participants. One method of evaluating whether MZ and DZ twins are drawn from comparable environments is to examine the distributions of relevant covariates between the two groups. If there are any significant differences between the MZ and DZ twins, we can interact the covariate in question with $A$ (the additive genetic component) in the ACE model to see if it impacts the heritability estimate. For example, there is a statistically significant difference in age between the MZ and DZ twins. However, when we add a variable for age to the ACE model, we do not find any evidence that the heritability of partisanship strength changes with age. Similarly, we find that MZ twins...
are somewhat more likely to be Republicans and ideologically intense.\(^2\) However, neither of these variables significantly moderates the heritability estimates. Thus, while this sample is small and not randomly selected, it does not appear to be systematically biased in a way that affects our ability to use the data to estimate the influence of genes on partisan attachment. Finally, because the sample was self-selected (both into attendance at the Twinsburg Festival and into the study section of the festival), we may have a disproportionate share of people who “like to participate,” indicative of some underlying tendency to associate or form group attachments. This could serve to increase the proportion identifying as strong partisans in our sample as compared to the general population, but it should not bias the relative contributions of genes and environment to that behavior. Any self-selection bias likely applies to DZ and MZ twins equally.

### Results

In the behavior genetics literature, a simple comparison of polychoric correlations is frequently used as a first test of the rate of twin concordance in behavior (for a detailed explanation of this method, see Alford, Funk, and Hibbing 2005). In our observations, the correlation in partisan strength is significantly different \((p = .0002)\) for MZ twins \((.46, 95\text{ percent confidence interval } [CI] = .34, .57)\) and DZ twins \((.16, 95\text{ percent CI } = –.15, .48)\) (see Table 2). Using the three variance equations noted in the appendix, it is easy to show that

\[
\sigma^2_A = 2(COV_{MZ} – COV_{DZ}).
\]

With some further manipulation, it is also possible to show that a similar relationship holds for correlation coefficients—heritability can be estimated as twice the difference between the correlations, which yields a value of \(.58\). In other words, a simple interpretation of the data suggests that genes account for about 58 percent of the variance in strength of partisan attachment.

However, this simple method does not account for the fact that our dependent variable is ordered and categorical, and it assumes that the latent distribution of partisan strength is normal even though we only observe four distinct values. We therefore estimate a full ACE model that includes parameters for heritability \((a^2)\), the common environment \((c^2)\), and the unshared environment \((e^2)\), as well as three thresholds between the four ordered categories that define the underlying distribution of partisanship strength (see the appendix). We use the software package MX to estimate this model (Neale et al. 2006).

The results suggest that heritability \((h^2)\) generates about 46 percent of the variance in strength of partisan attachment. The 95 percent CI for the estimate is \((5\text{ percent}, 57\text{ percent})\), indicating that we can reject the hypothesis that genes play no role in partisanship strength. The ACE model also suggests that the environment plays a role, although this is primarily due to the unshared environmental factors \((e^2)\), which account for 54 percent of the variance \((CI = 43\text{ percent}, 67\text{ percent})\). The shared environment \((c^2)\) does not seem to play a significant role \((0\text{ percent}, CI = 0\text{ percent}, 37\text{ percent})\). Measures of model fit indicate that an AE model is better than the ACE model. All of the models presented include controls for age and gender. These control variables only influence the estimation of the three thresholds and do not enter into the estimation of \(a^2\), \(c^2\), or \(e^2\).
Previous research indicates a modest genetic influence on vote choice (e.g., choice between Labor versus Conservative parties) but suggests that it could be attributed to an underlying genetic component in perceived attitudes and perceptions that intermediate the relationship with vote choice (Hatemi et al. 2007). Other research indicates that individual differences in political partisanship can be attributed mostly to the environment (Eaves, Eysenck, and Martin 1989; Olson, Vernon, and Jang 2001; Bouchard et al. 2003).

To test these theories in our sample, we repeated the analysis using a directional measure of partisanship in Table 3. Although the ACE model was the best fit of any of the models tried, we do not find a significant role for heritability in describing the direction of partisanship. This result is consistent with prior work suggesting the heritability of party choice is low (Alford, Funk, and Hibbing 2005). In other words, although genes appear to play a role in how strongly we attach to a given party, we do not find much evidence that they influence which party will be chosen.

We also wondered if these results could be explained by variation in the tendency for individuals to have extreme ideologies. After all, Alford, Funk, and Hibbing (2005) and Hatemi et al. (2007) found that the direction of political attitudes (liberal vs. conservative) is heritable. If so, then ideological intensity might also be heritable, helping to explain the link from genes to ideology to partisanship. In other words, genes might help explain who becomes an extreme liberal or conservative and, therefore, who becomes a strong Democrat or Republican. To test for this possibility, we ran an ACE model on strength of ideology, a scale that folds over the traditional liberal-conservative scale (see note 2). Table 4 shows that we cannot reject the null hypothesis that...
strength of ideology is not heritable. A is estimated to be 13 percent but not significantly different from 0. Therefore, our strength of partisanship result cannot be explained as merely the by-product of a genetic contribution to extreme ideological orientations.

Finally, it is important to note the possible effects of assortative mating on our results. One assumption of the ACE model is that the distribution of parent genotypes is independent. If partisanship strength is partially heritable and if strong partisans tend to have children with other strong partisans, for example, then there will be an increased concordance in partisanship strength in their children. However, this possibility actually serves to make it more difficult to detect differences between MZ and DZ twins. For instance, if a trait follows a pattern of perfect assortativity and is 100 percent genetically transmitted, we would observe a concordance of 1 for both MZ and DZ twins. The finding that the concordance between the two types of twins is the same would suggest that heritability does not contribute to the expression of the trait. Consequently, high assortativity tends to bias downward the estimate of heritability. If people choose mates based in part on their disposition to partisanship, then the ACE model estimates will be conservative and the contribution of heritability will actually be underestimated.

Discussion

We find that heritability plays a significant role in partisanship, accounting for almost half of the variance in strength of partisan identification. This heritability is probably not an artifact of ideological orientation since strength of ideology is not significantly heritable in the same sample. Nor is it an artifact of heritability in the direction of partisanship, which also fails to be significant for this sample. Instead, variation in the decision to identify with any political party appears to be strongly influenced by genetic factors.

Our findings replicate the work conducted by Peter Hatemi in his 2007 dissertation and in Hatemi et al. (forthcoming). Using a different sample, with data collected almost twenty years after the data analyzed in Hatemi’s work, we find nearly identical results. This replication across samples and time should reduce the concern that these findings are isolated or accidental. Furthermore, our finding builds on previous research demonstrating a genetic basis for other forms of political behavior, such as voter turnout (Fowler, Baker, and Dawes 2008; Fowler and Dawes 2008; Dawes and Fowler 2008), political attitudes (Alford, Funk, and Hibbing 2005; Hatemi et al. 2007), conservatism (Alford, Funk, and Hibbing 2005; Settle et al. 2008), and trust (Cesarini et al. 2008). It also reinforces the importance of examining the role of genetic factors in explaining political behavior instead of focusing solely on a multitude of environmental variables.

One implication of these results is that previous conceptions of the transmission and acquisition of partisanship should be reformulated. Strength of partisanship attachment—and identification as an independent (Mattei and Niemi 1991)—has in the past been attributed to political socialization from parent to child (Campbell et al. 1960; Converse 1969; Niemi and Jennings 1991; Hyman 1959; Greenstein 1965). However, our results suggest that the correlation between parent and child partisanship behavior is more likely to result from shared genes than the family environment. In fact, in our model we cannot reject the hypothesis that the common environment has no impact on strength of partisanship attachment whatsoever. Of course, given the broad literature on partisanship and parental socialization, we doubt the effect of parental socialization is really zero, but these results do suggest that we can rule out common familial experience as a major contributing factor to partisanship strength.

Our results also speak to the literature on the stability and nature of partisanship over time. Partisanship was originally thought to be stable and long-enduring (Campbell et al. 1960; Converse 1969), but this finding has been challenged (Abramson 1976, 1979a, 1979b; Fiorina 1981; Popkin 1991; Achen 1992; Mackuen, Erikson, and Stimson 1989; Brody and Rothenberg 1988; Converse and Markus 1979; Meier 1975; Page and Jones 1979). Exposing a role for heritability in determining partisan strength introduces an important, previously uncharacterized, explanation into the durability debate. Genetic expression is stable, and we show here that genes explain some variance in the strength of partisan attachment. Therefore, although we only observe individuals at a single point in time, we would expect individuals to exhibit some degree of stability in their partisan behavior. In this sense, our findings can help reconcile the debate in the literature as to whether partisanship is affective and immutable or changeable and responsive to current conditions. If the stable component of partisanship is conceived of as a genetic predisposition toward group affiliation, then the short-term effects that change partisanship
strength could be viewed as the unshared environmental factors that combine with genetic predispositions to change partisanship strength in an individual.

This would be consistent with Scarr and McCartney’s (1983) theory of how genetic and environmental differences combine to produce variation in development. They argued that the role of genotype determines which environments are experienced by individuals and which environments individuals seek for themselves; essentially, people seek out experiences to reinforce their genetic predispositions. Those inclined to be partisan seek out opportunities to do so, which has the effect of strengthening their partisan attachment even further. This finding helps clarify some of the debate over the endogenous versus exogenous nature of partisan strength by demonstrating roles for both. People may have a genetic predisposition toward developing strong attachment to a political party, but there is still room for this predisposition to be shaped by both shared and unique environments.

Another implication of our results is that we might better think of the acquisition of partisanship in two distinct parts, one strongly influenced by genes and the other strongly influenced by the environment. The literature has already conceptualized party identification as consisting of two components: a direction component, which indicates the specific party with which an individual identifies; and a strength component, which reflects the intensity of that identification (Converse 1976). Our results suggest that partisan intensity is heritable but partisan direction is not.

Our finding is consistent with the pattern of findings from studies of other social behaviors, such as religious beliefs and practices. Strength of attachment to a group, such as strength of partisanship or religiosity, has a strong heritable component, perhaps because of its relationship to fundamental processes in early human history. For example, we can imagine that the strength of one’s affiliation and association with groups was of more consequence when survival depended more directly on group cooperation. Evolutionary models of cooperation show that some environments favor group participation in the production of public goods, while other environments favor self-reliance because of the competition between contributors and free-riders (Fowler 2005; Hauert et al. 2007). These models predict heterogeneity in strategies, with some individuals joining groups and others trying to survive on their own. It is possible that this heterogeneity extends to several kinds of groups, including religious organizations and political parties.

However, the selection of a particular political party or a specific religious denomination probably has little impact on fitness. Instead, it is strongly correlated with environmental influences, such as demographic and socioeconomic factors (Alford, Funk, and Hibbing 2005; Eaves, Eysenck, and Martin 1989; Olson, Vernon, and Jang 2001; Bouchard et al. 2003). Thus, it is possible that genes more generally impact the inclination and strength of a predisposition toward political behavior, not its direction. This is consistent with the finding that strong identifiers who switched parties between elections are more likely to become strong identifiers of the new party than independents or weak identifiers (Katz 1979). There is little reason to think that there should be a genetic basis for specific group attachment, as the organization, principles, and practices of groups change over time. In other words, genes may contribute to the tendency toward group attachment but not necessarily the groups with which an individual will choose to associate.

Also of interest is conceptually linking the genetic tendency toward partisan attachment with other behaviors like political participation. Knowing that there is a heritable component to partisan attachment suggests that we must reexamine the theoretical mechanism by which partisan strength affects these other behaviors. For example, strong partisans are more likely to vote for a candidate of their party whom they do not like because of party loyalty and attitudes about split-ticket voting (Campbell, Gurin, and Miller 1954), and strength of partisanship is one of the best predictors of straight-ticket voting (Campbell et al. 1960; Brody, Brady, and Heitshusen 1994). Strength of partisanship is also related to political knowledge (Delli Carpini and Keeter 1996), the motivation to vote (Fowler 2006), and the formation of attitudes toward new candidates and public policy issues (Campbell et al. 1960; Converse and Markus 1979). Does a genetic predisposition toward partisan attachment also predispose a person to these other political behaviors, or does affiliation with a party itself mediate these other outcomes?

There are several limitations in our study. First, our sample is relatively small and self-selected. While we do not think that this has biased our analysis in any way, we must keep the limitations of the sample in mind when generalizing to the population at large. Excluding those younger than age eighteen from our sample means that we are not examining the role of heritability in the initial stages of partisan attachment and instead are focusing on the strength
of attachment once it has been formed. Second, our measure of partisan attachment, while frequently employed in the literature, has been subject to the critique that it does not adequately address the theoretical differences between partisans and nonpartisans; partisanship appears to be multidimensional and non-monotonic (Kamieniecki 1988; Petrocik 1974), and consequently, standard scales of partisanship strength may not adequately address how partisanship is correlated with political behavior. The 7-point scale used in our study may be best for measuring attitudes toward political parties in general because the main differences between subgroups of independent voters is in their orientation toward the symbols of political independence (Craig 1985). However, a scale such as that discussed in Greene (2002) or Weisberg and Hasecke (1999) may better measure the social-psychological identity aspect of partisan strength.

Third, the ACE model used in this study establishes that genes do play a role in partisan attachment, but it cannot expose the exact mechanism by which genes and the environment interact to produce the phenotype. To best understand partisan attachment, in the future, we must also examine these interaction effects. The significant contribution of the unshared environment in our study opens the door for an examination of factors that could serve as mediators for the gene-attachment mechanism. It seems likely that we are tapping into a general predisposition toward attachment, interest, or engagement and that environmental factors can play an important role in channeling that predisposition into behavior.

Although our use of the ACE model does not allow us to specify the contribution of any one gene in particular, the most likely candidate of genes identified to date is the DRD2 gene (Dawes and Fowler 2008). The A1 allele of this gene has been related to decreased dopamine signaling in the brain (Jonsson et al. 1999), and the consequences of altered dopamine receptors include social detachment (Breier et al. 1998; Farde, Gustavsson, and Jonsson 1997; Jonsson et al. 1999), social alienation (Hill et al. 1999), antisocial personality disorder (Ponce et al. 2003), and avoidant personality types (Blum et al. 1997). All four of these behaviors could reasonably be linked to a decreased tendency toward group attachment and affiliation, and studies testing for various alleles of this gene have shown an association between DRD2, partisan attachment, and voting (Dawes and Fowler 2008).

The resurgence of interest in biopolitics calls for a systematic approach to the study of political heritability to move toward a more explanatory basis for the research program (Fowler and Schreiber 2008), and scholars have made significant strides in this direction (Alford and Hibbing 2008; Medland and Hatemi forthcoming). The first step, like the one we take here, is to establish which political behaviors are heritable and which are not. Scientists in other fields continue to explore the mechanisms by which genetic variation affects general psychological and behavioral tendencies. The next step for political scientists is to apply this knowledge in the search for specific genes, neural and physiological mechanisms that may underlie political behaviors, as well as their potential evolutionary basis (McDermott, Fowler, and Smirnov 2008). In particular, it is extremely important to combine our new understanding of biology with our prior investigations of environmental causes of political behavior.

For example, Fowler, Baker, and Dawes (2008) originally showed that political participation is heritable, and this study was followed by a molecular study showing an association between MAOA, 5HTT, and voter turnout (Fowler and Dawes 2008). In particular, the association with 5HTT was moderated by church attendance, which ceased to be have a significant main effect on turnout. Since 5HTT has previously been associated with social behavior, this suggests that the development of a sense of community is more likely to be at the heart of past associations between attendance and turnout rather than the development of civic skills as some have suggested.

Alford, Funk, and Hibbing (2005) originally showed that ideological orientations were heritable, and then their study was followed by a molecular genetic study (Settle et al. 2008). The molecular study investigated an association between self-reported political ideology and the 7R variant of the dopamine receptor D4 gene (DRD4), which has previously been associated with novelty seeking. Settle et al. (2008) found that the number of friendships a person has in adolescence is significantly associated with liberal political ideology among those with DRD4-7R. Among those without the gene variant, there was no association. In other words, it was the interaction of a particular gene and a particular environment that mattered. Oxley et al. (2008) also followed up the original heritability study with research in the political physiology of ideological orientations, showing that people who support conservative policies are more likely to exhibit startle reflexes when presented with visual and auditory fear stimuli.

Thus, the finding that strength of party identification is heritable suggests that more work should be
done. Biology appears to play a role in partisanship, so the next step is to identify which genes and which mechanisms are interacting with which kinds of environments to affect partisan identification. Novel approaches such as the use of the ACE model and genetic association studies have the potential to revolutionize the way we interpret the political world. In other parts of political science, the study of institutions has improved our understanding of political outcomes because it helps us understand how legislatures, courts, and other bodies are constrained in their behavior. Similarly, the study of genes potentially promises better understanding of the constraints imposed on basic political psychology. Genes are the institutions of the human body, and we ignore them at our peril if we want to develop a full understanding of human psychology and political behavior.

Appendix

The ACE Model

More formally, the components of the ACE model are derived from known relationships between three observed statistics (Evans, Gillespie, and Milne 2002):

\[ \sigma^2_p = \sigma^2_A + \sigma^2_C + \sigma^2_E \]
\[ COV_{MZ} = \sigma^2_A + \sigma^2_C \]
\[ COV_{DZ} = \frac{1}{2}\sigma^2_A + \sigma^2_C. \]

In this equation, \( \sigma^2_p \) is the observed phenotypic variance (the same for MZ and DZ twins), \( COV_{MZ} \) and \( COV_{DZ} \) are the observed covariances between MZ and DZ twins, and \( \sigma^2_A, \sigma^2_C, \) and \( \sigma^2_E \) are the variance components for genes, common environment, and unshared environment, respectively. These relationships yield three equations and three unknowns, so it is possible to infer the unobserved portions of variance attributable to each factor.

The known relationship between the phenotypic variance and the variances of A, C, and E, as well as the relationship between MZ and DZ twin covariance and the variances of A and C, allows for var(A), var(C), and var(E) to be estimated.

The structural equation specification of our model is as follows:

\[
\begin{align*}
\text{VAR(Conscience)} & = a^2 + c^2 + e^2 \\
\text{COV(MZ)} & = a^2 + c^2 \\
\text{COV(DZ)} & = 0.5a^2 + c^2.
\end{align*}
\]

This is a system of three equations and three unknowns, so it is identifiable. The parameter estimates are solved for by

\[
\begin{pmatrix}
\sigma^2_p \\
\sigma^2_A \\
\sigma^2_C \\
\sigma^2_E
\end{pmatrix} = \begin{pmatrix}
1 & 0 & 0 \\
0.5 & 1 & 0 \\
0.5 & 0.5 & 1
\end{pmatrix}^{-1}
\begin{pmatrix}
\text{COV}_{MZ} \\
\text{COV}_{DZ} \\
\text{COV}_{Partisan} \end{pmatrix}.
\]

Heritability, or the proportion of the variance in partisanship explained by genetic factors, can be estimated as \( a^2/(a^2 + c^2 + e^2) \). We use the software package MX to estimate this structural equations model (Neale et al. 2006). Since our dependent variable is (ordered) categorical, the model assumes that latent distribution of partisanship is normal even though we only observe four distinct values. Therefore, in addition to estimating \( a^2, c^2, \) and \( e^2 \), the model estimates three thresholds associated with the underlying distribution.

Note: The ACE model consists of additive genetic factors (A), shared or common environmental factors (C), and unshared environmental factors (E).

Notes

1. We employ a version of the question that reads, “Generally speaking, do you usually think of yourself as a Republican, a Democrat, or what?” Respondents are forced to select one of seven categories, consisting of strong Democrat; Democrat; independent, but closer to Democrats; independent, but independent, but closer to Republicans; Republican; or strong Republican. The standard Michigan/National Election Studies (NES) question leads to the same 7-point scale but derives the scale from a two-question series.

2. We measured liberalism and conservatism with the answer to the following question: “We hear a lot of talk these days about liberals and conservatives. Here is a scale on which the political views that people might hold are arranged from extremely liberal to extremely conservative. Where would you place yourself on this scale?” Response options are very liberal, liberal, slightly liberal, moderate, slightly conservative, conservative, and very conservative. Similar to partisanship intensity, ideological intensity was a version of the ideological scale, folding it over to use moderate as the minimum value and very conservative and very liberal as the maximum values.

3. The ACE model (additive genetic factors [A], shared or common environmental factors [C], and unshared environmental factors [E]) assumes that genetic effects are only additive; therefore there are no dominance effects. However, a dizygotic (DZ) correlation that is less than half of the monozygotic (MZ) correlation is generally considered evidence of dominance. While this is the case for our point estimates, the confidence interval for the DZ correlations is fairly wide due to our small sample size. To formally test the null hypothesis that the DZ correlation is at least as large as half the MZ correlation, we bootstrapped one thousand MZ and DZ correlation coefficients. We failed to reject the null hypothesis that DZ \( \geq 0.5 \times MZ \) (\( p = .71 \)), suggesting that the ACE model is the correct one to use for estimation.

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Do Genes Contribute to the “Gender Gap”?

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The nature and mechanisms underlying the differences in political preferences between men and women continues to be debated with little consideration for the biology of sex. Genetic influences on social and political attitudes have been reported for each sex independently, yet neither the magnitude nor sources of genetic influences have been explored for significant differences between males and females. In a large sample of adult twins, respondents indicated their attitudes on contemporary social and political items. Finding significant differences in the magnitude of genetic, social, and environmental variance for political preferences, and the potential for different genes in males and females to influence these phenotypes, we provide evidence that sex modulates the effects of genetic and environmental differences on political preferences.

It is undeniable that human males and females tend to behave differently. It is widely accepted across disciplines that both physiology and the environment make profound contributions to behavioral differences, and there is considerable evidence that these differences are in part due to genetic variation, which may differ between the sexes (Eaves, Eysenck, and Martin 1989). While it is widely held that humans have evolved to possess the ability to transcend their biology (Lumsden and Wilson 1981) and enjoy the capacity to do what they choose, their preferences are biologically influenced. However, relatively few studies have explored the role of endogenous differences on political preferences (for exceptions see Alford, Funk, and Hibbing 2005; Fowler, Baker, and Dawes 2008; Hatemi et al. 2007; Hatemi et al. N.d.). Those which exist provide little clarity on whether or how these mechanisms differ between the sexes.

In the biological sciences, sex is a term related to fundamental reproductive ability; simply whether one is male or female. In political research most studies combine gender and sex and primarily view gender as a social construct. Thus, most examinations of sex differences focus on social conditions, such as gender roles. Political scientists typically assume that sex differences reflect differences in social identity, which are primarily due to differences in socialization (Chodorow 1995). As such, the vast majority of political studies convolute the independent effects of social forces and anatomical sex.

While the concept of sex is explicit, the sources of inherent differences between males and females are not. In addition to hormonal and chromosomal differences (females have two X chromosomes while males inherit one X and one Y chromosome), behavior differences may arise from variation in the timing or magnitude of gene action, at times triggered by environmental stimuli. Differences observed between males and females are likely due in part to a combination of these effects. For example, females are two to three times more likely to suffer from migraines and differences in hormones such as estrogen explain some of this variation (Ligthart et al. 2006). In some instances only with certain environmental factors, such as stress, are sex differences in hormonal and genetic influences on migraines present (Brandes 2006). Thus, even for biomedical traits, by no means is the environment unimportant. It very well may be that for social traits the environment is the most salient influence, as social behaviors take place in a social context.

While the topic of sex differences is highly charged in politics and the media, a more complete understanding of the interplay between biology and culture in the convoluted developmental pathway from DNA to complex social behavior is a necessary step towards resolving the biological and social origins of sex differences. Merely demonstrating that there are different genetic pathways to behavior between the sexes and that this may explain some of the manifest
differences in behavior in no way implies that the role of gender defined socially cannot in turn modulate the expression of otherwise similar biological influences on behavior. Ultimately “gender roles” may modulate the expression of “genes” as a function of social context. However, whether stimulated by different environmental pressures or different hormonal and neurochemical pathways, the same gene or group of genes may exert different influences on political preferences for each sex. Genetic influence is no more deterministic than social influence, and in order to better explain the source of differences in political preferences between men and women, both genetic and social influences that affect political behavior are considered.

**Gender and Politics: the Traditional Discourse**

Most literature on political behavior assumes sex differences are socialized and remains silent about genetic or biological influences (Carroll 1988; Chodorow 1978, 1995). The main focus in the literature points toward the greater attraction of the Democratic Party to women than men and the greater support women give to liberal issue positions (Cook and Wilcox 1991). Explanations of this “gender gap” focused on policy preferences such as abortion, equal rights, violence, foreign policy, minority rights, equity, and criminal justice (Manza and Brooks 1998; Norrander 1999; Smith 1984; Togeby 1994). Men were more likely to support the use of force or violence whereas women were less willing to use force in foreign policy and less apt to support war (Carroll 1988). It was argued that women were more concerned with crime as they see themselves as more vulnerable, were more in favor of stronger punitive measures for rape and domestic violence, and were more likely to convict those accused of violent crimes. However, women were also more supportive of prevention policies, gun control measures, and treatment for criminal behavior (Applegate, Cullen, and Fisher 2002; Hurwitz and Smither 1998; Lambert 2003; Lerner and Keltner 2000). On public protection or “compassion” issues such as education, welfare, minorities, the poor, sick, elderly, or unemployed, women were substantially more likely to support liberal positions (Shapiro and Mahajan 1986; Trevor 1999).

A considerable portion of the research on sex differences for political preferences has been based on Gilligan’s (1982) theoretical premise: women and men approach values from different perspectives. Women’s “psychology” is based on an ethic of care versus men’s ethic of rights or justice (e.g., women are caretakers and nurturers whereas men are not). While these differences are believed to be socially constructed, even Gilligan acknowledges that “Clearly these differences arise in a social context where factors of social status and power combine with reproductive biology to shape the experience of males and females” (1982, 2).

Neuropsychological factors, physiological differences, genetic influences, or differences in the biological environment (e.g., hormones introduced in utero) largely remain unaddressed as either directly influencing political preferences, or providing a source for the differences in “psychology” (Bussey and Bandura 1999; Correll 2004). This is inconsistent with scholarship outside of political science. For example, Money and Ehrhardt (1972) reported that females who were treated with androgens when still in the womb (normally only present in male fetuses) displayed similar behavior patterns as males throughout their lives. The ‘treated’ females were more vigorously active, self-assertive and competitive, preferred outdoor activities, played with “male” toys, and took greater interest in hierarchical positioning. In contrast to their peer group, once reaching adulthood, romance and marriage took second place to career advancement and personal achievement.

With few exceptions, the literature examining sex differences on political behaviors have taken little note of these and similar findings and seldom addressed the importance of biological conditions related to sex. Rather, the focus remained on social, cultural, personal, and economic conditions (Howell and Day 2000). However, an important leap in the political science literature opened the door to incorporate physiological, biological, and evolutionary theses. Building upon the work of Eaves (1977), and Martin et al, (1986), Alford, Funk, and Hibbing (2005) found substantial genetic sources of variation for a wide array of political attitudes. However, while implying differences in genetic variation may exist between the sexes, the study did not test for significant differences in the magnitude of genetic influence or explore the potential for different genes to account for the genetic influence on political attitudes between men and women.

**An Evolutionary Explanation for Sex Differences in Political Behavior**

Interdisciplinary scholarship offers alternative theories to explain sex differences on modern day social roles by including inherent sources of behavior (Campbell...
According to Pratto (1996), human abilities to mitigate the problems of today did not evolve as an adaptation to today’s environment, but are modifications of previously evolved skills from a prevailing environment that occurred over the last 2 million years. This evolutionary model has been used to explain the pathways of a wide variety of contemporary behaviors including: the nuclear family, antipathy for strangers, self-motivated learning, exploratory behavior, territoriality, fear, ethical behavior, authority acceptance, cooperation, play, group affiliation, aggression, competitiveness, and hierarchical groups (e.g., Fehr and Fischbacher 2003; Fehr and Gachter 2000).

Accordingly, evolutionary forces in certain domains differed between males and females in prehistoric societies, and these differing forces have led to innate processes which contribute to behavioral differences between the sexes. Because males and females shared most adaptive problems in primitive societies, the majority of human behavioral and physical adaptations are sexually anamorphic (e.g., all healthy humans have a prefrontal cortex, sleep, etc.). However, adaptive problems which were continually disparate between the sexes, such as child bearing, are expected to have led to sexually differentiated specializations and preferences (Buss 1999; Tooby and Cosmides 1990).

A core evolutionary pressure for women has been childbirth and childcare. Thus, it is argued that women have evolved greater sensitivity than men to those in need, a stronger focus on mate selection, and a more acute empathetic disposition (Gangestad and Simpson 2000; Gangestad and Thornhill 1997). In similar fashion, men have evolved psychological mechanisms that place more value on social status, increased competitiveness, and risk taking because the limiting resource of males’ reproductive success had to do with the availability of fertile women, and because women had a propensity to choose males who are of higher status (Low 2005; McBurney, Zapp, and Streeter 2005; McDermott, Fowler, and Smirnov 2008). Moreover, the need for cooperative alliances and regard for status and hierarchy in hunting and warfare has heightened the desire for males to establish group alliances (Huberman, Loch, and önculer 2004; Wrangham et al. 1999). According to Pratto (1996), males’ greater interest in political activities is a reflection of these evolved preferences with specific concern for the establishment and maintenance of social dominance and status.

Contemporary observations in early childhood reinforce these theories. Cross cultural studies of children found that the largest behavior differences between the sexes occurs in children between three and six years old, providing evidence that complex social behaviors are inherited as well as learned (Maccoby and Jacklin 1974; Money 2002). Males develop stronger aggressiveness, outwardly directed predispositions, greater self assertiveness, contentious tendencies, adventurousness, and the need for individual achievement early on. Whereas females develop a more actively sympathetic disposition, inward directed demeanor, maternal impulses, heightened domestic, social, and personal concerns, as well as more interest in art, linguistics, and literature at a very early age (Cosmides and Tooby 2000; Geary 1998). Political scientists typically attribute these differences to socialization. However, if socialization were the determinant, the gap should widen with age or have a more acute effect when children are fully aware of and immersed in their social environment, and not in the three-to-six-years-old range.

**The Heuristic Value of Genetics**

Evolutionary psychology strives to explain inherent differences in behavior by reference to the long history of evolutionary adaptation. However, empirical tests of this theory require approaches different from traditional social science designs (e.g., Alford, Funk, and Hibbing 2005). The greater complexity and heterogeneity of modern society compared to the Pleistocene era (e.g., dating websites, abortion clinics, defense of marriage acts, condoms, suicide bombers, etc.), presents a problem for the use of evolutionary explanations on modern political preferences. Specifically, the evolutionary psychology focus on universals offers limited scope for empirical exploration and it is not readily apparent how to test if “yesterday’s” biology is influencing “today’s” political behaviors differently for each sex (Kanazawa 2001; Oota et al. 2005).

However, there are clues that the genetic study of individual differences may provide some insight into evolutionary history and modern behavior because natural selection modifies the expression of genetic and environmental differences differently in response to different adaptive challenges (Fisher 1930; Lerner 1954; Mather 1966). While the relationship between “heritability” and “selection” is unresolved...
in population genetics, human or otherwise, if sex really has been responsible for major differences in evolution we expect the genetic system of today to bear some footprints of natural selection.

Although there are no studies we know that empirically examine the potential for different genetic pathways on political attitudes between men and women, or differentiate the variance for political traits between biological sex and social forces, behavior geneticists have been addressing similar issues for some time (Martin et al. 1986). Behavioral genetic techniques developed in an attempt to understand why individuals in a population differ from one another and include both genetic and environmental influences (Medland and Hatemi forthcoming). These analyses are concerned with accounting for variation around a population mean, but estimate effects of covariates on means. Instead of testing to see if opinions on the death penalty are genetic, one would test if individual differences in opinions on the death penalty are influenced by genes. Variance is partitioned into estimates of genetic, social, and unique environmental influences, thereby providing a means to explore whether genetic and social influences on certain behaviors are more or less pronounced in either sex and if the source of genetic influence differs between males and females.

The potential for differences in genetic influence between the sexes have been explored for many social traits (e.g., Eaves, Eysenck, and Martin 1989), however heritable differences between the sexes on political preferences have not. The trait most commonly associated with political preferences in the psychology and genetics literature is personality. There is overwhelming evidence, in numerous large-scale studies across cultures and continents, that personality is substantially influenced by genes (e.g., Bouchard 1997; Jang, Livesley, and Vernon 1996). However, the evidence is mixed regarding different genetic influences by sex. Finkel and McGue (1997) found no evidence that different sets of genes influences personality in each sex, nor did they find that the same genes influence each sex differently for the higher order dimensions of Emotionality. However for dimensions of Alienation, Control, and Absorption the same genes contributed differently to the total variance for males and females. In one of the largest studies on personality, Eaves et al. (1999) found that the magnitude of genetic influence differed for Extraversion, but not Neuroticism, and provided confirmatory evidence that while the magnitude of genetic influence differed, the same genes were influencing personality in males and females.

Variance components analyses are highly informative in identifying genetic influence, but they cannot identify exactly which genes are responsible for the genetic influence. Genome wide and allelic analyses identify regions of the genome seemingly correlated with a given trait, and how a specific gene influences a trait, respectively. Numerous candidate genes have been identified in the study of personal temperament and social behaviors and offer a starting point for exploring which genes could be related to political phenotypes. For example, the androgen genes ADRA2 and PNMT have been found to influence harm avoidance and cooperativeness (Deupree et al. 2006); dopamine receptors DRD3, DRD4, COMT, and MAOa are correlated with risk-taking behavior, inattention, impulsivity, spirituality, self-transcendence, high neuroticism, low extraversion, anxiety, reward dependence, aggression, antisocial behavior, and voter turnout (Chen et al. 1999; Fowler and Dawes 2008; Harpending and Cochran 2002). In the glutamate and nicotinic systems, GRIN1 and CHRNA4 have been shown to be related to cooperativeness; and in the serotonin system, 5-HT2a, SLC6A4, 5-HTTLPR, and TPH are related to impulsivity, harm avoidance, assertive behavior, anxiety, aggression, creative expression, and depression (Caspi et al. 2003; Hariri et al. 2002).

The most profound genetic sex differences exist in the vasopressin and oxytocin systems. The genes OXTR (oxytocin), and AVPR1a (vasopressin) are related to affiliative behaviors, mating, pair-bonding, parenting, and trust. These neuropeptides play an important role in the regulation of affiliation, social attachment, and social recognition. While there is very limited exploration of vasopressin and oxytocin in human social behavior (for an exception see Prichard et al. 2007 and Gordon et al. 2008), the investigation of the neurobiological pathways underlying complex social behaviors are widely explored in animal studies (Williams et al. 1994). Repeated experiments find oxytocin promotes pair bonding and mating in female prairie voles, but has no significant effect on males (Young et al. 2001). However, in males, but not females, higher vasopressin uptake increases pair bonding and has been shown to be associated with increased investment in childcare (Hammock and Young 2005; Pitkow et al. 2001; Winslow et al. 1993). Humans and voles share certain neurochemical pathways, and the aforementioned studies offer a basis to explore the potential for different genes in human males and females to influence similar behaviors.

Rather than ignore the potential for endogenous influences that differ between the sexes, we challenge
the assumption that biological pathways to political behaviors are the same for both sexes. Males and females are biologically different and should interact with and respond to the environment differently. The relationship between the genetics of “now” and what may or may not have happened historically remains ambiguous. However, if human’s current genetic dispositions are the result of their evolutionary past, we expect significant differences to be present in the magnitude of genetic influence between men and women on many political issues, similar to studies which explored personality. More importantly, for political preferences expected to be modern representations of constructs that were under intense selection pressures which differed between the sexes, it is likely that different genes are responsible for differing genetic variation between the sexes. This expectation is strengthened by the recent findings on the vastly different role vasopressin has in males and oxytocin in females for pair bonding, mating, and childcare. Therefore, we expect political preferences which affect reproductive success and mating to be influenced by different genes in males and females.

Variance components estimates provide a means to partition the relative importance of individual variation for each trait. However, any trait with a significant genetic influence does not mean that there is “a” gene for that trait or that the trait is an item under intense evolutionary selection. One school of thought suggests that traits strongly related to fitness should be less heritable because genetic differences are removed by selection. Extensive published literature reviews give only partial support for this view. It is also suggested that nonadditive genetic effects will predominate for so called “fitness traits” (Keller 2007). This view has some empirical support in other species but is difficult to demonstrate in humans due to the absence of controlled mating. Among the many mechanisms that have been invoked to account for the persistence of genetic polymorphisms are: adaptive interdependence of traits; adaptive significance of allele changes with shifting environment; frequency dependent selection; disruptive selection; and gene by environment interaction. Any or all of the above may prevail in a given historical context. While any attempt to relate the presence or absence of genetic variation to adaptive significance of particular traits should be met with caution, it is clear that strong directional selection will, if anything, eradicate genetic differences and not enhance them.

Methods

Behavior genetic population samples are often centered on twins, and the family members and peers of twins. While intentionally sampling nonindependent individuals violates statistical assumptions of independence, explicitly modeling the degree of relatedness for the individuals in the sample corrects for these violations. The power of twin samples is based on the knowledge that monozygotic twins are genetically identical, whereas dizygotic twins arise from two different eggs fertilized by different sperm. Using twin pairs raised by the same parents in the same environment provides a natural experiment controlling for the familial environment (see Medland and Hatemi forthcoming).

Using structural equation modeling (SEM), variance can be decomposed into additive genetic (A), common environment (C), and unique environment (E) and tested for sex differences. “Additive genetic” is the combined genetic effect of all genes (inherited traits). “Common environment” includes that which is shared by family members and includes cultural norms and familial socialization. “Unique environment” refers to all environmental stimuli that are unique to the individual (i.e., personal experience). Without retest measures unique environment includes measurement error.

Sample

The Virginia 30,000 Health and Life Style Questionnaire (VA30K) consist of the kinships of twins aged 18–88 years. Here we limit our analyses to the 14,763 twins, including opposite sex twin pairs. The VA30K includes assessments of political and social attitudes, sociodemographics, personality traits, and life events. Completed questionnaires were received from 70% of twins invited to participate (for details on sampling technique and demographics see Lake et al. 2000).

The political attitudes assessment consisted of a 28-item contemporary attitudes inventory based on the Wilson-Patterson (1968) format. Three items (Democrats, Republicans, Liberals) were excluded as a more appropriate item, Party Identification, was available. Church attendance (Religiosity) was also included (see Table 1 for the list of 27 items used in the analyses). Previous analyses of the differences in attitudes between sexes for the VA30K are consistent to findings in the general political behavior scholarship. Attitudes on the Death Penalty, Living Together, Abortion, Nuclear Power, Capitalism, the Draft, Republicans, Property Tax, and Military Drill were supported more by males, whereas attitudes on Gay
Table 1  Quantitative Sex Limitation Variance Components (95% confidence intervals)1

<table>
<thead>
<tr>
<th>Modela</th>
<th>Parameters Estimates</th>
<th>Parameter Estimates</th>
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<tbody>
<tr>
<td></td>
<td>a²</td>
<td>c²</td>
</tr>
<tr>
<td>Live Together</td>
<td>ACEbc</td>
<td>.51 (.41–.68)</td>
</tr>
<tr>
<td>Busing</td>
<td>ACEb</td>
<td>.31 (.16–.31)</td>
</tr>
<tr>
<td>Divorce</td>
<td>ACEbc</td>
<td>.25 (.16–.29)</td>
</tr>
<tr>
<td>School Prayer</td>
<td>ACEb</td>
<td>.32 (.16–.48)</td>
</tr>
<tr>
<td>Capitalism</td>
<td>AEbc</td>
<td>.47 (.43–.52)</td>
</tr>
<tr>
<td>Abortion</td>
<td>ACEbc</td>
<td>.26 (.12–.41)</td>
</tr>
<tr>
<td>Foreign Aid</td>
<td>ACEb</td>
<td>.40 (.29–.45)</td>
</tr>
<tr>
<td>Women’s Lib</td>
<td>ACEbc</td>
<td>.34 (.18–.49)</td>
</tr>
<tr>
<td>Religiosity</td>
<td>ACEbc</td>
<td>.48 (.32–.67)</td>
</tr>
<tr>
<td>Censorship</td>
<td>AEbcd</td>
<td>.38 (.33–.42)</td>
</tr>
<tr>
<td>Death Penalty</td>
<td>ACEbd</td>
<td>.35 (.22–.48)</td>
</tr>
<tr>
<td>Pacifism</td>
<td>AEbd</td>
<td>.31 (.27–.35)</td>
</tr>
<tr>
<td>Segregation</td>
<td>AEbcd</td>
<td>.37 (.32–.37)</td>
</tr>
<tr>
<td>Draft</td>
<td>AEbd</td>
<td>.37 (.32–.41)</td>
</tr>
<tr>
<td>X-Rated</td>
<td>AEbcd</td>
<td>.51 (.47–.56)</td>
</tr>
<tr>
<td>Modern Art</td>
<td>AEbcd</td>
<td>.40 (.36–.43)</td>
</tr>
<tr>
<td>Moral Majority</td>
<td>AEbd</td>
<td>.42 (.38–.47)</td>
</tr>
<tr>
<td>Property Tax</td>
<td>AEbd</td>
<td>.42 (.41–.46)</td>
</tr>
<tr>
<td>Socialism</td>
<td>AEbd</td>
<td>.38 (.34–.38)</td>
</tr>
<tr>
<td>Immigration</td>
<td>AEbd</td>
<td>.46 (.46–.49)</td>
</tr>
<tr>
<td>Party ID</td>
<td>CEbcd</td>
<td>.81 (.78–.84)</td>
</tr>
<tr>
<td>Astrology</td>
<td>AEb</td>
<td>.47 (.43–.47)</td>
</tr>
<tr>
<td>Gay Rights</td>
<td>ACEbd</td>
<td>.34 (.24–.45)</td>
</tr>
<tr>
<td>Military Drill</td>
<td>AEbd</td>
<td>.36 (.31–.40)</td>
</tr>
<tr>
<td>Unions</td>
<td>AEb</td>
<td>.41 (.36–.46)</td>
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<tr>
<td>Fed Housing</td>
<td>AEb</td>
<td>.34 (.36–.46)</td>
</tr>
<tr>
<td>Nuclear Power</td>
<td>AEbd</td>
<td>.34 (.30–.39)</td>
</tr>
</tbody>
</table>

Notes: (a) Only best fitting models shown (thresholds corrected for age). ACE represents a model in which all three variance components are significant; CE represents a model in which common and unique environmental influences are significant; AE represents a model in which genes and environment are significant; a², c², and e² represent additive genetic, common environmental, and unique environmental influence respectively; Δ represents the difference between males and females (b) equated thresholds for MZ and DZ pairs (c) equated thresholds for males and females (d) equated variance components for males and females.3

1 Portions of the quantitative sex limitation analyses were initially included as part of a larger dissertation project (see Hatemi 2007).

Rights, Federal Housing, Liberals, Democrats, Busing, Censorship, Modern Art, Unions, Divorce, School Prayer, Socialism, Moral Majority, and Pacifism were supported more by females (Eaves et al. 1999).

The Wilson-Patterson (WP) inventory is administered by presenting subjects with a stimulus phrase such as “Abortion” and eliciting an “agree,” “disagree,” or “uncertain” response. As with any measure, it can be argued that individual WP items may not be ideal measures to test biological, evolutionary, or even political hypotheses. From an evolutionary perspective, items which affect reproductive success (fitness) are the most likely to be influenced by different genes, and many of the attitudes do not appear to be items on which divergent selection pressures would have taken place between males and females. However, while not all items are ideal, the index is constructed of a wide variety of items based on five underlying factors: sex and mating, force and militarism, economics and property, politics and group affiliation, and religion (see Eaves et al. 1999). Items related to mating and sex (Living Together, X-rated, Divorce, Abortion, Gay Rights) are appropriate for fitness hypotheses, while the items that are not provide the opportunity to falsify the hypotheses.

**Analyses**

Employing a maximum likelihood approach minimizes the discrepancies between observed and predicted
correlation between the latent common environment factors, on average 50% of their discriminating genes. Correlations between monozygotic twins (MZ), who share 100% of their genes and 0.5 for dizygotic twins (DZ), including opposite sex pairs, who share on average 50% of their discriminating genes. Correlations between the latent additive genetic factors (A) are 1.0 for monozygotic twins (MZ) who share 100% of their genes and 0.5 for dizygotic twins (DZ), including opposite sex pairs, who share on average 50% of their discriminating genes. Correlations between the latent common environment factors (C) were 1.0 in both MZ and DZ twin pairs, as both twins were raised by the same parents, in the same environment. The latent factor for the unique environment (E) is uncorrelated. The difference between the genetic correlation of MZ and DZ twins provides the leverage for estimating the genetic contribution to individual differences.

Common in behavior genetic analyses, observed frequencies for the ordinal political attitude measures were calculated and incorporated into a threshold model that assumes that each variable has an underlying normal distribution of liability. In order to correct for age effects, we used a full-information approach fitting the structural models to the raw data. Thresholds are expressed as z values which discriminate between categories that correspond to the frequency of the political attitudes. Thresholds were tested for similarity across sex and twin zygosity groups and corrected for age effects.

In order to investigate the presence and nature of genetic differences in the variance between the sexes, a sex limitation parameter is included (noted as A\textsubscript{Male} in Figure 1). The general approach to fitting a sex-limitation model to twin data is described by Heath, Jardine, and Martin (1989). The full sex-limitation model permits quantitative sex differences, which assumes the same sources of variation for males and females (same genes), but allows for differences in the magnitude of genetic and environmental effects for each trait, and qualitative sex differences, where the model allows for different genes to influence the behavior for each sex. Opposite-sex twins are necessary in this process as they share the same familial environment, but experience it as members of the opposite sex, thereby providing a natural experiment to examine sex differences. The correlation between genetic effects in unlike sex pairs will be a function of the extent to which the same genes affect both males and females. In the extreme case, where completely different genes are expressed in males and females, the genetic correlation is expected to be zero.

The genetic correlation between opposite-sex twin pairs (OS) may be less than 0.5 if there are qualitative sex specific genetic effects. If dropping the sex specific genetic effect does not significantly alter the fit of the model (if the difference in fit between the models is not significant), this indicates that although the genetic effects may differ in their magnitude between males and females, the influence is from the same genetic source regardless of sex. Alternatively, if the model which includes the separate sex specific “A” parameter results in a better fitting model, the results indicate that some, but not necessarily all, the genetic influences on

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**Figure 1  Sex Limitation ACE Model**

![Sex Limitation ACE Model Diagram](image)

Notes: This path diagram is an explicit representation of the model where the expected covariance between two variables is computed by multiplying together all the coefficients in a chain, and then summing over all possible chains. For example, the variance of a phenotypic trait for a MZ twin is calculated as: \((a \times 1 \times a) + (c \times 1 \times c) + (e \times 1 \times e) = a^2 + c^2 + e^2\). “A” is additive genetic \(2^\text{MHz-MDZ}\), “C” is common environment \((2^\text{MDZ-MDZ})\) and “E” is unique environment \((1 - r_{MZ})\). As indicated by the sex specific subscript for the path coefficients \(A_{\text{Male}}\) the model allows a, c, and e, to be estimated separately for each sex. The sex specific parameter can be placed on either one of the sexes (but only one, males in this example) and provides the same results.
the trait are specific to one sex (i.e., a qualitative sex difference).

Model Fitting

Model fitting is used as a method of hypothesis testing to determine the relative importance of genetic and environmental influence. The full model, which includes three sources of variance (ACE), (a fourth sex-specific source in the qualitative model), was tested against progressively reduced models by comparing the fit of the model in which the component (A, C or E) is freely estimated to the fit of the model in which either the A or C parameter has been set to zero. The difference in model fits, assuming that the models are nested, is asymptotically distributed as a chi-square distribution with the degrees of freedom equal to the difference in the number of estimated parameters between the two models. For example, to compare the difference in fits between an ACE model (in which the covariation is due to the combination of additive genetic, common environmental and unique environmental effects) and a CE model (in which the covariation is due to common environmental and environmental effects only) against a \( \chi^2 \) distribution, the A parameter is dropped from the full model and model fit is compared. Quantitative sex differences (different magnitude of genetic influence) were tested by comparing the fit of nested models that equated the separate path coefficients for males and females to the full model. Qualitative sex differences (different genes) were assessed by comparing the fit of Model 1, in which correlations between genetic or environmental latent factors were estimated to the fit of Model 2 in which these correlations were fixed to 0.5 and 1.0, respectively.

Model Limitations

Consistent with all empirical analyses, there are assumptions and limitations built into sex-limitation twin analyses. Univariate estimates do not account for the possibility of gene by environment interaction (GxE) or gene-environment covariation (rGE). More advanced twin models that include the effects of genes and environments can provide information on how genetic influences are expressed through and by environmental conditions (for more on GxE and rGE, see Medland and Hatemi forthcoming). Importantly, as with all population-based samples, results are population specific, and generalizations should be made only after the results are replicated on different populations. Our sample is from a U.S. population in the 1980s. Defined sex roles are present in many aspects of U.S. culture, and populations from other regions of the United States in different time periods are necessary for replication.

Sex-limitation models assume that opposite-sex twin pairs are influenced by the common environment to the same extent as same-sex twin pairs for the trait under analysis. While it is implausible that parents’ would raise their opposite-sex offspring in a dissimilar manner for political behaviors, it is also likely that boys grow up as “future men” and girls as “future women.” Differences in peer groups, social activities, and other social processes may influence environmental similarity. However, any dissimilarity would have to affect the trait of interest. While there is no indication that potential sex differences in rearing exist for political behaviors (e.g., parents raising daughters to be pro-choice and sons pro-life, etc.), a more extended family model including parents would provide more insight.

It is also assumed that any differences in the similarity of environments between DZ and MZ co-twin pairs do not affect the trait under examination. Empirical support for this assumption and its implications have been widely discussed, and numerous methods have been employed to validate the assumption (for a review see Kendler and Gardner 1998). These include testing twin environmental similarity by controlling for perceived zygosity versus true zygosity (rated by self-reports, parents, friends, teachers, etc.), modeling self-report and parental report of parental treatment, including ratings of physical similarity, as well as specific environmental measures. There are extremely few traits in which unequal environments have an influence and none so far reported for political preferences. While unequal environments may be found for dressing alike as toddlers, it has no influence on co-twin similarity of political behaviors later in life. Rather, what scholarship is available provides evidence that MZ and DZ co-twin pairs are influenced by the common environment to the same extent as same-sex twin pairs for the trait under analysis. While it is implausible that parents’ would raise their opposite-sex offspring in a dissimilar manner for political behaviors, it is also likely that boys grow up as “future men” and girls as “future women.” Differences in peer groups, social activities, and other social processes may influence environmental similarity. However, any dissimilarity would have to affect the trait of interest. While there is no indication that potential sex differences in rearing exist for political behaviors (e.g., parents raising daughters to be pro-choice and sons pro-life, etc.), a more extended family model including parents would provide more insight.
found a new home in political science (e.g., Charney 2008; Suhay, Kalmoe, and McDermott 2007). This is not unsurprising, as political scientists are only now becoming more familiar with the genetics methodology and literature. However, the focus of these challenges are to a large degree limited to those committed to a theory that only social forces influence social behaviors. These include philosophical objections to applying scientific methods to the analysis of political behavior (e.g., „They’re trying to make political science into a science, when really it’s not”; Evan Charney quoted in Cohen 2008). Often, critics choose not to address the most recent and relevant literature on familial modeling, including models which provide estimates of twin-specific environmental variance (e.g., Eaves et al. 1999; Eaves and Hatemi 2008; Hatemi et al. 2007b). Nor have they addressed DNA based techniques that validate the use of twin designs. Visscher et al. (2006) used non-twin sibling’s exact genetic similarities (DNA) in a variance components model, thereby removing the assumption of equal environments. Using this method Visscher et al. (2006) reported genetic estimates for height similar to those reported using twin models. While the method has not yet been used for political attitudes, the method provides strong evidence that MZ/DZ models are robust. The method is free from the criticisms assigned to twin models and is available for critics to falsify the equal environment assumption on a trait-level basis. This is important as none of the critiques (e.g., Beckwith and Morris 2008; Charney 2008; Suhay, Kalmoe, and McDermott 2007) provide any empirical evidence of unequal environmental bias on political traits or attempt to empirically test the potential for unequal environments.

Disregarding the overwhelming evidence that necessitates the inclusion of inherent influences on human behaviors, there are also those who believe that the exploration of inherent mechanisms which influence social behaviors is equivalent to the sexist, xenophobic, and racist eugenics movements of the last century. Beckwith maintains that exploring genetic influences for social traits can only lead to “absolving of society of any responsibility for its inequities” (1993, 332). However, this moralistic argument is highly selective, and ignores important genetic discoveries that have lead to increased social tolerance, including the now discredited refrigerator mother hypothesis, which blamed autism on mother’s emotional frigidity (see Bettelheim 1967). Rather, twin studies are credited with changing the discourse of the medical community and public image of mothers with autistic children, “The heritability of autism has been one of the most important changes in our conception of the condition since the first pioneering descriptions” (Hill and Frith 2003, 282).

**Results**

The magnitude of genetic variance differed between males and females for most traits, providing strong evidence confirming the hypothesis that social forces cannot account for all the differences in individual variation within and between males and females (see Table 1). Rather, there is substantial evidence that for many political preferences genes are having markedly different influences on behavior in males and females. In 9 of the 27 items significant sex differences in the magnitude of genetic variance were present: Living Together (.51), Busing (.19), Divorce (.17), School Prayer (.15), Capitalism (.14), Abortion (.12), Foreign Aid (.09), Women’s Lib (.03), and Church Attendance (.01).

While the difference in the magnitude of genetic influence found between the sexes on political attitudes confirms the need to address sex when exploring genetic sources of political preferences, interpreting the quantitative results to address specific hypotheses based upon evolutionary foundations are not as straightforward. The magnitude of genetic variance was not necessarily greater or lesser in females for political items that address those in need, evoke emotional responses, build stronger domestic support systems or tap into an “ethic of care.” Nor was there less or more genetic influence on attitudes regarding the use of force, status, or group alliances in males or that men have more genetic variance for most political items. The findings are trait specific, akin to sex differences on personality, and offer little recognizable pattern.

However, the remarkable difference in the genetic estimates of Living Together solicits further discussion. In the full model (all sources of variation included), genetic influence on Living Together accounted for 0% of the variance in males, but over 50% in females. Though not intended to identify different genetic

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1Highly adaptive traits are expected to show smaller genetic variability if the traits were under intense selection (Falconer 1981; Fisher 1930). If males or females were faced with certain selection pressures for political preferences unique to one sex, the amount of genetic variance should actually be very small. This does not undermine genetic influences while the variance is not genetic, the mean effect may be. However counterintuitive, evolutionary designs may predict lower genetic variance in the sex for which the item is most likely related to selection, as the traits that matter most are the most tightly regulated.
sources, when quantitative analyses result in sizeable
genetic influence for one sex, while none for the other
(absent of model fitting), the findings offer reason to
suspect that different genetic influences are present
on the trait, or that possibly gene by gene interaction
specific to one sex may be present.

Furthermore, in most items (20 out of 27) the
magnitude of common environmental influence was
similar between males and females. In general, the
common environment accounts for individual differ-
ces in females to the same degree as males, but
affects the sexes differently for each specific item.
Unique environmental influences accounted for the
majority of the variance in all attitudes except Party
Identification for males and School Prayer, Abortion,
and Party Identification for females. Whether evalu-
ating traditional social science theories or evolu-
tionary theories, there is no evident pattern to the
quantitative variance component analyses, except to
say that for individual differences in political prefer-
ces, in general genes matter, and often influence
males and females to differing degrees depending on
the trait. In many ways, this finding runs contrary to
what is inferred by the extant literature. Any attempt
to adopt a universal explanation for the magnitude of
sex differences on all political behaviors is not sup-
ported by the data. Rather, a theory specific to each
item or possibly each group of related items appears
to be appropriate.

**Qualitative Results: Different Sources of
Genetic Influence**

Possibly the most intriguing aspect of sex limitation
modeling is the potential that different genes may be
influencing political preferences for either sex. Table 2
presents the results of the qualitative model fitting for
sex differences on the 27 social and political items
and provides evidence that different genes may be
influencing the preferences of males and females for
one attitude, Divorce. A model in which genes specific
to one sex fits marginally better than a model where
the same genes are influencing attitudes on Divorce
($3.62 \Delta \chi^2$ for 1 d.f., $p=.057$).²

If the evolutionary psychology theses are to be
believed, for certain traits females and males have
been under divergent natural selection pressures for
tens of thousands of years, and it is not surprising
that different genes, potentially on the sex chromo-

²We found no evidence of significantly different sources of common
environment influence across sexes on any attitudes. However, for
attitudes on Divorce and Women’s lib, no such test was possible, as
the common environmental estimate was zero in males.

somes, influence political behaviors that directly or
indirectly influence reproduction (i.e., Divorce and
Living Together). However, it might equally be ar-
gued that attitudes toward Abortion, Gay Rights, and
X-rated are also what one might expect to be connected
with fitness, equally evolutionarily novel, and under
divergent selection pressures for males and females. If
true, these items would also be expected to be poten-
tially influenced by different genes. This is apparently
not the case. Thus, while not presuming to make a
general claim in the absence of overwhelming evidence
that different genes for males and females influence
all political traits related to fitness, the findings on
Divorce and Living Together offer a prima facie case
for considering the evolutionary rationale for sex
differences on political preferences related to fitness.

However, the reduced DZ opposite-sex correla-
tion does not by itself preclude the critical impor-
tance of sex roles, because it might be argued that
the sex limitation of genetic differences may be a
(phylogenetically) recent response to greater social
emphasis on the differentiation of sex roles. While we
can find no evidence in our analyses to support this
claim, it is possible to consider that social gender roles
are so important that they even modify the expres-
sion of genes differentially in the two sexes.

In summary, the analyses provide empirical evi-
dence that the same genes influence males and females
differently for many political preferences. The data
also support the conclusion that modern political
preferences related to sexually dimorphic adaptive traits
(such as reproduction), are the most likely candidates
for different sources of genetic influences in males
and females; a finding which supports themes long
suspected by evolutionary theorists.

**Discussion**

Cultural and biological theorists agree that there are
basic differences in the outlooks and preference for-
mation of males and females, but the source of these
differences remains an area of disagreement. The anal-
yses here provide evidence that differences between
men and women go beyond obvious superficial anat-
omical contrasts or how they are treated by members
in society. For better or worse, genetic influences are
manifested in many aspects of political behaviors and
these influences differ for males and females, both in
magnitude and sources of variation.

The findings support the conclusion that the
tortuous developmental pathway from genes to
political preference cannot be simplified to a universal theory that explains all differences between and within the sexes, or even the preference on one attitude. Rather, differences in the magnitude of genetic and environmental variance between the sexes occur for some political attitudes, but not others. Thus, the data do not support the broad application of social psychology or evolutionary psychology theories to explain behavior differences, or even explain all behaviors suggested as appropriate to either given theory. Accordingly, we suggest it is necessary to examine political preferences on an individual item basis for each sex. The findings support the claim that the environment (social or other) cannot be used in isolation to explain behavior differences between males and females, nor can all differences in modern political behaviors between the sexes simply be attributed to genes or presumptions about primitive man.

By no means do the findings infer that the social environment or personal experience does not have explanatory capacity, or that it is not the strongest determinant in behavior differences. In certain cases such as Party Identification, it appears the social environment is almost all that matters, for either sex. Rather, the social environment does not account for the majority of variance in most political preferences in the population sampled. Environmental, familial, and heritable influences each account for different amounts of the variance on a trait level basis and are likely interactive. Genetic influences for social traits may only be apparent under social conditions. More complex models, with specific measures for environmental influence, tailored to each individual trait are required to explore this possibility.

The qualitative analyses provide a much clearer finding and possibly more radical departure from

<table>
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<tr>
<th>Model</th>
<th>Sex Specific A (1)</th>
<th>fixed A to .5 (2)</th>
<th>p-values 1 vs. 2 (1df)</th>
<th>Genetic correlation between males and females</th>
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Notes: Significant qualitative sex-specific genetic effects noted in bold. Columns 2 and 3 give the - twice log likelihood (-2LL) for model (1) in which the correlation between the latent A variables are estimated for the DZ opposite sex twins, and model (2) in which the correlation between the latent A variables are set to .5 for the DZ opposite sex twins. Column 4 gives the p-values for the 1 degree of freedom chi-square test comparing the -2LL from Model 1 to that of Model 2. Column 5 provides the estimated A correlation between males and females.
traditional social science beliefs. Considering that different genes act in a different manner for males and females to influence political preferences offers an explanation that addresses human biology as well as the social environment people live in. If the results are to be believed and replicated, the need to investigate genetic systems that act differently in males and females for a host of political and social traits is apparent, with a specific focus on issues related to mating and reproductive success.

The links between genes, neurological chemical pathways, emotive and cognitive processes, and social behaviors remain understudied. Controlled mating and repeated experiments provide evidence that vole pair-bonding behavior is strongly influenced by inducing or blocking vasopressin uptake in males and regulating oxytocin uptake in females. Only two attitudes, Divorce and Living Together, resulted in qualitative sex differences for genetic influence. The finding is provocative, as these two attitudes are also the issues most related to pair bonding in our 27-item index. Existing studies on humans are limited, and the findings here justify the need for additional research on the relationship between these neuropeptides, human affiliative behaviors, and political preferences.

Human behavior is influenced partly by biological factors; the precise amounts and mechanisms are partly dependent upon sex. This does not mean genetic sex differences predetermine behavior; quite the contrary, the effect of the environment is equally strong or stronger and significantly different between men and women. Nor should we consider variance components estimates as absolute mutually exclusive values. Genetic and environmental influences are interactive. Here we report findings from an initial univariate classical twin model; future models may provide more clarity on how these components interact with important covariates. Genetic differences in social behavior can only be elucidated in a social context, and it could be that social roles are modulating the expression of genetic sex differences.

We are only in the early stages of employing genetic models to explore political preferences, and more sophisticated analyses with broader environmental and neurological measures, specific to each sex, are needed to explore the heuristic value and implications of these initial findings. However, it is clear that the etiology of political behavior is not homogeneous for the sexes. Genes matter and they matter differently for each sex. The evidence implies that sex differences for political preferences are best explained using an approach which includes a combination of factors and theories unique to each topic, with a particular focus on issues related to reproductive fitness. Specifically, the findings support the use of a more nuanced theory that addresses the roles of genes and environment, but also one that is embedded within a model that is consistent with the complex social, environmental, developmental, and biological elements specific to each sex. Doing so will challenge existing political behavior models which only consider social determinants. However, ignoring the influence of inherent sources of political behavior renders contemporary research on sex differences incoherent at a time when the issue of sex remains central to the U.S. electorate.

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