Are Political Orientations Genetically Transmitted?

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We 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 beliefs 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

### Footnotes

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 reuptake (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 more, 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. 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

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4 However, recent research suggests that the prenatal environment is so important that it can cause variation even in fetuses inhabiting the uterus at the same time. Prescott, Johnson, and McArdle (1999) present evidence that MZ twins sharing the same chorion, the outermost extraembryonic membrane, are more similar in terms of personality and cognitive abilities that MZ twins in separate chorions.
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 compo-
nent, frequently stronger than attitude covariance at-
tributable to shared environment, we predict that polit-
cal attitudes will also be heavily heritable. Heritability
estimates calculated by previous researchers for atti-
duates associated with psychological conservatism are
quite high, while the relevant models typically show
little or no effect for shared environment (the remain-
der 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).5 Careful studies of
adopted children confirm the finding that genetics mat-
ter more than parentally created environment in influ-
encing social attitudes and behaviors, personality traits,
and intelligence.6

We further predict that attitudes on political is-
issues tracking most closely to central personality traits
should be the most heritable since personality traits
are generally heritable and since the heritability of so-
cial attitudes is likely derivative of the heritability of
various personality traits (see Bouchard and Loehlin
2001 and Eaves, Eysenck, and Martin 1989). For ex-
ample, one of psychology’s “Big 5” personality traits is
general “openness” and it seems likely degree of open-
ness 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,
we seem that group identification is something that
is heavily influenced by the environment, especially
shared environment, and is mostly unconnected to ge-
etics. Children of Methodists are likely to be Metho-
dists not because there is a gene for Methodism or even
a personality particularly oriented toward Methodism,
but because of parental socialization. Thus, even as atti-
itudes 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 ge-
etic 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 re-
versed 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 anticip-
ating 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 po-
litical scientists, political attitudes have been at best
a sidenight, 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 vari-
bles in twin studies have political relevance. For ex-
ample, the heritability of conservatism is frequently as-
sessed (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 admin-
istered by presenting subjects with a short stimulus

5 Conservatism is not unusual in this regard. Rushton, Littlefield, and
Lumsden (1986, 7340) find that approximately 50% of the variance
in altruism is the result of “direct genetic inheritance,” with family
environment responsible for 0%.
6 Adoption studies measure the correlation of biological parents and
adopted children where the biological parents have had no contribu-
tion to the rearing (environment) of the child. The most recent adop-
tion study, utilizing surveys of Korean-American adoptees randomly
assigned to families in the United States, concludes that roughly 75%
of variance in children’s educational attainment is attributable to
the educational attainment of their biological parents, and only 25% is
attributable to the adoptive parents, thus dramatically confirming
the earlier findings of a substantial correlation between biological
parents and adopted children and a surprisingly paltry correlation
between adoptive parents and children (Sacerdote 2004). This par-
allels, with an entirely distinct methodology, the basic finding of the
twin studies (see Plomin et al. 1997, 1998 and Rhee and Waldman
2002).
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.7 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 2.0 × .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 (2.0 × .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 $2.0 \times .4 = .8$, and $1.0 - .4 = .6$). 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 nonexistent but small relative to shared environment is also called into question. We see from Table 1 that the

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7 Our thanks go to Professor Lindon Eaves at Virginia Commonwealth University for making the VA30K data available to us. The data collection methods for both studies are summarized in Lake et al. 2000 as follows: “The Australian sample was ascertained through two cohorts of twins. The first cohort was recruited in 1980–1982 from a sampling frame which comprised 5967 twin pairs aged 18 years or older (born 1893 to 1964) then enrolled on the Australian NHMRC Twin Registry (ATR). Responses were obtained from 3808 complete pairs...and these were followed up with a second mailed questionnaire in 1988–1990 with responses from 2708 complete pairs...The second cohort of twins, born 1964–1971, was recruited from 1988 to 1991 and was mailed similar questionnaires in 1989–1991, with responses from 3,769 individuals of 4,269 eligible pairs...In total there were 21,222 respondents in the Australian sample, of whom 20,945 had valid scores for EPQ Neuroticism. The United States twins were ascertained from a population-based birth registry for the Commonwealth of Virginia and from a volunteer sample through the American Association of Retired Persons (AARP), described in detail by Truett et al. (1994). Their first-degree relatives and spouses were recruited in a similar fashion to the Australian sample, and in total there were 24,905 respondents (of 29,080) with valid scores for Neuroticism and for whom the zygosity of the proband twins could be determined. The response rates were 70% for twins and 45% for relatives” (224–25). The original U.S. twin data collection was funded in part by NIH grants GM30250 and AG04954, by ADAMHA grants AA06781, AA07728, AA07555, and MH40928, and by a gift from R. J. R. Nabisco.
TABLE 1. Genetic and Environmental Influences on Political Attitudes: The 28 Individual Wilson–Patterson Items

<table>
<thead>
<tr>
<th>Attitude Item</th>
<th>Polychoric Correlation MZ</th>
<th>Polychoric Correlation DZ</th>
<th>Heritability, (2 * (MZ - DZ))</th>
<th>Shared Environment, ((2 * DZ) - MZ)</th>
<th>Unshared Environment, (1 - MZ)</th>
<th>(z) for (MZ–DZ) Differencea</th>
</tr>
</thead>
<tbody>
<tr>
<td>School Prayer</td>
<td>0.66 2,687</td>
<td>0.46 1,774</td>
<td>0.41</td>
<td>0.25</td>
<td>0.34</td>
<td>9.83</td>
</tr>
<tr>
<td>Property Tax</td>
<td>0.47 2,643</td>
<td>0.27 1,748</td>
<td>0.41</td>
<td>0.06</td>
<td>0.53</td>
<td>7.66</td>
</tr>
<tr>
<td>Moral Majority</td>
<td>0.42 2,614</td>
<td>0.22 1,717</td>
<td>0.40</td>
<td>0.03</td>
<td>0.58</td>
<td>7.16</td>
</tr>
<tr>
<td>Capitalism</td>
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<td>0.34 1,720</td>
<td>0.39</td>
<td>0.14</td>
<td>0.47</td>
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</tr>
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<td>0.28 1,721</td>
<td>0.39</td>
<td>0.09</td>
<td>0.52</td>
<td>7.39</td>
</tr>
<tr>
<td>The Draft</td>
<td>0.41 2,641</td>
<td>0.21 1,753</td>
<td>0.38</td>
<td>0.02</td>
<td>0.59</td>
<td>6.94</td>
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<td>0.04</td>
<td>0.68</td>
<td>6.38</td>
</tr>
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<td>0.07</td>
<td>0.60</td>
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</tr>
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<td>0.12</td>
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</tr>
<tr>
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<td>0.25 1,726</td>
<td>0.36</td>
<td>0.07</td>
<td>0.60</td>
<td>6.53</td>
</tr>
<tr>
<td>Foreign Aid</td>
<td>0.41 2,669</td>
<td>0.23 1,771</td>
<td>0.35</td>
<td>0.06</td>
<td>0.59</td>
<td>6.42</td>
</tr>
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<td>X-Rated Movies</td>
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<td>0.46 1,783</td>
<td>0.35</td>
<td>0.28</td>
<td>0.37</td>
<td>8.15</td>
</tr>
<tr>
<td>Immigration</td>
<td>0.45 2,658</td>
<td>0.29 1,748</td>
<td>0.33</td>
<td>0.12</td>
<td>0.55</td>
<td>6.20</td>
</tr>
<tr>
<td>Women's Liberation</td>
<td>0.46 2,666</td>
<td>0.30 1,779</td>
<td>0.33</td>
<td>0.13</td>
<td>0.54</td>
<td>6.27</td>
</tr>
<tr>
<td>Death Penalty</td>
<td>0.56 2,684</td>
<td>0.40 1,775</td>
<td>0.32</td>
<td>0.24</td>
<td>0.44</td>
<td>6.83</td>
</tr>
<tr>
<td>Censorship</td>
<td>0.40 2,629</td>
<td>0.25 1,718</td>
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<td>0.10</td>
<td>0.60</td>
<td>5.36</td>
</tr>
<tr>
<td>Living Together</td>
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<td>0.52 1,771</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 2,625</td>
<td>0.24 1,733</td>
<td>0.29</td>
<td>0.09</td>
<td>0.62</td>
<td>5.24</td>
</tr>
<tr>
<td>Gay Rights</td>
<td>0.60 2,658</td>
<td>0.46 1,767</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 2,653</td>
<td>0.24 1,743</td>
<td>0.27</td>
<td>0.11</td>
<td>0.62</td>
<td>4.83</td>
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<tr>
<td>Busing</td>
<td>0.43 2,650</td>
<td>0.30 1,766</td>
<td>0.26</td>
<td>0.15</td>
<td>0.57</td>
<td>4.92</td>
</tr>
<tr>
<td>Nuclear Power</td>
<td>0.42 2,646</td>
<td>0.29 1,744</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 2,639</td>
<td>0.34 1,726</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 2,659</td>
<td>0.34 1,765</td>
<td>0.26</td>
<td>0.21</td>
<td>0.53</td>
<td>4.99</td>
</tr>
<tr>
<td>Abortion</td>
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<td>0.52 1,768</td>
<td>0.25</td>
<td>0.39</td>
<td>0.36</td>
<td>6.23</td>
</tr>
<tr>
<td>Modern Art</td>
<td>0.43 2,662</td>
<td>0.30 1,765</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 2,665</td>
<td>0.26 1,766</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 2,629</td>
<td>0.35 1,734</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 2,648</td>
<td>0.31 1,748</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).

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

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 noncommittal (?) 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 .14, 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 shared environment factors accounting for only one-third of the variation. This decline in the role of unique individual and environmental factors accounting for only 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 all 28 items and a score of zero indicating that the respondent was unwilling to offer 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 assumption is that the parents are not related to each other in any close degree, and this is typically true, as close relatives generally do not mate, and the amount of average shared genetic code drops geometrically as we move away in relatedness and quickly approaches zero. This assumption that mates are genetically uncorrelated on the trait of interest is, however, violated if mate choice is itself based on the trait of interest. If, for example, parents have identical genetic codes for a trait of interest, then the shuffling of that genetic code produced by sexual reproduction will not result in any variation among DZ twins, or any other siblings, with regard to their genotype for that trait. 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 $(2 * DZ) - MZ$ 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
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 offsprings’ 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 partialed 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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**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(MZ – DZ))</td>
<td>Heritability, (2(MZ – DZ))</td>
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<tr>
<td>Astrology</td>
<td>Male</td>
<td>Female</td>
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<tr>
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<td>0.49</td>
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<td>Pacifism</td>
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<td>0.00</td>
</tr>
<tr>
<td>Censorship</td>
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</tr>
<tr>
<td>Socialism</td>
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<td>0.36</td>
</tr>
<tr>
<td>Military Drill</td>
<td>0.42</td>
<td>0.24</td>
</tr>
<tr>
<td>Immigration</td>
<td>0.29</td>
<td>0.35</td>
</tr>
<tr>
<td>Death Penalty</td>
<td>0.27</td>
<td>0.35</td>
</tr>
<tr>
<td>Women’s Liberation</td>
<td>0.33</td>
<td>0.35</td>
</tr>
<tr>
<td>Segregation</td>
<td>0.34</td>
<td>0.23</td>
</tr>
<tr>
<td>Modern Art</td>
<td>0.31</td>
<td>0.21</td>
</tr>
<tr>
<td>Abortion</td>
<td>0.26</td>
<td>0.24</td>
</tr>
<tr>
<td>Divorce</td>
<td>0.20</td>
<td>0.28</td>
</tr>
<tr>
<td>Mean</td>
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<td>0.29</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Attitude Item</th>
<th>Australian Data</th>
<th>Shared Environment, (2(DZ) – MZ)</th>
</tr>
</thead>
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<tr>
<td>Astrology</td>
<td>0.30</td>
<td>0.32</td>
</tr>
<tr>
<td>Pacifism</td>
<td>0.26</td>
<td>0.48</td>
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<td>Military Drill</td>
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<td>0.42</td>
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<td>0.54</td>
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<td>Modern Art</td>
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<td>Legalised Abortion</td>
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<td>Divorce</td>
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</tr>
<tr>
<td>Mean</td>
<td>0.24</td>
<td>0.37</td>
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</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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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.
utilized 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.9

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 Groudine 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

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9 In this case, the different results with regard to socialism could reflect different meanings of the phrase in the two countries. In Australia, the term socialism is closer to a party identification label, whereas in the United States it has more loaded ideological connotations. Likewise, the addition of the qualifier “nonwhite” to immigration raises questions of what the key stimulus is.
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, genetics 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 (Carmines 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—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 intrinsisnec 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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The Origins of Political Attitudes and Behaviours: An Analysis Using Twins

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1. Introduction

In what must surely rank as one of the strangest episodes in the entire history of science, two generations of our immediate forebears in the social sciences managed to virtually ignore the “Darwinian” theory of biological evolution and to exclude from their purview any sustained consideration of the role of biological factors in the shaping of human behaviour. (Corning, 1971: 321)

The curious practice Corning describes above has continued through a third generation of political scientists and sociologists and is now reaching into a fourth. Although bio-evolutionary perspectives are now mainstream in psychology and the paradigm is beginning to have a major impact in the emerging fields of behavioural economics and neuroeconomics, most political scientists and sociologists are still “missing the revolution” (Barkow, 2006). Some change is on the horizon in those disciplines, but the general picture there is one of splendid isolation from the concepts, methods and findings of evolutionary biology and behavioural genetics.¹

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This paper offers those who study Canadian politics an opportunity to consider their subject matter from the perspective of behaviour genetics. Using twin data, it provides a biological heritability analysis of a number of facets of Canadian politics, including political attitudes, feelings toward the major parties, party identification and vote choice.

While the conventional approaches to politics are in many ways invaluable, they have some shortcomings. In particular, they share what van den Berghe calls the “orthodoxy of sociocultural environmentalism” (1990: 177). This view assumes that political beliefs, attitudes and behaviours are a product of one’s social location or situation, plus the culture or ideology to which one has been exposed—biological or genetic influences are not considered. Some scholars writing on American politics, for example, have maintained that the most important sources of political orientations are found in a person’s immediate social milieu, in particular one’s family, religious congregation, friendship network, or work environment (for example, Berelson et al., 1954). Here the assumption is that political outlooks are acquired through social learning: one picks up or absorbs the views of the people in one’s immediate environment. This perspective is illustrated in words of Robert Altemeyer, who maintains that we “get our opinions where we get our corn pone—at home” (quoted in Bourchard and McGue, 2003: 960). Different homes, different attitudes, according to this argument. Any correspondence in attitudes or voting among family members is usually assumed to be the result of similar environmental influences.

A related conventional view maintains that party identification is crucial in understanding political matters, and that one acquires a particular partisan attachment primarily through familial socialization (for example, Campbell et al., 1960). People are said to develop an enduring psychological attachment to a particular party, which affects a host of political attitudes and makes it very likely that the person will vote for the party. Again, similarities in political orientations and behaviours are presented as, ultimately, a consequence of similar socialization experiences.

A contrasting approach to American politics maintains that political views and decisions are the result of a rational calculus on the part of the citizen (for example, Downs, 1957; Key, 1966; Fiorina, 1981; Popkin, 1991; Lupia and McCubins, 1998). In these sorts of studies, consistent support for a particular party is portrayed as a series of rational decisions rather than the outcome of social learning or a psychological attachment to the party. The authors of these studies assume that political actors are rational and self-interested, but by and large they are silent on where the actors’ goal preferences come from (Alford and Hibbing, 2005; Orbell, 2005). They too tend not to see bio-evolutionary processes as applicable to the phenomena they study.
Abstract. This article provides a behaviour genetic heritability analysis of several political issues, including social and economic conservatism, general interest in politics, attitudes toward the major Canadian federal parties, federal party identification and national vote choice. Substantial genetic effects were found for four of six political attitude scales, with heritability values ranging from 41 per cent to 73 per cent. Genetic effects are also reported for several individual items (including feelings toward the major federal parties, party identification and vote choice), with heritabilities from 33 per cent to 62 per cent. The implications of these results for conventional political analyses are explored. Also presented is a theoretical interpretation of political heritability that is derived from an evolutionary perspective which suggests that political personalities or temperaments have evolved that are analogous to the heritable personality structures proposed by psychologists.

Résumé. Cet article propose une analyse sur l’héritabilité de la génétique du comportement concernant plusieurs questions politiques, y compris le conservatisme social et économique, l’intérêt général pour la politique, les attitudes envers les principaux partis fédéraux canadiens, l’identification à un parti et le choix de vote au niveau national. Des effets génétiques notables ont été recensés pour quatre des échelles politiques d’attitude sur six, les taux d’héritabilité s’étendant de 41 pour cent à 73 pour cent. Des effets génétiques ont également été recensés pour plusieurs autres éléments étudiés (y compris les sentiments envers les principaux partis fédéraux, l’identification à un parti et le choix de vote), les taux d’héritabilité allant cette fois-ci de 33 pour cent à 62 pour cent. Cette étude explore l’incidence de ces résultats sur des analyses politiques conventionnelles. Il s’agit aussi d’une interprétation théorique de l’héritabilité politique dérivant d’une perspective évolutionnaire, qui suggère que les personnalités ou les tempéraments politiques ont évolué et que ces derniers sont analogues aux structures de personnalité transmissibles proposées par les psychologues.

Scholars analyzing Canadian politics have considered similar issues. For example, there is an ongoing debate about the extent to which the American notion of party identification applies to Canadian politics (for example, Meisel, 1973: 67; Sniderman et al., 1974; Jenson, 1975; Clarke et al., 1979, 1984, 1991, 1996; Stephenson et al., 2004; Gidengil et al., 2006). Some of those who argue in favour of adopting the party identification model have assumed that party allegiances are passed on within families from generation to generation via socialization (for example, Sniderman et al., 1974: 285–86; but see Johnston, 1985).

Those who reject the idea that party identification plays a key role in Canadian politics often adopt a brokerage model in which party allegiances are said to be weak. Under the brokerage paradigm, Canadian elites try to minimize conflict among regional, ethnic, religious and other groups in order to maintain national unity (for example, Dawson, 1970: 415; Clarke et al. 1996: 50). The major parties avoid associating themselves with particular factions of Canadian society in order to unify the country and to attract as many votes as possible. As a result, the leading parties adopt policies that are broadly similar and non-ideological in nature. Elections are won or lost depending on how effectively the party in power deals with the issues and events of the day, the opposition’s ability to come up with alternative programs, and the public’s assessment of the party leaders. Biological influences on political attitudes and vote choice are not considered.
Some researchers maintain that in recent years Canadian politics has become increasingly ideological, and hence that the brokerage model may be less applicable to the contemporary scene than it was to previous eras (for example, Cross and Young, 2002; see also Campbell and Christian, 1996; Johnston, 1988; Blake, 1988). There is also a sizeable literature on the role of class, religion and gender in Canadian politics (for example, Ogmundson, 1975; Pammett, 1987; Nieuwbeerta, 1996; Blais, 2005; Bashevkin, 1993; Young, 2000). But regardless of the theoretical position or paradigm adopted, only rarely does one find biological characteristics invoked as a possible explanation for Canadian political phenomena (for an exception, see Laponce, 1981).

To be sure, the conventional approaches have made major contributions to our understanding of politics. For example, political parties are sometimes found to appeal disproportionately to people in certain demographic categories, for instance white, Protestant males over the age of 30, or perhaps well-educated francophones employed in the public sector. While it is important to be aware of such propensities, these sorts of analyses can be taken further. Not everyone in the identified categories thinks or acts in the same way; and rarely does one come across efforts to explain why people with the same demographic characteristics who are in similar social situations may behave differently. Presumably, such differences are attributed to differential socialization or exposure to different events or experiences. Conversely, if people take similar political positions or have the same attitudes about political matters, it is assumed that they have been subject to the same circumstances and socio-cultural influences. That political similarities and differences may be partly explainable in terms of biological or genetic factors is not normally entertained.

The approach taken in this study is meant to complement, not replace, conventional social science explanations for political attitudes and behaviours. It simply adds to the discussion a heretofore neglected or ignored factor, namely biological effects. Moreover, the position taken here is that the best explanations for political phenomena will arise from a melding of the traditional and biological approaches to politics. Either path taken separately leads only to intellectual dead ends and serious misunderstandings.

2. A Different but Complementary Approach

For decades now, psychologists and behaviour geneticists have examined the role of heredity in the development of political attitudes (for example, Eaves and Eysenck, 1974; Feather, 1978; Scarr and Weinberg, 1981; Martin et al., 1986; Truett et al., 1992; Loehlin, 1993; Eaves et al., 1997;
Understandably, political phenomena have been mainly a sidelight for these researchers and fully developed measures of political variables have not been used (Alford et al., 2005: 157). Also, political scientists and sociologists have not fully explored the implications of the findings of these studies.

A primary purpose of this paper is to build on the recent work of a handful of political scientists who have made the case that genetic factors contribute to political attitudes and behaviours (for example, Alford and Hibbing, 2004, 2007a; Alford et al., 2005; Hibbing and Smith, 2007; Carmen, 2006; Hatemi, Medland et al., 2007; Hatemi, Alford et al., 2007; Hatemi, 2007; Fowler et al., 2008; Fowler and Dawes, 2008; Dawes and Fowler, 2009). One contribution we seek to provide is a fuller assessment of the heritability of conservatism than that offered in earlier works. Unlike much of the existing research on the topic, this analysis is not based on a version of the Wilson-Patterson (W-P) conservatism scale (Wilson, 1975). The W-P scale was considered unsatisfactory because it excluded attitudes toward economic issues and it combined unequivocally political items (for example, disarmament, socialism, empire building) with items of only tangential political relevance (learning Latin, conventional clothes, chaperones). By contrast, we use scales and individual items that measure different dimensions of conservatism (including attitudes toward economic equality) and which focus on political topics.

Distinguishing between types of conservatism is important because there can be tensions and disagreements among people typically described as conservative. In Canada, for example, there are important ideological differences between “tories” (who consider the right to economic gain to be subordinate to national and political goals, and who value tradition, community and assistance to the disadvantaged) and “business liberals” (who place primary importance on free enterprise, free markets, fiscal restraint and economic self-reliance) (Christian and Campbell, 1990: 100–73). These tensions became manifest in recent years with the merging of the Progressive Conservative party and the Reform/Alliance party, which produced an amalgam of old-line tories, market liberals and social conservatives that has not been entirely peaceful. A glaring illustration of this is former Progressive Conservative Prime Minister Joe Clark’s endorsement of Paul Martin over Stephen Harper in the 2004 federal election campaign. Similarly, Layman and Carsey (2002: 791) have shown that at the mass level in the United States, positions on social welfare issues and racial and cultural topics represent distinct issue dimensions, despite the tendency toward convergence in these three attitude areas at the elite level.

Another matter that receives attention here which is not examined elsewhere is the heritability of a general interest in politics, an issue that
has implications for established theories of political participation and political knowledge, and which may have practical relevance for political campaigns.

Also presented is a theoretical interpretation of political heritability, one that is derived from an evolutionary perspective. It is proposed that political personalities or temperaments have evolved that are analogous to the heritable personality structures proposed by psychologists. Specific hypotheses are derived from this perspective and evidence is brought to bear on them, although the data permit only a preliminary assessment of this issue.

3. An Introduction to Twin Studies

Since few readers of this journal will be familiar with behavioural genetics or more specifically twin studies, a brief introduction to those topics is offered here. Twins are categorized into two basic types: monozygotic (MZ) and dizygotic (DZ). The former, known informally as identical twins, share virtually all of their genetic structure—they are essentially clones of each other, although miniscule genetic differences are known to occur. MZ twins develop in utero when a single fertilized ovum divides into two identical cells, resulting in two embryos. DZ twins, popularly referred to as fraternal (although the term covers females as well as males), develop when two ova fertilized by two separate sperm attach themselves to the uterine wall at the same time. DZ twins share, on average, approximately 50 per cent of the genetic material that varies between human beings, and are as genetically similar to each other as non-twin full siblings. It is the fact that MZ twins have twice the genetic similarity of DZ twins that forms the basis of the classic twin methodology.

The classic twin design is used to determine the general causes of the variation of a particular characteristic or behaviour in a population; it cannot be used to explain the qualities of a particular individual (but see Sesardic, 2005: 51–56). Population variation in the model used here is seen as resulting from three broad sources commonly referred to as “A,” “C,” and “E.”

1. Additive genetic effects (A). Such effects occur if the joint impact of alleles (the different forms that particular genes can take) at a locus or across loci is the sum of their individual effects. With additive genetic effects, the influence of alleles does not change if other alleles are present (Plomin et al., 2008: 161).

2. Variations in “common environments” (C). Having a common environment generally refers to growing up in the same household, but it can include any experience shared by both twins, such as being in the same class in school. Siblings growing up in a politically conser-
ervative (or liberal) household have a common environment in that they are exposed to the same parental political ideology.

3. Unshared environmental differences (E), which are experiences not shared by co-twins. For example, at age 20 a particular woman may join the military while her sister opts for a career in dance; or perhaps a teenaged boy has a charismatic hockey coach who inspires him to excel in athletics, while his brother doesn’t have contact with that coach.

The classic twin design centres on the difference between the Pearson’s r coefficient for MZ twins and that for DZ twins on a particular characteristic. Traditionally, A is estimated by doubling that difference:

$$A = (MZ_r - DZ_r) \times 2$$

The rationale involves the idea that MZ twins have twice the genetic similarity of DZ twins. Using this estimate, if a particular attribute were wholly determined by genetic influences, MZr would equal 1, and DZr would equal .5, resulting in a heritability estimate of $$(1 - .5) \times 2 = 1$$ or 100 per cent. If the attribute were not influenced at all by genetic factors, MZr and DZr are expected to be equal, yielding a heritability estimate of 0. For example, if the two Pearson’s r coefficients were .3, A = (.3 - .3) * 2 = 0 or 0 per cent.

Common environmental influences in the classical design are estimated as follows:

$$C = (DZr \times 2) - MZr$$

Using this estimate, if a characteristic were completely attributable to genetics, the effect of the common environment would be nil: (.5 * 2) - 1 = 0 or 0 per cent. At the other extreme, if genes play no role whatever in producing the characteristic, the correlations for MZ and DZ twins as mentioned are expected to be equal, making the estimate of the effect of common environments equal to the Pearson’s r of either of the two twin types. For example, if the two correlations were .3, the equation would produce: C = (.3 * 2) - .3 = .3.

In the classic twin design, unshared environmental influences are estimated by taking whatever remains after additive genetic and common environmental influences have been accounted for:

$$E = 1 - (A + C)$$

More recently, the use of structural equation modelling (SEM) has provided major improvements over the classic design, while retaining the same basic assumptions. This paper takes the SEM approach using Mx software (Neale et al., 2006), which allows for the simultaneous estimation of the additive genetic, common environment and unique environ-
ment effects; it can also assess whether a reduced model (such as AE, CE, or E-only) has a better fit to the observed data than the full ACE model by comparing fit indices.

The methodology used here is agnostic regarding which specific gene or genes are involved in political behaviour. However, some researchers have made efforts to establish a link between a particular gene or set of genes and certain forms of political activity, although the research is still very preliminary. Fowler and Dawes (2008), for example, found a link between the MAOA and 5HTT genes and voter turnout, and Dawes and Fowler (2009) found a relationship between the D2 dopamine receptor gene and partisan attachment. Settle and colleagues (2008) provide evidence that there is an association between the DRD4 gene and political ideology. Similarly, Oxley and colleagues (2008) present data indicating that physical traits can be used to predict some political attitudes.

A final introductory point concerns the old “nature versus nurture” debate. In the approach used here, the relative contribution of nature and nurture is taken as a matter to be resolved empirically—their influence or lack thereof is not declared a priori. Also, many researchers in the field maintain that the old debate was overly simplistic in that it did not take into consideration gene–environment interactions or correlations—nature via nurture comes closer to contemporary scientific attitudes on the subject (Ridley, 2003). For instance, through various epigenetic processes certain genes may not get expressed unless the environment is favourable. Moreover, the C and E components, which broadly speaking fall under the heading of nurture, often explain large portions of the variation observed. For example, MZ twins may experience different environments that may affect their physical, emotional and cognitive development, and which can lead to important differences between them. Nor are environmental influences ignored by researchers in the field of behaviour genetics. Scholars studying political heritability routinely acknowledge the importance of environmental factors (for example, Fowler et al., 2008: 244), and the effect of group, societal and global culture on behaviour in general is acknowledged to be crucial (Lumsden and Wilson, 1981; Richerson and Boyd, 2005).

4. Theoretical Considerations and Hypotheses

Comprehensive, empirically informed theories purporting to explain why political phenomena should be partially heritable have yet to be formulated by scholars and researchers. This is not surprising, given that the notion that political attitudes and behaviours could be partially heritable only became widely recognized by the political science community with the publication of a breakthrough article by Alford, Funk and Hibbing in
2005. Our position, inspired by the work of Hibbing, Alford, Boehm and others (for example, Hibbing and Alford, 2004; Alford et al., 2005; Alford and Hibbing, 2007b; Alford et al., 2008; Boehm, 1999) is that political attitudes and actions derive in part from innate psychological and cognitive traits that have evolved over many thousands (and probably millions) of years. As Alford and colleagues (2008: 325) point out, there is growing acceptance of the notion that political attitudes and orientations are related to more general, underlying traits that are not normally conceived of as political, such as openness to experience (see also Jost et al., 2003). The notion that political phenomena may be related to underlying psychological and cognitive characteristics goes back at least as far as the work of Adorno and his colleagues (1950) and the body of research that was produced in reaction to it.

The theoretical approach taken here involves the hypothesis that political orientations derive from the long history of human sociality. Pre-modern hominids lived in bands long before forming our current species, and modern Homo sapiens lived in small societies for the vast majority of the approximately 200,000 years of their existence. It stands to reason that after coping with intra- and inter-societal co-operation and conflict, the sorts of interactions now referred to as “politics,” humans would have developed some innate feelings and outlooks about those matters—constellations of orientations that could be described as “political personalities” or “political temperaments.” Just as we humans may have an evolved fear of heights that varies in magnitude from person to person and that may ultimately have genetic and hence physiological antecedents, we may also have political temperaments that are partially derived from our genes and physiology and which may interact with situational and cultural factors to produce political behaviour.

This theoretical stance would allow one to explore a number of political phenomena from an evolutionary and genetic perspective. One could examine several situations in our ancestral past that were relevant to human survival that may have produced dispositions that now affect political attitudes and orientations. Should foreign bands or societies be trusted or are they to be feared? Should societal leaders and others in dominance hierarchies be given deference or should they be challenged? Should individuals be responsible for their own welfare and safety or should there be some sharing of resources? Should people who deviate from group norms be tolerated or punished? Evolved dispositions toward these general issues may contribute to people’s attitudes toward specific aspects of contemporary politics such as the war in Iraq or the level of spending on social programs.

While we are a long way away from knowing whether such issues in our ancestral history actually are related to contemporary political phenomena, a number of testable hypotheses can be derived from this gen-
eral theoretical orientation. To take one, if there are heritable political 
temperaments, then there should be differences in the levels of concor-
dance between MZ and DZ twins on the political attitudes and behav-
iours measured in this study such that they produce substantial heritability 
estimates. A second hypothesis would be that those variables that mea-
sure general political attitudes should have higher heritability estimates 
than those measuring political behaviours. This hypothesis follows from 
the idea that attitudes reflect temperaments more closely than behav-
iours do. Political temperaments (like conventional personality factors), 
we propose, interact with and are confounded by cultural and situational 
factors to produce behaviours. For instance, just as a normally extroverted 
person may have a largely heritable propensity to loquaciousness but under 
certain conditions (such as situations in which communication is strictly 
regulated) may move closer to the communication style of an introvert, 
so too may a person who normally shows deference to elites grow more 
rebellious under certain political conditions. In those circumstances there 
would be greater differences in temperaments than there would be in 
behaviours. Insofar as attitudes are more closely related to temperaments 
than behaviours are, we should expect higher heritability estimates for 
attitudes.

5. Data and Methods

5.1 Procedure

Adult twins (selected from a registry maintained by Philip A. Vernon at 
the University of Western Ontario) completed a political issues question-
naire as part of a larger battery of questionnaires (see Vernon et al., 2008). 
Upon receipt of their completed questionnaires, each individual was sent 
$25 to compensate them for their time, and their names were entered 
into a draw for one of ten $100 prizes.

5.2 Participants

The sample consisted of a total of 570 individuals, which included 548 
adult twins (385 MZ and 163 DZ individuals) residing in Canada and 
the United States. Of the 570 people in the study, 412 were Canadian 
and 158 American. Of these twins, complete pairs were available from 
37 pairs of MZ males, 155 pairs of MZ females, 5 pairs of DZ males, 56 
pairs of DZ females, and 17 pairs of DZ opposite sex twins. Age infor-
mation was available for 538 individuals, with a mean of 40.5 years (SD = 
16.68) and a range from 16 to 92 years.
In the analyses below that deal with scale construction, demographic variables and self-placement on a left/liberal versus right/conservative political spectrum, all participants were analyzed. For the behavioural genetic analyses, only complete pairs of MZ and same-sex DZ twins were used.

5.3 Measures

The political issues questionnaire created for this study contained 52 items derived from a variety of sources. The first question dealt with general interest in national politics, and then a group of questions asked the Canadian respondents how much they liked the major national political parties. That was followed by a national political party identification question, the left/liberal versus right/conservative self-placement item and a question asking Canadian participants which party they normally voted for in federal elections. After that, 42 additional political attitude items were administered. All but two of the attitude items in this study use a 7-point Likert format; the two exceptions use a 5-point format.4

5.4 Results Preliminary to the Genetic Analyses

5.4.1 Factor structure of the 42 political attitude items

An exploratory factor analysis was conducted on the 42 political attitude items. The Kaiser-Meyer-Olkin value was found to be .83, suggesting that the correlation matrix was appropriate for factor analysis (Tabachnick and Fidell, 2001). Six factors were extracted based on a visual examination of the scree plot, and were found to account for 44.44 per cent of the variance. Following direct Oblimin rotation, the average absolute inter-factor correlation value was found to be .09 (range = .008 to .241), suggesting that the factors were fairly independent. Following that, a Varimax rotation was applied to the extracted factors.

Individual attitude items with high loadings on a single factor were used in the construction of scales. Six of the attitude items were not found to load clearly onto a single factor and were removed from the constructed scales. The six scales created (see appendix A) were given the titles of “religiosity/social conservatism” (for example, frequency of attending religious services and attitude toward same-sex marriage); “environmentalism” (sample item: “It is more important to protect the environment than to create jobs”); “economic equality” (sample item: “Governments should do more to reduce the gap between the rich and the poor”); “activist state on social issues” (for instance, supporting laws against making sexist statements in public, favouring gun control); “competition/business” (views towards competition in general and top business people); and “ethnic/racial minorities” (how much should be
done for racial minorities and whether immigration is beneficial to the country.

Table 1 lists the scale properties for the six scales created. Although the coefficient alpha values ranged considerably (.55 to .83), the number of items per scale is small (2 to 8 items). Also presented are the tests of the variance and mean differences between men and women for the six political attitude scales created. Higher scores indicate greater religiosity/social conservatism and greater support for environmentalism, economic equality, state activism, competition/business and racial/ethnic minorities.

5.4.2 General properties of the sample

In terms of interest in politics, the participants showed low to moderate interest with a mean of 3.69 (SD = 1.75) on a 1 (not at all interested) to 7 (very interested) scale. With regard to self-placement on the political spectrum, the response categories for Canadian respondents ranged from 1 (“very left”) through 4 (“middle of the road”) to 7 (“very right”); for Americans, the designations 1 “very liberal” and 7 “very conservative” were used. Those with responses from 1 to 3 were categorized as “left/liberal,” the 4 response was designated as “centre,” and those answering 5 through 7 were categorized as “right/conservative.” In the combined sample, 38 per cent were left/liberal, 25 per cent were centre, and 28 per cent were right/conservative (responses were missing for 9 per cent of the sample). Responses to the question dealing with self-identification

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**Table 1**

<table>
<thead>
<tr>
<th>Scale</th>
<th>N items</th>
<th>$\alpha$</th>
<th>Male M (SD)</th>
<th>Female M (SD)</th>
<th>$F$</th>
<th>$t$</th>
<th>$r$ with age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Religiosity/social conservatism</td>
<td>8</td>
<td>.83</td>
<td>32.21</td>
<td>30.29</td>
<td>.13</td>
<td>1.64</td>
<td>.22b</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(9.82)</td>
<td>(9.50)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Environmentalism</td>
<td>8</td>
<td>.81</td>
<td>35.07</td>
<td>36.20</td>
<td>.36</td>
<td>−1.16</td>
<td>−.04</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(8.54)</td>
<td>(7.57)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Economic equality</td>
<td>9</td>
<td>.71</td>
<td>33.44</td>
<td>35.54</td>
<td>2.34</td>
<td>−2.34</td>
<td>−.07</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(8.45)</td>
<td>(7.06)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Activist state on social issues</td>
<td>6</td>
<td>.65</td>
<td>24.41</td>
<td>26.10</td>
<td>12.44</td>
<td>−2.05</td>
<td>−.12*</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(7.24)</td>
<td>(5.63)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Competition/business</td>
<td>3</td>
<td>.59</td>
<td>15.11</td>
<td>14.70</td>
<td>.07</td>
<td>1.15</td>
<td>.04</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(3.18)</td>
<td>(2.99)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ethnic/racial Minorities</td>
<td>2</td>
<td>.55</td>
<td>8.96</td>
<td>8.58</td>
<td>.81</td>
<td>1.43</td>
<td>−.18b</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(2.11)</td>
<td>(2.23)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*p < .05, *p < .01 (two-tailed).

$\alpha$ = Cronbach’s Alpha; $F$ = test of variance; $t$ = mean differences.
with the national political parties showed that 25 per cent of the Canadian sample considered themselves to be Conservative, 38 per cent Liberal, 15 per cent NDP, 8 per cent Green, 2 per cent Bloc Québécois, and the remaining 12 per cent were independent or not affiliated with a political party. With regard to national election vote choice, 25 per cent of the Canadians stated that they normally vote Conservative, 35 per cent Liberal, 18 per cent NDP, 5 per cent Green Party, 1 per cent Bloc Québécois, and 7 per cent for other parties; 9 per cent stated that they do not vote.

5.4.3 Left/liberal versus right/conservative differences and attitude scale scores

As stated above, participants were asked to place themselves on a left/liberal versus right/conservative political spectrum. In order to assess the ability of this self-placement item to predict respondents' positions on the political attitudes scales created by factor analysis, and in so doing to provide evidence pertaining to whether global left/liberal versus right/conservative identifications are related to more specific attitude clusters, for each of the six political attitudes scales a one-way analysis of variance was conducted assessing the differences between left/liberal, centre, and right/conservative individuals.

Significant differences were found for each of the six scales. As reported in Table 2, the left/liberal group scored significantly higher than right/conservative people on the “environmentalism,” “economic equality,” “activist state,” and “minorities” scales, while the right/conservative group had significantly higher “religiosity/social conservatism” and “competition/business” scores. It seems that the self-placement item is meaningful in terms of its relationship to the created political attitude scales and hence that there is some correspondence between overall left/liberal versus right/conservative orientation and the more specific attitude dimensions.

6. Results of the Genetic Analyses

Within-twin pair correlations were computed for general interest in politics and self-placement on the left/liberal versus right/conservative spectrum, as well as the six political attitudes scales. They appear in Table 3. Although all but one of the MZ correlations are higher than the DZ correlations, the differences are small for some of them.

Univariate genetic analyses were conducted on the within-twin pair variance-covariance matrices using the Mx program. In these analyses, a score on a particular measure is expressed as a linear function of the three general factors mentioned above: additive genetic (A), common envi-
<table>
<thead>
<tr>
<th>Scale</th>
<th>Left/liberal M (SD)</th>
<th>Centre M (SD)</th>
<th>Right/conservative M (SD)</th>
<th>F</th>
<th>Significant post-hoc differences</th>
</tr>
</thead>
<tbody>
<tr>
<td>Religiosity/social conservatism</td>
<td>25.71 (8.22)</td>
<td>30.88 (7.25)</td>
<td>36.70 (9.68)</td>
<td>71.14&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Left/liberal—Centre Left/liberal—Right/conservative Centre—Right/conservative</td>
</tr>
<tr>
<td>Environmentalism</td>
<td>38.86 (7.35)</td>
<td>35.49 (6.54)</td>
<td>33.26 (8.46)</td>
<td>23.25&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Left/liberal—Centre Left/liberal—Right/conservative Centre—Right/conservative</td>
</tr>
<tr>
<td>Economic equality</td>
<td>36.94 (7.79)</td>
<td>35.86 (6.81)</td>
<td>32.73 (6.97)</td>
<td>14.24&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Left/liberal—Right/conservative Centre—Right/conservative</td>
</tr>
<tr>
<td>Activist state on social issues</td>
<td>26.01 (5.38)</td>
<td>27.15 (5.66)</td>
<td>24.42 (6.72)</td>
<td>8.09&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Left/liberal—Right/conservative Centre—Right/conservative</td>
</tr>
<tr>
<td>Competition/business</td>
<td>14.23 (3.27)</td>
<td>14.84 (2.57)</td>
<td>15.12 (3.07)</td>
<td>4.21&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Left/liberal—Right/conservative Centre—Right/conservative</td>
</tr>
<tr>
<td>Ethnic/racial minorities</td>
<td>9.28 (2.32)</td>
<td>8.78 (2.14)</td>
<td>7.98 (1.97)</td>
<td>15.56&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Left/liberal—Right/conservative Centre—Right/conservative</td>
</tr>
</tbody>
</table>

<sup>a</sup>p < .05, <sup>b</sup>p < .01 (two-tailed).
nvironment (C), and unshared environment (E). In conducting univariate genetic analyses, four models are tested (a full ACE model, an AE model, a CE model and an E only model). The model which has the best fit to the data (lowest chi-squared value, lowest chi-square per degree of freedom value and largest negative Akaike’s Information Criterion value) and which is most parsimonious is the one chosen as best fitting (Neale and Cardon, 1992). Once the best fitting model is decided, heritability ($a^2$), common environment ($c^2$) and unshared environment ($e^2$) values are computed from the standardized parameter estimates. These values estimate the proportion of the population variation in a particular characteristic that is attributable to each of the three factors.

The results of the univariate genetic analyses are also presented in Table 3. For both the general interest in politics question and left/liberal versus right/liberal spectrum item, the best fitting model was an AE model. Of the six political attitude scales, four (“religiosity/social conservativism,” “economic equality,” “competition/business,” and “minorities”) were found to be best fit by an AE model, with the remaining two scales best fit by a CE model.

A very high heritability estimate (62 per cent) was found for general interest in politics, with the remainder of the variation attributed to unshared environmental influences. Self-placement on the left/liberal versus right/liberal spectrum, as well as the “religiosity/social conservativism” and “economic equality” scales also showed high heritability components (57 per cent, 73 per cent and 58 per cent respectively), with the balance of the influence attributed to unshared environmental effects. The same pattern of substantial heritability with minimal common environmental effects was observed for the “competition/business” and

**Table 3**

<table>
<thead>
<tr>
<th>Attitude Scales</th>
<th>MZr</th>
<th>DZr</th>
<th>$a^2$ (95% CI)</th>
<th>$c^2$ (95% CI)</th>
<th>$e^2$ (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>General Interest</td>
<td>.63*</td>
<td>.37*</td>
<td>.62 (.52 to .70)</td>
<td>—</td>
<td>.38 (.30 to .48)</td>
</tr>
<tr>
<td>Left/liberal vs. Right/conservative</td>
<td>.60*</td>
<td>.07</td>
<td>.57 (.46 to .66)</td>
<td>—</td>
<td>.43 (.34 to .54)</td>
</tr>
<tr>
<td>Religiosity/social conservativism</td>
<td>.76*</td>
<td>.34*</td>
<td>.73 (.66 to .79)</td>
<td>—</td>
<td>.27 (.21 to .34)</td>
</tr>
<tr>
<td>Environmentalism</td>
<td>.59*</td>
<td>.42*</td>
<td>—</td>
<td>.56 (.46 to .66)</td>
<td>.44 (.34 to .54)</td>
</tr>
<tr>
<td>Economic equality</td>
<td>.57*</td>
<td>.39*</td>
<td>.58 (.46 to .67)</td>
<td>—</td>
<td>.42 (.33 to .54)</td>
</tr>
<tr>
<td>Activist state on social issues</td>
<td>.45*</td>
<td>.49*</td>
<td>—</td>
<td>.46 (.34 to .56)</td>
<td>.54 (.44 to .66)</td>
</tr>
<tr>
<td>Competition/business</td>
<td>.42*</td>
<td>.18</td>
<td>.41 (.28 to .52)</td>
<td>—</td>
<td>.59 (.48 to .72)</td>
</tr>
<tr>
<td>Ethnic/racial minorities</td>
<td>.52*</td>
<td>.27*</td>
<td>.52 (.40 to .61)</td>
<td>—</td>
<td>.48 (.38 to .60)</td>
</tr>
</tbody>
</table>

*p < .05, **p < .01 (two-tailed).

MZr = monozygotic correlation; DZr = dizygotic correlation; $a^2$ = additive genetic effects; $c^2$ = common environment effects; $e^2$ = unshared environment effects; 95% CI = 95% Confidence Interval.
“ethnic/racial minorities” scales (heritability estimates of 41 per cent and 52 per cent respectively), two additional facets of conservatism. These findings are consistent with those of Eaves and Eysenck (1974), who found high heritability for their R factor (“radicalism” versus “conservatism”), and with the substantial heritability scores that have been reported for social conservatism by researchers using versions of the W-P scale (for example, Martin et al., 1986; Truett et al., 1992; Eaves et al., 1997; Eaves et al., 1999; Bourchard et al., 2003; Alford et al., 2005; Hatemi, Alford et al., 2007). The findings reported here regarding “economic equality” are especially important in that, as noted, the studies based on the W-P scale do not assess economic conservatism. It appears that conservatism on both social and economic issues is heritable to a considerable degree.

Also examined were the items measuring how the respondents felt about the various Canadian national parties (with responses ranging from 1 “strongly dislike” to 7 “strongly like”). Listed in Table 4 are the within-twin pair correlations for those attitudes. Genetic effects were found for attitudes towards the Conservatives, Liberals, New Democrats and Bloc Québécois. No genetic effects are indicated for the Greens. The absence of heritability observed for feelings about the Green party parallels the finding of no significant heritability for the “environmentalism” scale noted above.

Finally, heritability tests were done for the Canadian respondents’ national party identification and normal vote choice in national elections, the latter being the only variable measuring political behaviour as opposed to attitudes or feelings. Because of small sample sizes for those who usually think of themselves as or vote for New Democrats,
Greens, or the Bloc, only respondents who normally identify themselves as or vote for Conservatives or Liberals were included in this analysis. Table 5 shows that genetic effects were found for both variables—50 per cent and 40 per cent for party identification, and 33 per cent and 36 per cent for normal vote choice, for Conservatives and Liberals respectively.

Discussion and Conclusions

The findings reviewed above indicate broad support for the hypotheses proposed in this study. All but two of the attitude measures in Table 3 had significant heritability components, as did all but one of the items in Table 4 measuring feelings toward the Canadian federal parties. All the measures of party identification and vote choice in Table 5 had significant heritabilities. Moreover, as predicted by the second hypothesis presented here, the two items in Table 5 measuring political behaviours (normal national vote choice) had heritabilities that were lower than the heritabilities observed for the attitude variables. It must be emphasized, however, that although these findings are consistent with the notion of evolved political temperaments, they should be regarded as preliminary.

The very high heritability estimate for general interest in politics, with the remainder of the variation attributed to unshared environmental influences, suggests that interest in things political is largely dispositional, and that socialization by family members has little effect on it. Among other things, this finding may inform discussions of how participatory the freest democracies can become—it may be that even with con-

---

**Table 5**

<table>
<thead>
<tr>
<th></th>
<th>MZr</th>
<th>DZr</th>
<th>a² (95% CI)</th>
<th>c² (95% CI)</th>
<th>e² (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>National Party Identification</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conservative</td>
<td>.51b</td>
<td>.12</td>
<td>.50 (.36 to .62)</td>
<td>—</td>
<td>.50 (.38 to .64)</td>
</tr>
<tr>
<td>Liberal</td>
<td>.40b</td>
<td>.13</td>
<td>.40 (.24 to .53)</td>
<td>—</td>
<td>.60 (.47 to .76)</td>
</tr>
<tr>
<td><strong>Normal National Vote Choice</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conservative</td>
<td>.33b</td>
<td>.14</td>
<td>.33 (.17 to .47)</td>
<td>—</td>
<td>.67 (.53 to .83)</td>
</tr>
<tr>
<td>Liberal</td>
<td>.36b</td>
<td>.15</td>
<td>.36 (.20 to .50)</td>
<td>—</td>
<td>.64 (.50 to .80)</td>
</tr>
</tbody>
</table>

*p < .05, b p < .01 (two-tailed).
MZr = monozygotic correlation; DZr = dizygotic correlation; a² = additive genetic effects; c² = common environment effects; e² = unique environment effects; 95% CI = 95% confidence interval.
considerable encouragement, active participation may not be forthcoming for some people because of the absence of a dispositional affinity for politics. For example, these findings may help to explain why, barring compulsory voting, voter turnout is usually well under 100 per cent even in hotly contested campaigns in democratic countries. Many social factors influence voter turnout, and political participation in general has many antecedents, but it may be time to add political temperament to the factors considered.

Similarly, the high heritability for general interest in politics may help to explain why there is considerable variation in the population on matters of ideological consistency, political knowledge and general political sophistication. Although many other factors are involved, such variation is to be expected if some people are predisposed to take an interest in politics while others are not.

The results reported above also indicate that dispositional factors may play a major role in determining one’s placement on the left or right side of the political spectrum, and that for the most part familial socialization has little to do with it. This may partially explain why people at one end of the political continuum react with such incredulity when encountering those at the opposite end, often thinking that their political opposites are stupid or crazy (or worse), when they may simply have different political dispositions. Similarly, these findings may help to explain why it is very difficult to politically “convert” a person from a right-wing to a left-wing orientation or vice versa, even after prolonged, reasoned argumentation.

The results presented above showed higher levels of heritability for feelings toward the Conservative and New Democratic parties compared to those for the Liberals. One possible explanation for this involves the idea that the Conservatives and the NDP are more ideologically oriented parties than the Liberals, although further research is needed to test that hypothesis. This study also showed higher heritability estimates for party identification among Conservatives as compared to Liberals, which may also reflect differences in ideological intensity between the two parties, but again more research is necessary.

Heritability analyses like the ones performed in this study are meant to be intellectual starting points rather than endpoints. They are designed to reveal to researchers possible avenues of further inquiry. The findings reported here point to a number of possibilities. More research on political temperaments is clearly called for, in particular to identify what their component parts might be and to examine how they might arise. Another potential area of research lies in examining the relationship between political temperaments and the personality factors identified by psychologists. And of course at the molecular level, attempts to indentify the genes, hormones and other biological elements associated with
political phenomena have only just begun. But the biggest challenge of all will be to integrate traditional approaches to the study of politics with bio-evolutionary perspectives.

Notes

1 The definitive statement on the rejection of bio-evolutionary models in sociology is van den Berghe (1990). Notable exceptions to the trend include van den Berghe (1973, 1978); Neilsen (1994); Blute (1997); Massey (2000); Sanderson (2001); and Baldus (2006). Some sociologists have embraced Darwinian evolutionary models in a macro or social evolutionary sense, for example Nolan and Lenski (2004), although they tend to analyze social, technological and cultural evolution and avoid strictly biological explanations; see also Dietz and others (1990). For a discussion of the limited influence of biological models in political science, see Hibbing and Smith (2007).

2 It may well be that many conventional political researchers consider genes to be relevant to politics, but very few make that assertion in print.


4 The American respondents were asked how much they liked the major US parties and which party they normally voted for in national elections, but because of small sample sizes they were excluded from the analysis.

5 For Canadians, the following introduction was used: “In politics, people sometimes talk of left (liberal) and right (conservative). Where would you place yourself on the scale below?” For Americans, the introduction was: “In politics, people sometimes talk of liberal and conservative. Where would you place yourself on the scale below?” The latter was not used for Canadians in order to avoid confusion between liberal and conservative attitudes and the federal parties bearing those names.

6 The item used was “Generally, how interested are you in national politics?” which employed a 7-point Likert response format going from “not interested at all” (1) to “very interested” (7).

7 Alford and others (2005) found only a modest heritability level (14 per cent) for party identification using American subjects. Hatemi, Medland and others (2007) found a moderate heritability component (24 per cent) for vote choice using Australian data.

References


The Origins of Political Attitudes


The Origins of Political Attitudes


Appendix A
The Six Factors

All items have a 7-point response scale (ranging from 1 “strongly disagree” to 7 “strongly agree”) unless otherwise indicated.

1) Religiosity/social conservatism

   a) How often do you usually attend religious services (e.g., church, mosque, synagogue, temple, etc.)? (5-point response scale)1
   b) Same-sex marriage (gay marriage) should not be permitted.1
   c) It should be possible for a woman to get a legal abortion if she wants it for any reason.2
   d) There should be no law against prayer in public schools.1
   e) Things would improve if more religious people became active in politics.1
f) It’s better to adjust one’s values to fit changing circumstances than to follow traditional values.  

3) Environmentalism

a) It is just fine to talk about protecting our environment, but as good citizens we must take a broader view. In order to have enough economic development and jobs for our people, we must be willing to accept some damage to our environment.  

b) If I were given a choice between having less pollution in the world or a strong national economy, I would pick a strong national economy.  

c) Our society has to try harder to protect the environment, even if that means putting some loggers or factory workers out of a job.  

d) Global warming is a scientific fact, not an opinion.  

e) Government regulation of business is necessary to keep industry from becoming too powerful.  

f) It is more important to protect the environment than to create jobs.  

g) A lumber company that spends millions for a piece of forest land has the right to cut down enough trees to protect its investment.  

h) Our society should do more to protect the environment, even if that means raising the price of gasoline.  

4) Economic equality

a) Under a fair economic system, all people would earn about the same.  

b) People who don’t get ahead in life should blame themselves, not the system.  

c) Governments should do more to reduce the gap between the rich and the poor.  

d) Economic inequality continues to exist because it benefits the rich and powerful.  

e) Personal income should not be determined solely by one’s work. Rather, everybody should get what he/she needs to provide a decent life for his/her family.  

f) In this country, most poor people are poor because of circumstances beyond their control.  

g) In this country, any person who is able and willing to work hard has a good chance of economic success.  

h) If incomes were more equal, nothing would motivate people to work hard.
Working people in this country do not get a fair share of what they produce.5

4) **Activist state on social issues**
   a) Discrimination makes it very difficult for women to get good jobs.12
   b) There should be a law against making sexist remarks in public.1
   c) We need stricter laws against sexual harassment.1
   d) It is the government’s responsibility to provide a job for everyone who wants one.13
   e) There should be a law against making anti-religious statements in public.1
   f) Only the police and the military should be allowed to have guns.7

5) **Competition/business**
   a) Competition, whether in school, work or business, leads to better performance and desire for excellence.5
   b) Our society’s top business people should be held up to our young people as models to be admired and imitated.5
   c) Competition, whether in school, work or business, is often wasteful and destructive.5

6) **Ethnic/racial minorities**
   a) The more immigrants we get, the better our country becomes.1
   b) How much do you think should be done for racial minorities in this country? (5-point response scale)7

---

1Item devised by the authors of this study.
2Item taken from the 2002 American General Social Survey.
3Item adapted from John Hibbing (2006, personal correspondence).
5Item taken from McClosky and Zaller (1984).
6Item taken from the 2006 Canadian Election Study, Post Election Survey.
7Item taken from the 2004 Canadian Election Study, Pre-Election Questionnaire.
8Item taken from the 2000 American General Social Survey.
9Item taken from Markus (1990).
10Item taken from Mirels and Garrett (1971).
11Item taken from Kluegel and Smith (1986).
12Item taken from the 2004 Canadian Election Study, Mail Back Questionnaire.
13Item taken from the 2006 American General Social Survey.
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.
Yet in spite of this everything-but-the-kitchen-sink approach, these models usually fit poorly to the data (Matsusaka and Paldà 1999). For example, one promi-
nient model includes 32 variables but accounts for only 31% of the variance in turnout (Plutzer 2002). More-
over, the theories underlying these empirical models
typically ignore genetic or biological sources of vari-
ation. Although political scientists are unlikely to op-
pose the idea that biology plays a role in political partic-
ipation, in print we hardly ever include this as a pos-
sibility. For example, the large literature on the role
of parents in voter turnout nearly always suggests that
the link between parent and child is the result of the
transmission of norms rather than the transmission of
genes (Plutzer 2002). As a result, our best work on the
subject frequently leaves the impression that political
participation is determined exclusively by environ-
mental factors.

Recently, social scientists have learned that vari-
ation in basic political attitudes like liberalism and
conservatism can be attributed to both genes and
environment (Martin, et al. 1986; Alford, Funk, and
Hibbing 2005; Eaves and Hatemi 2008; Hannagan
and Hatemi 2008), even as early as adolescence
(Abrahamson, Baker, & Caspi, 2002). While the choice
of a particular candidate or party does not appear to
be heritable (Alford, Funk, and Hibbing 2005; Hatemi
et al. 2007), it remains an open question whether or not
the act of voting (or, more broadly, any act of political
participation) is heritable. Given the difficulty scholars
have had in explaining participation solely based on
environmental accounts, we hypothesize that a signifi-
cant portion of the variation in voter turnout behavior
can be attributed to genetic factors.

Although we are not the first to suggest a link be-
tween genes and political participation, this study is the
first attempt to test the idea empirically. Some early
work studied the importance of personality in political
participation, but this literature focused exclusively on
environmental factors, asserting that people who are
reared in similar ways will have similar personalities
(Lane 1959; Levinson 1958) or that the role of person-
ality was to mediate social influences on participation
(Krause et al. 1970). Other early work explored the
importance of adolescent socialization in the develop-
ment of political behaviors, but these scholars never
considered the genetic link between parent and child.
Merelman (1971) addressed this shortcoming, arguing
that both genes and environment are probably impor-
tant. In fact, he explicitly recommended the use of
twin studies to investigate the heritability of political
participation. In his view, the main reason heritability
had been ignored was due to the difficulty in statistical
design and testing:

"[T]his natural tendency to examine one environmental
factor after another ad infinitum does a genetic explana-
tion something of an injustice. The problem is that while we can
examine environmental variables directly, we can usually
only infer genetic effects, and so our natural tendency is to
slight the latter perspective. In short, our procedures, fol-
lowing the line of least methodological resistance, impinge
heavily upon our theoretical perspectives." (1044)

In spite of Merelman’s exhortation, genetic studies of
participation were not forthcoming. Scholars con-
tinued to focus on personality factors underlying par-
ticipation like efficacy (Finkel 1985) and self-esteem
(Sears 1987) without mentioning the fact that these fac-
tors may themselves be heritable. A few scholars have
consistently argued on general principle that genes
must play a role in political behaviors like participa-
tion (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) monozy-
gotic (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 compa-
rable environments (more on this assumption below),
then we can use these concordances to estimate explicit-
ly the proportion of the overall variance attributed to
 genetic, shared environmental, and unshared environ-
mental 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 general-
izable to a non-twin population.

The twin study design has been shown to be an ex-
tremely powerful tool for identifying the relative de-
gree to which genetic and environmental factors in-
fluence an observed outcome (Evans, Gillespie, and
Martin 2002; Neale and Cardon 1992). The basic twin
model assumes that the variance in observed behavior
can be partitioned into additive genetic factors (A), and environmental factors which are shared or common to co-twins (C), and unshared environmental (E). This is the so-called ACE model. The role of genotype and environment are not measured directly but their influence is inferred through their effects on the covariances between twin siblings (Neale and Cardon 1992). No observed covariates are needed in the model because the degree to which they contribute to variance is a part of one of three variance components (A, C, and E). More formally, these components are derived from known relationships between three observed statistics (Evans, Gillespie, and Martin 2002):

\[
\sigma_p^2 = \sigma_A^2 + \sigma_C^2 + \sigma_E^2
\]

\[
COV_{MZ} = \sigma_A^2
\]

\[
COV_{DZ} = 1/2\sigma_A^2 + \sigma_C^2
\]

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). The

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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></th>
<th>DZ Twins</th>
<th></th>
<th>Difference of Means Test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Standard Error</td>
<td>Mean</td>
<td>Standard Error</td>
<td>p-value</td>
</tr>
<tr>
<td>Voter file data</td>
<td></td>
<td></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>
<td>0.03</td>
<td>0.79</td>
</tr>
<tr>
<td>Democrat</td>
<td>0.51</td>
<td>0.05</td>
<td>0.52</td>
<td>0.05</td>
<td>0.84</td>
</tr>
<tr>
<td>Republican</td>
<td>0.24</td>
<td>0.04</td>
<td>0.25</td>
<td>0.05</td>
<td>0.83</td>
</tr>
<tr>
<td>Third Party</td>
<td>0.05</td>
<td>0.02</td>
<td>0.05</td>
<td>0.02</td>
<td>0.88</td>
</tr>
<tr>
<td>Age</td>
<td>36.8</td>
<td>2.5</td>
<td>33.6</td>
<td>2.8</td>
<td>0.25</td>
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<tr>
<td>Same Address</td>
<td>0.47</td>
<td>0.07</td>
<td>0.52</td>
<td>0.08</td>
<td>0.69</td>
</tr>
<tr>
<td>Same Postal Code</td>
<td>0.54</td>
<td>0.07</td>
<td>0.64</td>
<td>0.07</td>
<td>0.84</td>
</tr>
<tr>
<td>Zillow.com data</td>
<td></td>
<td></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>
<td>0.05</td>
<td>0.89</td>
</tr>
<tr>
<td>House Value</td>
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<td>40,577</td>
<td>784,421</td>
<td>49,412</td>
<td>0.49</td>
</tr>
<tr>
<td>House Square Feet</td>
<td>2148</td>
<td>111</td>
<td>2106</td>
<td>137</td>
<td>0.86</td>
</tr>
<tr>
<td>Lot Square Feet</td>
<td>8062</td>
<td>392</td>
<td>9117</td>
<td>1014</td>
<td>0.15</td>
</tr>
<tr>
<td>SCTP data</td>
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<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>
<td>0.55</td>
<td>0.72</td>
</tr>
<tr>
<td>Extraversion</td>
<td>0.66</td>
<td>0.04</td>
<td>0.71</td>
<td>0.04</td>
<td>0.38</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>0.43</td>
<td>0.03</td>
<td>0.43</td>
<td>0.05</td>
<td>0.92</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>
<tr>
<th></th>
<th></th>
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<th></th>
<th></th>
<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>0.66</td>
<td>0.44</td>
<td>0.84</td>
<td>0.46</td>
</tr>
<tr>
<td>(N = 396)</td>
<td>(0.25)</td>
<td>(0.18)</td>
<td>(0.23)</td>
<td>(0.25)</td>
<td>(0.22)</td>
<td>(0.25)</td>
<td>(0.13)</td>
<td>(0.25)</td>
</tr>
<tr>
<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>
<td>0.49</td>
</tr>
<tr>
<td>(N = 247)</td>
<td>(0.25)</td>
<td>(0.19)</td>
<td>(0.23)</td>
<td>(0.24)</td>
<td>(0.21)</td>
<td>(0.24)</td>
<td>(0.12)</td>
<td>(0.25)</td>
</tr>
<tr>
<td>DZ twins</td>
<td>0.53</td>
<td>0.79</td>
<td>0.37</td>
<td>0.56</td>
<td>0.62</td>
<td>0.46</td>
<td>0.80</td>
<td>0.42</td>
</tr>
<tr>
<td>(N = 149)</td>
<td>(0.25)</td>
<td>(0.17)</td>
<td>(0.23)</td>
<td>(0.25)</td>
<td>(0.24)</td>
<td>(0.25)</td>
<td>(0.16)</td>
<td>(0.24)</td>
</tr>
<tr>
<td>Population</td>
<td>0.48</td>
<td>0.68</td>
<td>0.26</td>
<td>0.45</td>
<td>0.55</td>
<td>0.38</td>
<td>0.79</td>
<td>0.47</td>
</tr>
<tr>
<td></td>
<td>(0.25)</td>
<td>(0.22)</td>
<td>(0.19)</td>
<td>(0.25)</td>
<td>(0.25)</td>
<td>(0.24)</td>
<td>(0.17)</td>
<td>(0.25)</td>
</tr>
</tbody>
</table>

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. 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:

\[
\tau_{ij}^{MZ} = A_i + C_i + E_{ij},
\]

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:

\[
\tau_{ij}^{DZ} = A_{1i} + A_{2ij} + C_i + E_{ij},
\]

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

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\( \alpha = 0.78 \) reveals that these eight elections are reliable measures of a single scalar latent value for the propensity to vote.

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\[ \text{238} \]
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 based on a relatively small sample of 250 families yielded variance component point estimates and credible intervals that exhibited no significant bias.

Replicating the methods used in this literature, we assume that our unobserved random effects are normally distributed: $A \sim N(0, \sigma_A^2)$, $A_1 \sim N(0, \sigma_A^2/2)$, $A_2 \sim N(0, \sigma_A^2/2)$, $C \sim N(0, \sigma_C^2)$, and $E \sim N(0, \sigma_E^2)$. 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 shared 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 $\sigma_E^2 = 1$ and then use the estimates of $\sigma_A^2$ and $\sigma_C^2$ to derive the proportion of variance generated by each factor. This procedure generates estimates for the influence of heritability $h^2 = c_A^2/(c_A^2 + c_C^2 + c_E^2)$, common environment $c^2 = c_C^2/(c_A^2 + c_C^2 + c_E^2)$, and the unshared environment $c^e = c_E^2/(c_A^2 + c_C^2 + c_E^2)$. 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 $c^e$ 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 $\sigma_C^2 = 0$. 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 $\sigma_A^2$ and $\sigma_C^2$ 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.

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7 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).

8 The choice of normal distributions is for convenience—they imply $r$ is normally distributed as well since the sum of two normally distributed random variables is also distributed normal.

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.

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). In order to summarize the discrepancy between the model and data, a relevant measure must first be chosen. Using this measure, a posterior predictive $p$-value may be calculated to evaluate the fit of the model to the observed data (Gelman et al. 2004). Specifically, the predictive $p$-value is the proportion of the replicated datasets for which the discrepancy measure equals or exceeds its realized value. Large and systematic differences between the replicated and observed data, resulting in $p$-values close to zero or one, suggest a poor fitting model. In Table A-2 in the Appendix we compare the predicted percentage of individuals voting in zero to eight elections to the actual distribution. None of the discrepancies are statistically significant, indicating reasonable fit for the overall model (Gelman et al. 2004).

To test the sensitivity of our method we also employed a traditional structural equation model (SEM) to fit the data, using Mplus to estimate the A, C, and E components of variance in a common factor underlying 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

![FIGURE 2. Heritability of Voter Turnout in Los Angeles](image)

11 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 turnover, 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 effect 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 (Hopper 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 to find 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 election?...”
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 ($\alpha = 0.61$) reveals that it is reasonable to include these variables in a model in which a single scalar latent variable for participation is being estimated (see Verba, Schlozman, and Brady 1995, who report a similar $\alpha$ 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 ($T_{ij} = 1$) or abstain ($T_{ij} = 0$) in the election. As in the Los Angeles voting model, the observed phenotypes are dichotomous variables and we assume $\tau$ 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 $\delta_1 = 1$ 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.
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>
<thead>
<tr>
<th>TABLE A1. Summary of Model Results</th>
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<tr>
<td>Heritability</td>
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<td>h²</td>
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<td>Los Angeles Turnout</td>
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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 attributable to heritability (h²), common environment (c²), and unshared environment (e²), 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>
<thead>
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<th>TABLE A2. Posterior Predictive Checks</th>
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<td>Discrepancy Measure: % Voting in Value</td>
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<td>No elections</td>
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<td>One election</td>
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<td>Two elections</td>
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<td>Eight elections</td>
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REFERENCES


Two Genes Predict Voter Turnout

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Fowler, Baker, and Dawes (2008) recently showed in two independent studies of twins that voter turnout has very high heritability. Here we investigate two specific genes that may contribute to variation in voting behavior. Using data from the National Longitudinal Study of Adolescent Health, we show that individuals with a polymorphism of the MAOA gene are significantly more likely to have voted in the 2004 presidential election. We also find evidence that an association between a polymorphism of the 5HTT gene and voter turnout is moderated by religious attendance. These are the first results ever to link specific genes to political behavior.

Social scientists have shown that basic political attitudes like liberalism and conservatism are likely to be heritable (Alford, Funk, and Hibbing 2005, Hatemi et al. 2007). While the choice of a particular candidate or party does not appear to be heritable, a significant proportion of the variation in the decision to participate in politics can be attributed to genetic factors. Fowler, Baker, and Dawes (2008) recently studied the voting behavior of two populations of twins and showed that heritability accounted for 53% of the variation in validated turnout of those living in Los Angeles county and 72% of the self-reported turnout in a nationally representative sample of young adults. They also showed that heritability accounted for 60% of the variation in a general index of political participation, including contributing to campaigns, running for office, volunteering for political organizations, and attending protests. These results were the first to suggest that humans exhibit inherent variability in their willingness to participate in politics.

However, these initial results based on twin studies beg the question “which genes?” The natural place to start the search for such genes is among those that have already been shown to account for variation in social behavior. And among these, MAOA and 5HTT are prime candidates. These two genes transcribe neurochemicals that exert a strong influence on the serotonin system in parts of the brain that regulate fear, trust, and social interaction (Bertolino et al. 2005; Eisenberger et al. 2007; Hariri et al. 2002; Hariri et al. 2005; Heinz et al. 2005; Meyer-Lindenberg et al. 2006). MAOA and 5HTT have been studied for more than 20 years, and much is known about the way different versions of their genes regulate transcription, metabolism, and signal transfers between neurons, all of which have an effect on social interactions (Craig 2007). In particular, the less transcriptionally efficient alleles of these genes have been associated with a variety of antisocial behaviors (Rhee and Waldman 2002).

In this article, we hypothesize that people with more transcriptionally efficient alleles of the MAOA and 5HTT genes are more likely to vote. An association between a gene and political behavior may also be moderated by environmental factors. This phenomenon is known as a gene-environment (GxE) interaction (Shanahan and Hofer 2005). We therefore also hypothesize that an association between each of these genes and voting may be moderated by social activity. Using data from the National Longitudinal Study of Adolescent Health, we conduct gene and gene-environment association tests on the relationship between turnout and MAOA and 5HTT. The results show that both genes are significantly associated with the decision to vote. Moreover, the association between 5HTT and turnout is moderated by exposure to religious social activity. These findings have important implications for how we both model and measure political interactions.

Past Work on the Genetic Basis of Political Participation

Although we are not the first to suggest a link between genes and political participation, this study...
is the first to investigate an association between specific genes and political behavior. Early work studied the importance of personality in political participation, but this literature focused exclusively on environmental factors, asserting that people who are reared in similar ways will have similar personalities (Lane 1959; Levinson 1958) or that the role of personality is to mediate social influences on participation (Krause et al. 1970). Additional earlier studies focused on the importance of adolescent socialization in the development of political behaviors, but these scholars never considered the role of genes in the link between parent and child. Merelman (1971) explicitly addressed this shortcoming, arguing that both genes and environment are probably important. He lamented the fact that genetic explanations had been ignored by social scientists:

“[T]his natural tendency to examine one environmental factor after another ad infinitum does a genetic explanation something of an injustice. The problem is that while we can examine environmental variables directly, we can usually only infer genetic effects, and so our natural tendency is to slight the latter perspective. In short, our procedures, following the line of least methodological resistance, impinge heavily upon our theoretical perspectives.” (1044)

In spite of Merelman’s early call for attention, 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 considering the fact that these factors may be heritable. A few political scientists have 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 left the work of testing their hypotheses to others.

A wide range of studies have already shown that variation in prosocial personality and behavior can be attributed to genes (McGue, Bacon, and Lykken 1993; Rushton et al. 1986; Scourfield et al. 2004). This literature suggests that innate dispositions play an important role in an individual’s willingness to participate in social activities or to engage in acts that primarily benefit others. Meanwhile, a growing number of observational studies, theoretical models, and laboratory experiments suggest that prosocial attitudes and behavior are important factors for explaining voter turnout and political participation. For example, Knack (1991) creates an index of “social altruism” from questions about charity, volunteer work, and community involvement on the 1991 NES Pilot Study and finds a positive relationship between the index and turnout. Similarly, Jankowski (2007) finds a relationship between turnout and humanitarian norms from questions on the 1995 NES Pilot Study. Edlin, Gelman, and Kaplan (2007) show that a variety of aggregate features of turnout can be easily explained by incorporating prosocial preferences into the decision-theoretic calculus of voting, and Jankowski (2002) shows that this reasoning extends to a game-theoretic model. Finally, experimental studies utilizing dictator games to measure revealed social preferences show that individuals who are more willing to engage in costly giving to others are also more likely to vote (Fowler 2006a) and participate in politics (Dawes and Fowler 2007, Fowler and Kam 2007).

Thus, we hypothesize that genes may influence voting and political participation because they influence a generalized tendency to engage in prosocial behavior via their functional role in neurochemical processes. Although Fowler, Baker, and Dawes (2008) have already shown that a large fraction of the variation in voter turnout and political participation can be attributed to genetic factors, to date no specific genes have been identified in this process. It is crucial to point out at the outset that we cannot test, given our data, the potential causal pathways we suggest. Therefore, the goal of this study is to show association rather than causality.

Some Basic Genetics Concepts

Genes are distinct regions of human DNA that form the blueprint for molecules that regulate the development and function of the human body. There are an estimated 25,000 genes (most of which exist in multiple copies) in the 46 chains, or chromosomes, that make up all human DNA. Almost all human cells contain the same inherited DNA chains that are fixed from the moment of conception. This is an important point for social scientists. Since genes are fixed, they represent the purest measure of biological inheritance, virtually unaffected by environment and able to be collected at any point throughout a person’s life.

At conception individuals inherit one-half of their DNA from each parent, with one copy of each gene coming from the mother and one copy from the father. Some genes come in different versions, known as “alleles”—for example, sickle cell disease results from a particular allele coding for abnormal rather than normal hemoglobin. Each parent has two
For social behavior, this means focusing on genes that influence related behaviors or processes in the body. To determine whether genes affect voting behavior, we chose two candidate genes that have already received a great deal of attention for their association with antisocial behavior. These genes, MAOA and 5HTT, are critical to the metabolism of serotonin in the brain. As shown in Figure 1, serotonin is a chemical that is released when a neuron “fires” and sensed by a receptor on the receiving neuron, passing an electric potential across a gap called a nerve synapse (the nerve that fires is on the “presynaptic” side of the gap). Signals are carried throughout the body by the sequential firing of one neuron after another across these synapses. When an individual experiences stress, it causes increased neuron activity, stimulating the release of excess serotonin into the gaps between the synapses (Chaouloff, Berton, and Mormede 1999). If serotonin remains outside the cells, it can oxidize into a toxin that kills both the presynaptic and postsynaptic neurons. The body’s homeostatic response to this excess serotonin is to reabsorb it into the presynaptic neuron via a transporter in the cell wall, called 5HTT. Once the “reuptake” of serotonin is complete and it is back inside the neuron, an enzyme called monoamine oxidase A (MAOA) degrades the serotonin so that its components can be reabsorbed in the cell. The genes responsible for transcribing 5HTT and MAOA are eponymous—the 5HTT gene produces 5HTT and the MAOA gene produces MAOA.

Animal studies indicate that the serotonin system has an important effect on social behavior. Rhesus macaque monkeys with impaired serotonin metabolisms are impulsive and aggressive in response to social stressors (Kraemer et al. 1989) and studies of rodents show that acute emotional stress affects the way MAOA breaks down serotonin in several areas of the brain (Popova, Voitenko, and Maslova 1989; Virkkunen et al. 1995). In mice, social stress increases transcription of both MAOA and 5HTT (Filipenko et al. 2002) and knock-out studies that eliminate the MAOA gene in subjects cause enzymatic activity to come to a complete halt (Cases et al. 1995). In monkeys, 5HTT is densely concentrated in the output regions of the amygdala, which affects fear recognition (O’Rourke and Fudge 2006) and MAOA has been shown to alter the structure of the brain in mice (Cases et al. 1996). This evidence suggests that any deficiency in the genes that regulate serotonin metabolism will have a direct effect on the brain that tends to reduce the ability to process and respond to

Serotonin, Genes, and Social Behavior

Twin studies have already established that genetic factors account for a significant proportion of the variation in antisocial behaviors (Rhee and Waldman 2002), including substance abuse, impulsivity, criminality, precocious sexuality, and a combination of these behaviors called antisocial personality disorder (ASPD). However, twin studies cannot establish which genes are implicated. It is likely that dozens, if not hundreds of genes influence sociability (Mackay 2001; Plomin 2008). As a result, scientists typically start with “candidate” genes that are known to influence related behaviors or processes in the body. For social behavior, this means focusing on genes that affect brain development, neurotransmitter synthesis and reception, hormone regulation, and transcription factors (Damberg et al. 2001).
social stress. These effects have been linked specifically to a genetic polymorphism in monkeys that is closely related to that observed in humans (Newman, et al. 2005, Suomi 2003). There is also strong evidence that the serotonin system affects complex social traits in humans (Balciuniene and Jazin 2001), and 5HTT and MAOA frequently serve as targets for antidepressants and illegal recreational drugs (Craig 2007, Livingston and Livingston 1996).

The 5HTT gene contains a 44 base-pair variable-number tandem repeat (VNTR) polymorphism² in the promoter region³ that is believed to be responsible for variation in transcriptional activity. The transcriptional efficiency of the “long” version of this allele is associated with a much higher basal activity than the shorter allele (Lesch et al. 1996; Little et al. 1998). MAOA has a 30 base-pair VNTR polymorphism located in the promoter region. The “high” version of this polymorphism significantly increases the transcriptional efficiency of MAOA (Denney, Koch, and Craig 1999; Denney et al. 1994; Sabol, Hu, and Hamer 1998). The less transcriptionally efficient alleles of both 5HTT and MAOA have been linked to antisocial behavior (Vanukov et al. 1995; Contini et al. 2006; Domsche et al. 2005; Hsu et al. 1996; Lawson et al. 2003; Saito et al. 2002; Samochowiec, Lesch et al. 1999; Schmidt et al. 2000) which appears to be mediated by certain parts of the brain. For example, the development of the amygdala and orbitofrontal cortex has been linked to a small genetic locus which contains the gene for MAOA (Good, Lawrence, and Thomas 2003). Furthermore, a number of studies show that the amygdala becomes “hypersensitive” during the presentation of aversive or threatening social stimuli in individuals with either the short 5HTT allele (Bertolino et al. 2005; Hariri et al. 2002; Hariri et al. 2005; Heinz et al. 2005) or the low MAOA allele (Meyer-Lindenberg et al. 2006). Eisenberger et al. (2007) report similar results, noting that the link to antisocial behavior results from an increased sensitivity to negative socioemotional experiences (though in their study they claim the effect is mediated via the dorsal anterior cingulate cortex).

Not all studies show a direct relationship between these polymorphisms and behavior. Instead, developmental or concurrent environments may moderate an association between genes and observed social behavior. A gene-environment (GxE) interaction has been identified in many cases for antisocial behavior (Caspi et al. 2002; Foley et al. 2004; Haberstick et al. 2005; Kim-Cohen et al. 2006; Nilsson et al. 2006), the most famous of which is the Caspi et al. (2002) paper. This work shows that exposure to stressors like child abuse at early developmental stages may interact with the low MAOA polymorphism resulting in antisocial behavior later in life. This is an important point—in these studies the gene itself was not associated with the behavior. Rather, it was the combination of both gene and environment that yielded a significant association.

Two Hypotheses for Genes and Turnout

A growing literature suggests that voter turnout is a prosocial behavior that is strongly influenced by other-regarding preferences (Edlin, Gelman, and Kaplan 2007; Fowler 2006a; Fowler and Kam 2007; Jankowski 2002, 2007). Given that polymorphisms of MAOA and 5HTT appear to influence antisocial behavior, we therefore hypothesize that they will also be associated with voting behavior. One difficulty of the voting experience is that one’s preferred candidates sometimes lose. This loss has been theorized to reduce future motivations to vote (Bendor, Diermeier, and Ting 2003; Fowler 2006b) and Kanazawa (1998) has even shown empirically that turnout declines among those whose favorite candidates lost the previous election. In addition, people may prospectively consider how they will feel about a loss before

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²A VNTR polymorphism is a repeated segment of DNA that varies among individuals in a population.

³A promoter region is the regulatory region of DNA that tells transcription enzymes where to begin. These promoter regions typically lie upstream from the genes they control.
deciding whether or not to vote, or whether they will even pay attention to the election. Those who are overly sensitive to social conflict may choose to stay home and ignore politics, while less sensitive individuals will not take the potential emotional stress caused by the loss of their favorite candidates into consideration. Thus, we expect individuals with the “high” MAOA polymorphism and “long” 5HTT polymorphism will be more likely to turn out to vote.

However, an association between either MAOA and 5HTT and voting may not be direct. Instead, an association between a gene and turnout may be moderated by environmental factors. A vast literature on turnout suggests the importance of voter mobilization efforts (Wielhouwer and Lockerbie 1994), religious group activity (Cassel 1999), and other kinds of social contacts that have an influence on political participation (Huckfeldt 1979, Verba, Schlozman, and Brady 1995). Religious group activity in particular has been singled out as one of the strongest predictors of voter turnout, even more so than socioeconomic status (Olsen 1972; Sallach, Babchuk, and Booth 1972). However, scholars have had difficulty interpreting this association. Religious groups might stimulate political activity directly or as byproducts of their tendency to increase civic skills, political interest, feelings of efficacy, access to political information, and a sense of civic duty. Testing all of these possible explanations, Cassel (1999) suggests that the main reason for the association is that religious groups build a sense of belonging to a larger community. However, it may not be possible to build such a sense in people who are too averse to social conflict, since they will resist appeals to become involved. We therefore hypothesize that MAOA and 5HTT, when interacted with religious group activity, may be significantly associated with turnout. Specifically, individuals who are actively involved in their religious organizations and who have the “high” MAOA allele or the “long” 5HTT allele will be more likely to vote than others.

Data

All of our analysis is based on individual-level genetic and survey data collected as part of The National Longitudinal Study of Adolescent Health (Add Health). Add Health is a study that explores the causes of health-related behavior of adolescents in grades 7 through 12 and their outcomes in young adulthood. The first wave of the Add Health study (1994–95) selected 80 high schools from a sampling frame of 26,666. The schools were selected based on their size, school type, census region, level of urbanization, and percent of the population that was white. Participating high schools were asked to identify junior high or middle schools that served as feeder schools to their school. This resulted in the participation of 145 middle, junior high, and high schools. From those schools, 90,118 students completed a 45-minute questionnaire, and each school was asked to complete at least one School Administrator questionnaire. This process generated descriptive information about each student, the educational setting, and the environment of the school. From these respondents, a core random sample of 12,105 adolescents in grades 7–12 were drawn plus several over-samples, totaling more than 27,000 adolescents. These students and their parents were administered in-home surveys in the first wave. Wave II (1996) was comprised of another set of in-home interviews of more than 15,000 students from the Wave I sample and a follow-up telephone survey of the school administrators. Finally, Wave III (2001–2002) consisted of an in-home interview, six years later, of 15,170 Wave I participants. The result of this sampling design is that Add Health is a nationally representative study. 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 represent all regions of the country: the Northeast makes up 17% of the sample, the South 27%, the Midwest 19%, and the West 17%.

In Wave I of the Add Health study, researchers created a genetically informative sample of sibling pairs based on a screening of the in-school sample of 90,114 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 Rower 1998). Allelic information for six genetic markers are available for 2,574 individuals as part of Wave III, including markers that identify alleles of MAOA and 5HTT. Details of the DNA collection and genotyping process

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4A breakdown for those providing DNA samples is presented in the appendix.

5We do not use the Add Health sampling weights because more than a third of subjects in the genetic sample had a co-sibling that was interviewed as part of Wave III but not as part of the original Wave I sampling frame (Lessem et al. 2006). Therefore, sampling weights could not be constructed for these subjects. Limiting our analysis to only individuals in the genetic sample for which weights could be determined would greatly reduce statistical power.
Geneic Association

Genetic association studies test whether an allele or genotype occurs more frequently within a group exhibiting a particular trait than those without the trait. For example, is the frequency of a particular allele or genotype higher among voters than non-voters? However, a significant association can mean one of three things: (1) The allele itself influences voting behavior; (2) the allele is in “linkage disequilibrium” with an allele at another locus that influences voting; or (3) the observed association is a false positive signal due to population stratification.

Population stratification occurs because groups may have different allele frequencies due to their genetic ancestry. Turnout in these groups may be the product of their environments, alleles other than the one of interest, or some unobserved reason. For example, two groups may not have mixed in the past for cultural reasons. Through the process of natural selection or genetic drift these groups may develop different frequencies of a particular allele. At the same time, the two groups may also develop divergent behaviors that are not influenced by the allele but completely by the environment in which they live. Once these two groups mix in a larger population, simply comparing the frequency of the allele to the observed behavior would lead to a spurious association.

There are two main research designs employed in association studies, case-control designs, and family-based designs. Case-control designs compare the frequency of alleles or genotypes among subjects that exhibit a trait of interest to subjects who do not. As a result, case-control designs are vulnerable to population stratification if either group is especially prone to selection effects. A typical way to control for this problem is to include controls for the race/ethnicity of the subject or to limit the analysis to a specific racial or ethnic group. Family-based designs eliminate the problem of population stratification by using family members, such as parents or siblings, as controls. Tests using family data compare whether offspring exhibiting the trait receive a risk allele from their parents more often than would be expected by chance. This design is very powerful in minimizing...
type I error but also suffers from much lower power in detecting a true association. Xu and Shete (2006) show, based on extensive simulation work, that a case-control association study using a mixed-effects logistic regression outperforms family-based designs in detecting an association while at the same time effectively limiting type I error.

Model

To test for genetic association we employ a mixed-effects logistic regression model (Guo and Zhao 2000; Xu and Shete 2006):

\[ P[Y_{ij} = 1|Z_{kij}, U_j] = \logit(\beta_0 + \beta_G G_{ij} + \beta_E E_{ij} + \beta_{GxE}(G_{ij} \ast E_{ij}) + \beta_K Z_{kij} + U_j) \]

where \( i \) and \( j \) index subject and family respectively. For the MAOA gene, \( G = 1 \) if the subject’s genotype is HH, and \( G = 0 \) for genotypes HI or II (where H represents having a copy of a 336, 351, or 381 base-pair “high” allele, and I represents having a copy of a 291 or 321 base-pair “low” allele). For the 5HTT gene, \( G = 1 \) if the subject’s genotype is LL or Ls and \( G = 0 \) if the subject’s genotype is ss (where L represents having a copy of the 528 base-pair “long” allele and s represents having a copy of the 484 base-pair “short” allele). The variable \( E \) is an environmental variable we believe moderates the influence of the genotype on voting behavior. We test one such variable, regular attendance of religious services. \( Z \) is a matrix of variables to control for underlying population structure of the Add Health samples as well as potentially mediating factors like age, gender, income, and education that have been found to significantly influence turnout. Finally, the variable \( U \) is a family random effect that controls for potential genetic and environmental correlation among family members.

To control the effects of the underlying population structure, we include indicator variables for whether a subject self-reported as black, Hispanic, Asian, or Native American (base category is white). Following the policy of the United States Census, Add Health allows respondents to mark more than one race. Since this complicates the ability to control for stratification, we exclude these individuals (\( N = 117 \)), but supplementary analysis including them yields substantively identical results. We also exclude from the data analysis noncitizens and people less than 18 years of age on Election Day since they are not legally eligible to vote. This leaves us with a sample size of 2,329 individuals.

The odds ratio of \( \beta_G \) is an individual’s odds of voting if he or she is HH genotype for the MAOA gene compared to an individual with an HI or II genotype. A significant odds ratio means that lacking a “short” allele is associated with higher turnout when compared to having at least one “short” allele. For the 5HTT gene, the odds ratio of \( \beta_G \) is an individual’s odds of voting given he or she has at least one “long” allele (LL or Ls) compared to having no long alleles (ss). Therefore, a significant odds ratio implies that having a “long” 5HTT allele is associated with the decision to vote. Finally, the odds ratio of \( \beta_{GxE} \) is whether having a specific genotype combined with being exposed to an environmental effect influences turnout behavior, even after controlling for both main effects.

Results

Table 1 shows the results of several specifications of the models to test the hypothesis that genes are associated with voter turnout and whether the association is moderated by religious service attendance. Each of these specifications includes variables for age, gender, and race to control for population stratification. Model 1 shows that the “high” allele of MAOA is significantly associated with increased voter turnout (\( p = 0.03 \)). This model suggests that the odds of a person with the “high” version of the MAOA gene voting are 1.26 times greater than that of a person with the “low” version. Model 2 rejects the hypothesized relationship between 5HTT and voting (\( p = 0.99 \)). Thus, only MAOA appears to be directly associated with turnout.

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8We classified genotypes based on transcriptional efficiency. The long allele of the 5HTT gene and the high allele of the MAOA gene has been associated with higher transcriptional efficiency (Denney, Koch, and Craig 1999, Denney et al. 1994, Lesch et al. 1996, Little et al. 1998, Sabol, Hu, and Hamer 1998). Males are homozygous for the MAOA gene (HH or II), however females may be heterozygous (HI). Following previous studies, we classify homozygous females as the Low genotype and all homozygous males as the High genotype (Fan et al. 2003, Frazzetto et al. 2007). Previous research has shown that being homozygous for the short allele (ss) makes one more vulnerable to negative environmental stimuli compared to being heterozygous for the short allele or homozygous for the long allele (Ls or LL; Caspi et al. 2003). Therefore, we combined Ls and LL into the Long genotype and ss into the Short genotype.

9Our sample contains 451 single-person families, 884 families with two siblings, 34 families with three siblings, and two families with four siblings.

10We also checked whether the odds ratio was significantly influenced by race or gender and it was not. We added an interaction with High MAOA and gender (male=1) which was not significant (\( p = 0.98 \)) and also an interaction with High MAOA and race (white=1) which was not significant (\( p = 0.79 \)).
The next two models test the possibility that attendance of religious services moderates an association between each of the genes and turnout. Model 3 suggests that no moderation relationship exists for MAOA (p = 0.93), but it also shows the robustness of the direct association with turnout since including attendance and an interaction in the model does not alter the significance of the main effect (p = 0.02). In contrast, Model 4 indicates that an association between 5HTT and voting is in fact moderated by attendance (p = 0.01). The odds of voting for those with the “long” version of the 5HTT gene and who frequently attend religious services are 1.58 greater than people with the “short” version.

To test the robustness of the direct and moderated associations, we model both of them simultaneously in Model 5. The results show that both odds ratios remain significant at p < 0.04.12

In Figure 2 we summarize our results for MAOA and the interaction between 5HTT and attendance by simulating first differences from the coefficient covariance matrix of Model 1 and Model 4. Holding all else constant and changing the MAOA gene of all subjects from “low” to “high” would increase average turnout in this hypothetical population by about 5 percentage points. Changing the 5HTT gene of all religious attendees from “short” to “long” would increase average turnout in that group by about 10 percentage points.

Model 6 includes a number of factors previous studies have found to influence turnout. These variables may in fact mediate the relationship between the genes we have identified and turnout.13 For example, MAOA and 5HTT may be associated with a disposition towards partisanship, which is known to significantly influence political participation (Bartels 2000). We might also expect genes to contribute to variation in socioeconomic factors like income (Bowles and Gintis 2002), which in turn would yield greater participation. Also, several twin studies have suggested that variation in cognitive ability can be attributed to

### Table 1 Models of Association Between MAOA, 5HTT, and Voter Turnout.

<table>
<thead>
<tr>
<th></th>
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<th>Model 2</th>
<th></th>
<th>Model 3</th>
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Note: Variable definitions are in the appendix. All results are expressed in odds ratios (OR). Standard errors (SE) are also presented.

11 We also checked whether the odds ratio was significantly influenced by race or gender and it was not. We added an interaction with Long 5HTT*Attend and gender (male = 1) which was not significant (p = 0.25) and also an interaction with Long 5HTT*Attend and race (white = 1) which was not significant (p = 0.32).

12 We also tested whether there was a significant gene-gene (GxG) interaction between 5HTT and MAOA by regressing Long 5HTT, High MAOA, Long*High, race controls, gender, and age on turnout. The estimated parameter on the interaction was insignificant (p = 0.89).

13 A variable M mediates the relationship between an independent variable X, in our case a genotype, and a dependent variable Y, in our case voting, if (1) X significantly predicts Y, (2) X significantly predicts M, and (3) M significantly predicts Y controlling for X (Baron and Kenny 1986).
genetic factors (McGue and Bouchard 1998). If so, then variation in the ability to process political information, which has an impact on turnout (Verba, Schlozman, and Brady 1995), may also be linked to genes. We can measure at least part of this ability using the Picture Vocabulary Test (PVT) administered by Add Health, which is thought to be a good measure of verbal IQ (Rowe, Jacobson, and Van den Oord 1999). Variation in educational attainment is another factor that has been found to be heritable (Baker et al. 1996; Heath et al. 1985) and is frequently shown to influence turnout (Leighley and Nagler 1992). In order to test whether these variables are potentially mediators, we regress each of them separately on High MAOA and Long 5HTT along with race, age, and gender controls. Since High MAOA and Long 5HTT are not significantly associated with any of these variables, we can rule them out as mediators.

Even after including all of these variables in the model, both High MAOA and the interaction between Long 5HTT and attendance remain significant. We also observe something rather unexpected in Model 6—the main effect of religious service attendance ceases to be significant ($p = 0.17$). In other words, it appears that the direct effect of church attendance on voter turnout that has been reported in so many other studies (see Cassel 1999 for a review) may be driven by two factors: (1) a spurious association caused by the relationship between other correlates of turnout and religious service attendance, and (2) the previously unmodeled interaction between religious service attendance and the functioning of the serotonin transporter, 5HTT. To test this assertion we simply remove 5HTT from the Model 6 specification (not shown). Religious attendance returns to exerting a significant direct effect on turnout ($p = 0.00001$). Our results show that individuals with the “short” 5HTT allele (the base category of the 5HTT variable) who are active in religious organizations are not more likely to vote. Similarly, individuals with the “long” allele who are not as active in religious organizations are not more likely to vote. In fact, voting is only higher among those who are both strongly exposed to the sense of community offered by religious groups (Cassel 1999) and potentially better equipped to handle the potential pain associated with social risks due to a fully-functioning serotonin metabolism conferred by a “long” 5HTT allele.

It is worthwhile pointing out how these results are reminiscent of the Caspi et al. (2002) findings on child abuse. Prior to that publication, scholars had reported a weak but significant relationship between receiving abuse as a child and abusing one’s own children as an adult. What Caspi and his colleagues showed was that this weak effect was moderated by the MAOA gene. People with the “high” MAOA allele who suffered abuse as a child were not more likely to abuse their own children. Similarly, people with the “low” allele who had not been abused were not more likely to abuse their own children. It was only those who experienced the trauma of child abuse and who lacked the protective effect of a fully-functioning serotonin metabolism conferred by a “high” MAOA allele that continued the cycle of violence. In the case of both voting and child abuse, bringing genes into the study of social behavior not only highlights the role of biology—it clarifies and sharpens the effect of the environment.

**Discussion**

The results of this analysis are clear: we have found that two extensively studied genes are significantly

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**Figure 2** Changing MAOA and 5HTT to High Activity Allele Yields Significantly Higher Turnout.

Note: First differences in turnout simulated from the coefficient covariance matrix of Model 1 and Model 4, assuming all other values are held at their means. Horizontal bar indicates 95% confidence interval.

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14 The $p$ values for High MAOA and Long 5HTT in each regression are presented in the appendix.
associated with voter turnout. Further, these are the first two genes ever directly associated with political behavior. The empirical approach we employ in this paper improves on the twin study designs of Fowler, Baker, and Dawes (2008) in a profoundly important way. Twin studies are valuable for determining the influence of genes over observed behavior, but they are agnostic about causality. By focusing on specific genes, our analysis is able to suggest potential causal pathways through which genes influence turnout. A significant body of research has found that the two genes we study, 5HTT and MAOA, influence social behavior via their impact on the serotonin metabolism and research within political science has identified prosociality as a significant determinant of turnout, thereby establishing a potential causal chain leading from these genes to observed political behavior. Again, we cannot test any causal pathway given our data so we are merely speculating based on previous work done in behavior genetics and political science.

More broadly, these results represent an important step for political science as a discipline. Specifically, they show that incorporating genetic information into our theories and analysis may contribute to a greater understanding of political behavior. The environment-only approach used for so long in political science has frequently conceptualized human behavior as a “blank slate” on which any tendencies could be drawn, regardless of the unique biology of each individual (Pinker 2003). However, the results presented here refute the blank slate theory of political behavior. Although the environment is extremely important for turnout and other political acts, perhaps even more so than genes, we can no longer act as if genes do not matter at all. Genetic differences are likely to have important consequences for a whole range of political behaviors.

We believe the significant interaction we find between 5HTT and church attendance adds an important element missing from existing theories explaining the relationship between religious activity and turnout. Cassel (1999) suggests religious groups build a sense of belonging to a larger community, but any sense of belonging is likely mitigated by social anxiety or an aversion to potential social conflict. Brady, Verba, and Schlozman (1995) argue that civic skills, acquired through church activities, increase the likelihood of voting. Specifically, they find that regular attendance prompts individuals to perform acts on behalf of their religious organization that enhance their civic skills, in turn better equipping them for involvement in the political process. These acts include writing a letter, taking part in decision making, giving presentations or speeches, organizing or chairing a meeting, and/or contacting government officials on behalf of the church. However, the leap from church attendance to completing these acts is not a surety for every individual. These acts often require extensive interaction with others in a social setting, something that is likely to be uncomfortable for those with social anxiety and fear of social rejection. Even the simple act of writing a letter may be difficult when the person writing it knows others will evaluate it. Rosenstone and Hansen (1993) suggest that religious organizations provide a venue amenable to the discussion of politics, thus lowering the cost of gathering information. Since voting is less costly for regular attendees compared to those who do not attend, we should observe a higher turnout rate among this group. As in the case with the development of civic skills, engaging in political discussions with fellow members of one’s church is likely difficult for those with antisocial tendencies. Therefore, if our hypothesis is correct, we would expect individuals with the “long” 5HTT allele to be most likely to benefit from political discussions in a church setting.

Even if one concedes genes do influence political behavior, it is tempting to assume that since they are not causally proximate to observed behaviors they can be safely ignored for practical purposes. However, this thinking is mistaken. Genes are the institutions of the human body—they constrain individual behavior just as political institutions constrain the behavior of groups of people. In this article we demonstrate that possessing a particular gene is associated with voting activity. Even after controlling for factors known to influence turnout, having a high MAOA allele raises the likelihood of voting by about 5%. Among people active in their religious organizations, having a long 5HTT allele raises the likelihood of voting by about 10%. We theorize that since low efficiency MAOA and 5HTT alleles limit the degree to which individuals are socially oriented, these alleles inhibit their desire or ability to participate in the political process.

Our theory that genetic differences within a population, in part, explain variation in political behavior is in stark contrast to game-theoretic models of voter turnout that typically predict very little variation in participatory behavior (Aldrich 1993). In these models, 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
The empirical literature has embraced variation in turnout behavior with models that test dozens of explanatory variables (Plutzer 2002; Timpone 1998; Verba, Schlozman, and Brady 1995). These models include: 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 1992b), income (Leighley and Nagler 1992a), occupational prestige (Nie, Powell, and Prewitt 1969a, 1969b), and home ownership (Highton and Wolfinger 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 (Wielhouwer and Lockerbie 1994), campaigns (Ansolabehere and Gerber 1994), civic education (Somit et al. 1958), polling locations (Gimpel and Schuknecht 2003), and barriers to registration (Rosenstone and Wolfinger 1978). However, not one of these articles has considered the possibility that genes may account for this variation.

Genes may also help us to explain 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 also be due to the inheritance of particular alleles of genes like MAOA and 5HTT. 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 that...
genes like MAOA and 5HTT are fixed, it might also be largely due to inherent genetic variability within the population. Future longitudinal and family studies of voter turnout should investigate what role MAOA and 5HTT play in the transmission of political behavior over time within individuals and between parents and children. In particular, it will be interesting to understand better why these two genes that affect the serotonin system behave differently—why MAOA is associated with behavior directly, while 5HTT interacts with exposure to social activity.

Future work should use genetic association studies to identify specific genes that are implicated in political behaviors and attitudes. 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). It is important to emphasize that there is likely no single “voting gene”—the results presented here suggest that at least two genes do matter and there is some (likely large) set of genes whose expression, in combination with environmental factors, influences political participation. Finally, we offer a word of caution. Association studies like ours require further replication before their findings can be truly considered anything more than suggestive, therefore more work needs to be done in order to verify and better understand the specific associations we have identified.

Acknowledgments

This research was funded by National Science Foundation grant number SES-0719404. The most recent version of this paper can be found at http://jhfowler.ucsd.edu. The contact author can be reached at jhfowler@ucsd.edu.

Appendix

Variable Definitions

*High* is an indicator variable for having two of the 336, 351, or 381bp alleles of the MAOA gene. *Long* refers to having at least one 528bp allele of the 5HTT gene. *Partisan* is the answer to the question “Do you identify with a specific political party?” *Attendance* is constructed from the response to the question, “How often have you attended [church/synagogue/temple/mosque/religious] services in the past 12 months?” The categories of attendance are never, at least a few times but no more than once a month (baseline), and more than once a month. We center this variable on the category at least a few times a month (never = -1, at least once a month = 0, more than once a month = 1). Other race/ethnicity indicator variables based on the questions “Are you of Hispanic or Latino origin?” and “What is your race? [white/black or African American/American Indian or Native American/Asian or Pacific Islander].” *Age* is self-reported age, and *Male* is an indicator taking the value of 1 if the respondent is a male and 0 for a female. *Income* is the log of the response to the question, “Including all the income sources you reported above, what was your total personal income before taxes in [2000/2001]?” *Cognitive Ability* is the score on the Picture Vocabulary Test, which measures verbal intelligence. *College* is an indicator variable taking the value 1 if the respondent completed at least one year of college and 0 for no college. It is based on the question, “What is the highest grade or year of regular school you completed?”

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<td>High MAOA</td>
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</tr>
<tr>
<td>Long 5HTT</td>
<td>81.4</td>
</tr>
<tr>
<td>Partisan</td>
<td>37.3</td>
</tr>
<tr>
<td>College</td>
<td>55.5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Sample means.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
</tr>
<tr>
<td>Age</td>
<td>21.9</td>
</tr>
<tr>
<td>Income</td>
<td>12912</td>
</tr>
<tr>
<td>Cognitive Ability</td>
<td>99.12</td>
</tr>
<tr>
<td>Attend</td>
<td>2.1</td>
</tr>
</tbody>
</table>
Table 4  Percentage of subjects exhibiting these characteristics by race.

<table>
<thead>
<tr>
<th>Race / Ethnicity</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>White</td>
<td></td>
</tr>
<tr>
<td>Vote</td>
<td>44.6</td>
</tr>
<tr>
<td>Long 5HTT</td>
<td>81.6</td>
</tr>
<tr>
<td>High MAOA</td>
<td>53.4</td>
</tr>
<tr>
<td>Black</td>
<td></td>
</tr>
<tr>
<td>Vote</td>
<td>52.5</td>
</tr>
<tr>
<td>Long 5HTT</td>
<td>91.6</td>
</tr>
<tr>
<td>High MAOA</td>
<td>33.2</td>
</tr>
<tr>
<td>Hispanic</td>
<td></td>
</tr>
<tr>
<td>Vote</td>
<td>39.6</td>
</tr>
<tr>
<td>Long 5HTT</td>
<td>76.0</td>
</tr>
<tr>
<td>High MAOA</td>
<td>46.1</td>
</tr>
<tr>
<td>Asian</td>
<td></td>
</tr>
<tr>
<td>Vote</td>
<td>40.9</td>
</tr>
<tr>
<td>Long 5HTT</td>
<td>55.2</td>
</tr>
<tr>
<td>High MAOA</td>
<td>31.9</td>
</tr>
<tr>
<td>Nat Am</td>
<td></td>
</tr>
<tr>
<td>Vote</td>
<td>63.5</td>
</tr>
<tr>
<td>Long 5HTT</td>
<td>73.4</td>
</tr>
<tr>
<td>High MAOA</td>
<td>55.8</td>
</tr>
</tbody>
</table>

Table 5  Test of High MAOA and Long 5HTT as potential mediators.

<table>
<thead>
<tr>
<th></th>
<th>High MAOA</th>
<th>Long 5HTT</th>
</tr>
</thead>
<tbody>
<tr>
<td>DV</td>
<td>p</td>
<td>p</td>
</tr>
<tr>
<td>Partisan</td>
<td>0.41</td>
<td>0.36</td>
</tr>
<tr>
<td>Income</td>
<td>0.84</td>
<td>0.77</td>
</tr>
<tr>
<td>Cognitive Ability</td>
<td>0.09</td>
<td>0.35</td>
</tr>
<tr>
<td>College</td>
<td>0.91</td>
<td>0.95</td>
</tr>
</tbody>
</table>

Note: Table presents p values for High MAOA and Long 5HTT in models with partisanship, income, cognitive ability, and college attendance as dependent variables. Regressions also include race, age, and gender controls.

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Friendships Moderate an Association between a Dopamine Gene Variant and Political Ideology

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Scholars in many fields have long noted the importance of social context in the development of political ideology. Recent work suggests that political ideology also has a heritable component, but no specific gene variant or combination of variants associated with political ideology have so far been identified. Here, we hypothesize that individuals with a genetic predisposition toward seeking out new experiences will tend to be more liberal, but only if they are embedded in a social context that provides them with multiple points of view. Using data from the National Longitudinal Study of Adolescent Health, we test this hypothesis by investigating 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. Among those with DRD4-7R, we find that the number of friendships a person has in adolescence is significantly associated with liberal political ideology. Among those without the gene variant, there is no association. This is the first study to elaborate a specific gene-environment interaction that contributes to ideological self-identification, and it highlights the importance of incorporating both nature and nurture into the study of political preferences.

In his influential collection of essays, Ideology and Utopia, Karl Mannheim (1936) sought to explain the meaning and origin of political ideology. As a founder of the “sociology of knowledge” school of thought, he broadened a view, first championed by Marx, that individual political attitudes are derived from groups and their relationships to the whole of society. While Marx focused particularly on class relations, Mannheim observed that political ideology is the product of the total social context of each individual. To understand a person’s political ideology, we need only examine his or her political environment.

Mannheim’s work would influence several generations of scholars (e.g., Bell 1959; Haas 1992; Huntington 1957; Lipset 1983; North 1978; Rapoport 1974). While many of these scholars disagreed with parts of Mannheim’s argument, they all agreed with the premise: that the social and institutional environment is paramount for explaining a person’s political attitudes and beliefs. When individuals say they are “liberal” or “conservative,” they are referring to their ideas about the issues of the day that are specific to a place and a moment in history. Remove them to another context and their ideology will change.

This literature contrasts with a growing body of work that suggests ideology is not purely a product of the social environment or historical moment. Increasingly, it is becoming apparent that political ideology also has a “core” element that is rooted in innate predispositions, personality, and “motivated social cognition” (Jost et al. 2003). In fact, psychologists have asserted for many years that social conservatism is heritable (Bouchard and McGue 2003; Cloninger, Svrakic, and Przybeck 1993; Eaves and Eysenck 1974) and that genetic factors account for a significant proportion of variation in social attitudes (Martin et al. 1986; Tesser 1993). Alford, Funk, and Hibbing (2005) were among the first to present these findings to the political science discipline, showing that genetic variation helps to explain both the direction (liberal vs. conservative) and strength of ideological opinions in a very large sample of twins. Since then, scholars have found that political attitudes related to vote choice are also heritable (Hatemi et al. 2007) as is the strength of
partisan attachment (Hatemi et al. 2009a; Settle, Dawes, and Fowler 2009). Likewise, genetic variation is important for explaining variation in political behaviors that are known to be influenced by ideology, like voting and other forms of political participation (Fowler, Baker, and Dawes 2008; Fowler and Dawes 2008; Dawes and Fowler 2009). These findings suggest that we should revise our environment-only understanding of political attitudes and ideology. Political ideology is rooted in general social psychological tendencies and has inheritable and durable components that may be constrained or exacerbated by the influence of the social context.

Social and genetic theories about the nature and origin of political ideology need not be at odds with one another. In fact, it is likely that genes influence political ideology by partially regulating the way we react to the total social context. If so, then one way forward in our understanding of the biological and social bases of political attitudes and beliefs is to search for specific gene-environment interactions that may play a role in the development and maintenance of political ideology. Behavior geneticists note that complex social behaviors are polygenic (Mackay 2001; Plomin 2008); likewise there are probably many gene-environment interactions that play a role in the acquisition of political ideology. A logical way to start our search is to examine gene variants that are already known to contribute to variation in social behaviors that are related to political ideology. One such gene is the D4 dopamine receptor gene (DRD4), which regulates dopamine activity in the brain (Cloninger, Svrakic, and Przybeck 1993; Wiesbeck et al. 1995). The 7R allele of this gene has been associated with novelty-seeking behavior (Auerbach et al. 2001; Benjamin et al. 1996; Benjamin et al. 2000; De Luca et al. 2001; Ebstein et al. 1996; Noble et al. 1998; Schmidt et al. 2002; Strobel et al. 1999; Tomitaka et al. 1999), which is a tendency that is related to openness (De Fruyt, Van De Wieleb, and Van Heeringen 2000), a psychological trait that has been associated with political liberalism (Jost et al. 2003; Pratto et al. 1994; Peterson and Lane 2001; Peterson, Smirles, and Wentworth 1997). This prior research suggests one possible pathway from genes to personality to ideology, but it does not consider the important role of social context. We therefore investigated how the 7R allele might interact with an important social variable: the number of friendships a person forms.

This article is the first to identify a specific gene-environment interaction that is associated with the direction of a person’s ideological self-identification. We show that among those who carry the 7R allele of the DRD4 gene, the number of friends a person has in adolescence is positively associated with liberal self-identification in early adulthood. Among those who do not carry the 7R allele, there is no relationship between number of friends and ideology. Moreover, we show that the 7R allele is not directly associated with the reported number of friends, nor is it directly associated with ideology. Instead, it is the combination of this specific gene variant with a specific social environment that may contribute to the development of a liberal political ideology.

**Ideology: Past Research and Support for a Genetic Basis**

We define ideology as a general belief system that encompasses a wide set of idea-elements that come together in a nonrandom fashion (Converse 1964; Gerring 1997). Here, we refer more specifically to the liberal-conservative continuum commonly understood as organizing American politics (Treier and Hillygus 2005). It has been long debated whether ideology is rooted in issue preferences (Converse 1964) or whether ideological labels are symbolic and affect-oriented (Conover and Feldman 1981). Early studies suggest that Americans have little constraint in their ideology, that they have few opinions about public policy or political parties, that their opinions on policy change frequently, dramatically, even randomly, and that they have limited capacity to process political information (Campbell et al. 1960; Converse 1964). These early findings have been critiqued from both a methodological and conceptual perspective, and the general consensus has been that the public is not particularly ideological or sophisticated, but neither is it totally unreasoning nor unopinionated (Sniderman, Brody, and Tetlock 1993). Ideological labels are more salient (Hinich and Munger 1997; Holbrook 1996; Treier and Hillygus 2005) and more meaningful as party leaders and elites polarize (Abramowitz and Saunders 1998; Hetherington 2001; Hinich and Munger 1997; Jacobson 2003; McCarty, Poole, and Rosenthal 2006; Schreckhise and Shields 2003). They are also useful (Jacoby 2004; Lau and Redlawsk 1997), even if not everyone precisely agrees on what the terms “liberal” and “conservative” mean.

Furthermore, there is a strong association between parental and offspring political attitudes. With few exceptions past scholars have attributed this to
the environmental influences and political socialization to which parents expose their children (Jennings and Niemi 1968). However, recent evidence from a series of twin and extended kinship studies suggests that the mechanism by which parents pass their ideology to their children may in part be due to heredity, and that political attitudes themselves are genetically influenced (Alford, Funk, and Hibbing 2005; Eaves and Hatemi 2008; Hatemi, Medland, and Eaves 2009b; Tesser 1993). Studies based on large samples of twins from the United States, the United Kingdom, and Australia find that at least a third of the variation in political attitudes can be accounted for by genes and approximately half of the variation can be explained by unshared environment. Hatemi et al. (2007) replicated these results using an extended family design that includes parents and nontwin siblings. But no study has yet identified specific genes that are associated with ideology.

**Dopamine and DRD4-7R**

Dopamine, a member of the catecholamine family, is one of many different types of neurotransmitters which can be found in the brain, each with different functions. The D4 dopamine receptor DRD4 (one of five subtypes of dopamine receptors) is a protein transcribed by a gene with the same name (DRD4). This gene is commonly described by at least three polymorphic variations in its coding sequence (Van Tol et al. 1992), including the allele of interest in this study, the long form allele (7R). Among other behaviors, novelty seeking is thought to be mediated by genetic variability in dopamine transmission (Cloninger, Svrakic, and Przybeck 1993). A wide variety of genetic association studies have tested the link between polymorphisms of DRD4 and novelty-seeking behavior with generally positive results (Kluger, Siegfried, and Ebstein 2002; Savitz and Ramesar 2004; Schinka, Letsch, and Crawford 2002). There are several proposed mechanisms, most of which are related to the manner and frequency with which dopamine binds to its receptors.\(^1\)

Studies of animals indicate that DRD4 is involved in cortical excitability and behavioral sensitization. These alterations in cortical arousal affect “approach traits” such as novelty seeking and sensation seeking, which in turn affect personality and behavior (Eichhammer et al. 2005). People who score high on measures of novelty seeking have less tolerance for monotony and constantly seek the new and unusual (to them) in order to alter dopamine levels to affect mood; at the extremes, they are characterized as impulsive, exploratory, fickle, excitable, quick-tempered, and extravagant (Puttonen, Ravaja, and Keltikangas-Jarvinen 2005). People who score low on this measure tend to be more inclined to follow the rules (Golimbet et al. 2007). Those who score lower also tend to be more reflective, rigid, loyal, stoic, slow-tempered, and frugal.

**Novelty Seeking, Friends, and Liberalism**

Certain situational and dispositional factors may contribute to a cognitive-motivational orientation toward the social world that is either closed and invariant or open and exploratory (Kruglanski and Webster 1996). In fact, “openness to experience,” a construct conceptually related to novelty seeking, is the personality trait most commonly linked to political orientations (Cornelis et al. 2009; Jost et al. 2003; McCrae 1996; Mondak and Halperin 2008) and has been found to be negatively related to political conservatism generally (Van Hiel, Kossowska, and Mervielde 2000) and sociocultural conservatism specifically (McCrae 1996; Peterson, Smirles, and Wentworth 1997; Trapnell 1994; Van Hiel, Kossowska, and Mervielde 2000). The relationship between openness to experience and ideology holds when ideology is measured either as support for ideological political parties (Caprara, Barbaranelli, and Zimbardo, 1999; Van Hiel, Kossowska, and Mervielde 2000) or as ideological self-placement (Carney, Jost, and Gosling 2008; Van Hiel, Kossowska, and Mervielde 2000). The connection between openness to experience and ideology may even stem from the same genetic constructs: Verhulst, Eaves, and Hatemi (2009) suggest that the relationship between personality and political preferences is the result of shared genetic influence.

However, we argue that the DRD4-7R allele cannot by itself predispose someone to a liberal ideology. It requires a context in which people are exposed to certain social environments. Here, we focus on the number of friendships a person has because this is an essential measure of a person’s
social context. Psychologists have found that friendships promote growth in social cognition and self-concept (Staub 1995), increase feelings of social belonging (Bishop and Inderbitzen 1995), increase self-esteem (Bishop and Inderbitzen 1995), promote a better understanding of others' needs, foster mutual trust (Neibrzydowski 1995), encourage greater consideration in regard to society (Selman 1990, White et al. 1987), and promote prosocial behavior (Hartup 1983). For people who like new experiences, friendships thus serve to expose a person to the socio-political world, perhaps activating a political ideology that psychologically satisfies an openness to change and new experience.

Additionally, an increased number of friends may expose a respondent to a wider diversity of viewpoints. Although social networks are known to be homophilous (McPherson, Smith-Lovin, and Cook 2001), Huckfeldt, Johnson, and Sprague (2004) demonstrate that significant political disagreement persists between friends, suggesting that the more friends people have, the more likely they are to regularly engage with at least one person with a different point of view. And although disagreement might result in more political ambivalence and less political engagement (Mutz 2002), it does not reduce the intensity of a person’s political opinions (Huckfeldt, Johnson, and Sprague 2004, 203). Adolescents are in the process of learning about the social world; therefore they are being shaped by a wide variety of influences. Those who have more friends are exposed to more new experiences in childhood (Heiman 2000). For adolescents who are innately novelty seeking, a natural tendency toward openness to experience may create psychological satisfaction derived from novel experiences, including the desire to learn about and understand multiple points of view from one’s friends.

Finally, it is well known that peers and friends exert an influence on political preferences (Berelson, Lazarsfeld, and McPhee 1954), and recent work suggests that informal components in the school environment, such as the influence of peer attitudes, contribute to political socialization (Settle, Bond, and Levitt forthcoming).

For these reasons, we hypothesize that the combination of an innate desire for novel experience and many friends may contribute to the activation of a liberal ideology. People who have many friends may nonetheless remain uninterested in their friends’ point of view. Alternatively, people who crave new experiences may not get them from their social context if they have only a few friends. It is the interaction of the desire for new experience and many different pathways to these experiences that we hypothesize has an impact on political ideology.

## Add Health and Network Properties

We use data from the National Longitudinal Study of Adolescent Health (Add Health) to examine the moderating influence of friendships on the effect of DRD4 on political ideology.

Genetic markers are available for a sample of 2,574 individuals, including markers that identify alleles of DRD4. In our sample, 62% have no 7R alleles, 33% have one copy of the allele and 5% have two copies of the allele. The study has been described elsewhere (Fowler, Baker, and Dawes 2008); more detailed description of the study and genetic data can be found in the online appendix at http://journals.cambridge.org/jop and on the study web page (http://www.cpc.unc.edu/projects/addhealth). Information was gathered in the first wave about a subject’s social network. Students were allowed to nominate up to five female and five male friends. We will focus on a simple measure, the number of nonfamilial friends named by the respondent in the first wave.

Nearly 80% of the sibling-pairs sample participants in Wave I also participated in Wave III. Subjects were young adults (age 18–26) by the time of the third wave and were asked several questions about their political behavior and civic activity. Our dependent variable, self-identified ideology, is ascertained from responses to the question, “In terms of politics, do you consider yourself conservative, liberal, or middle-of-the-road?” Five responses were permitted, “very conservative,” “conservative,” “middle-of-the-road,” “liberal,” or “very liberal.” This ideology question is well-suited for our purpose of exploring the genetic basis of ideology because of its similarity to standard questions used on the National Election Study survey and other frequently cited surveys (Jost 2006).

## Genetic Association

Genetic association studies test whether an allele or genotype occurs more frequently within a group exhibiting a particular trait than those without the trait (e.g., is the frequency of a particular allele or genotype higher among liberals than conservatives?). Because a significant association has several possible explanations, there are two main research designs
employed in association studies to isolate the effect of an allele on a trait, case-control designs and family-based designs (Carey 2002). Due to potential population stratification in our sample, we chose to employ a family-based design, which eliminates the problem of population stratification by using family members, such as parents or siblings, as controls. Tests using family data compare whether offspring exhibiting the trait receive a particular allele from their parents more often than would be expected by chance. They do not rely on twins to study genetic variation; any kind of close family relation can be used (siblings, parents, etc.). (See the online appendix for a more detailed explanation of the research design.)

**Family-Based Design Methods and Results**

Gauderman (2003) showed that the family-based quantitative disequilibrium transmission test (QTDT) of association (Abecasis, Cardon, and Cookson 2000; Fulker et al. 1999) could be extended to accommodate gene-environment interactions. The model of allelic transmission we employ is:

\[
y_{ij} = \beta_0 + \beta_b b_i + \beta_w w_{ij} + \beta_E E_{ij} + \beta_{wE} E_{ij} w_{ij}
\]

\[
w_{ij} = g_{ij} - \bar{b}_i
\]

\[
\bar{b}_i = \frac{\sum g_{ij}}{n_i}
\]

where \(y_{ij}\) is the ideology of individual \(j\) in family \(i\), \(n_i\) is the number of family members, \(g_{ij}\) is the genotypic score which equals the number of 7R alleles (0, 1, or 2), \(b_i\) is the expected genotypic score, \(w_{ij}\) is an individual’s deviation from the expected genotypic score, and \(E_{ij}\) is the number of friendships an individual reports. A positive value for \(w_{ij}\) means that a child inherited an excess number of copies of the 7R allele from his or her parents than expected and thus a significant positive value for \(\beta_w\) indicates that an excess transmission of 7R alleles is associated with holding a more liberal ideology. A significant positive value for \(\beta_{wE}\) suggests that this association is moderated by the number of friendships one has.

The QTDT decomposes the genotypic score into two orthogonal components, the between-family component (the expected genotypic score) and the within-family component (the deviation from the genotypic score). The virtue of this design is that while the total association and the between-family component are sensitive to population stratification, which could result in a false signal of association, the within-family component is not (since family members share the same ancestry). Therefore, the QTDT effectively guards against population stratification.

We include individuals from the same family in the analysis, and thus the observations are not independent. Therefore, we use a generalized estimating equations approach with an independent working correlation structure for the clustered errors, to estimate the model. Only siblings that have different genotypes, in this case a different number of 7R alleles, are informative for the within-family component of variance since \(w_{ij}\) equals zero otherwise. However, families that share the same genotype are also included in our analysis for improved estimation of the between-family component. We have also included controls in the model for both age and gender, as there are numerous instances of age effects in gene-environment interactions and there are sex-specific genetic influences on political preferences (Hatemi, Medland, and Eaves 2009c). As is described in the behavior genetics literature (Eaves 1984), a statistical interaction between a gene and the environment will only be present when there is variation in both the gene and the environment. From a statistical perspective, the magnitude of the gene-by-environment interaction can be considered the extent to which the average performance of the gene and the environment fail to predict the response of individual combinations of genotypes and environments. When using genetic analysis, however, it may be best to view the interaction as the genetic control of sensitivity to the environment (Eaves 1984).

The results of the model are presented in Table 1.

The parameters of primary interest are \(\beta_w\) and \(\beta_{wE}\) because they represent formal tests of association. The estimate of \(\beta_w\) is not significant \((p=0.35)\) suggesting that there is not a direct association between the 7R allele and ideology. However, the estimate of the interaction \(\beta_{wE}\) is significant \((p=0.02)\), meaning that the number of friendships moderates an association between 7R and ideology. The interpretation of this result is that having more 7R alleles and more friends is associated with being more liberal. As a further test, we randomly resample our data set 5,000 times with replacement and calculate an empirical \(p\)-value based on these estimates of \(\beta_{wE}\). The empirical \(p\)-value is also significant \((p=0.01)\). To be sure that the interaction is not the result of a direct association between 7R alleles and ideology or friendships, we conduct additional
The results of these tests indicate that 7R alleles have no significant effect on the number of friends, and no direct impact on ideology. Only the interaction is significant.

Figure 1 is a graphical representation of the interaction between the presence of the alleles and the number of friendships. For those without any 7R alleles, the number of friends is not related to liberal ideology. Holding all else constant, for people who have two copies of the allele, an increase in number of friendships from zero to ten friends is associated with increasing ideology in the liberal direction by about 40% of a category on our five-category scale. In other words, 10 friends can move a person with two copies of the 7R allele almost halfway from being conservative to moderate or from being moderate to liberal.

To address further the nature of the friendship interaction, we report additional analyses in the appendix that demonstrate our results are not being driven by gendered patterns or interactions of friendship and that the results hold when using a dichotomous measure of friendship. We also report the results of the interaction model, but without the interaction term. The residual deviance for this model is higher than the model with the interaction, indicating that the interaction model fits better.

Discussion

For most traits, the effects of individual genes are too small to stand out against the combined influence of all other genes and environmental factors. Thus, our p-value of 0.02 on a sample of 2,000 individuals should be treated cautiously. The expectation in genetics is that only repeated efforts to replicate associations on independent samples by several research teams will verify initial findings like these. Thus, perhaps the most valuable contribution of this study is not to declare that “a gene was found” for anything, but rather, to provide the first evidence for a possible gene-environment interaction for political ideology.

Many large-scale analyses of political behaviors ignore the potential for genetic effects. Of those that do not, few offer a model which builds a hypothesis based on social and cultural influences that interact with a specific neurotransmitter that is regulated by a specific genetic marker. It is our hope that more
scholars will begin to explore the potential interaction of biology and environment, thus leading to the development of consortiums for social and political traits that will allow for the replication or combination of findings across samples.

Given these cautions, we reiterate the main results of our investigation. Using a family-based genetic association study, we find that political ideology in early adulthood is significantly associated with an interaction between a gene previously linked to novelty-seeking behavior (\textit{DRD4}-7R), and an environmental influence, the number of adolescent friendships. We do not claim that this evidence proves a causal relationship between \textit{DRD4} and political ideology. However, the association is consistent with a causal theory that we develop about the way genes and environments combine to affect political ideology.

It is important to note that the 7R allele by itself does not make a person liberal and neither does simply having a greater number of friends as a teenager. Additionally, the 7R allele does not cause an individual to have more friends (see the appendix), and twin studies have shown that the number of friends one names is not significantly heritable (Fowler, Dawes, Christakis 2009). Rather, it is the crucial interaction of two factors—the genetic predisposition of having a greater number of 7R alleles and the environmental condition of having many friends in adolescence—that is associated with being more liberal.

Geneticists are sometimes skeptical of associations where an interaction effect exists and a main effect does not. The reason for this skepticism is a concern with the potential to produce false positive results. If we were testing 100 genes and 100 environmental factors, there would be 10,000 possible interactions, and many of these would yield significant results. However, in our case, the Add Health data contains only eight genes, greatly reducing the number of possible interactions one could test. This does not necessarily reduce the possibility of false positive for a given test, but does offer protection from unreported multiple testing. Political scientists also typically guard against false positives by requiring \textit{ex ante} theorizing. In our case, we were only interested in \textit{DRD4} for political ideology because of its association with novelty-seeking behavior, and we developed an explicit theory that the interaction between \textit{DRD4} and friendships—and not the main effects—would be significant. Since our test was not able to contradict the theory, the way forward is to seek replication in different populations and age groups.

While our finding is statistically significant, the strength of the association is quite small. However, even in a biometric trait such as height, less than 15% of the variation has been attributed to specific genes. Genetic effects take place in complex interaction with other genes and environments, and it is likely the combination of hundreds if not thousands of genes interacting with each other and with external stimuli that influence political attitudes and behavior.

There are several factors that would be instrumental for future replication studies. There is no measure in the Add Health data that has been validated against typical measures of novelty seeking; the questions that are conceptually related in the survey are inappropriate for comparison to other studies which include the behavioral trait in the analysis. If we did have such a measure, we could test the extent to which it accounts for the associations we observe here. Additionally, both number of friends nominated and ideology were single self-report measures. While we have good reason to believe that self-reported ideology is an accurate representation of a person’s true ideological beliefs (Lau and Redlawsk 1997), we are using a standard, but very simple, measure that may not fully capture the breadth and depth of a person’s ideological beliefs. Finally, past work suggests that political sophistication plays an important role in the manifestation of ideology (Converse 1964; Sniderman, Brody, and Tetlock 1993), but we cannot address the role that political sophistication might play in our results because there were no reliable measures in Add Health (2007). All of these limitations suggest that we should develop datasets that include genes, psychological questions to create valid constructs, and political data.

In light of these and other findings, political scientists can no longer afford to view ideology as a strictly social construct, perfectly malleable and completely subject to historically changing circumstances. As Jost et al. (2003) suggest, there appears to be both a stable definitional core and changing peripheral associations involved in a conservative political ideology. Our work builds upon this growing literature in psychology and genetics by offering a genetic basis for the link between motivated social cognition and ideology.

Finally, the results here suggest that, contrary to Mannheim’s assertion and the body of work that followed him, the social and institutional environment cannot entirely explain a person’s political attitudes and beliefs. We must take into account the role of genes and gene-environment interactions in
the formation and maintenance of political beliefs. Our findings do not undermine the rich body of literature that has developed regarding the environmental influences that shape political behavior. Rather, we hope to complement prior work and seek to show how incorporating a role for specific genes into our models of political behavior can enrich our understanding of the origin and nature of these behaviors. Political scientists have a wealth of material from which to form hypotheses about potential gene-environment interactions that influence deeply held political ideas and values.

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