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# The Heritability of Partisan Attachment

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

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

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

## What Is Partisanship?

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

Since the 1970s, a debate in the literature has contested whether partisanship is affective, nearly immutable, and emotionally based or whether it is

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

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

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

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

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

However, while heritability plays a role in many political behaviors, we do not expect that it will play a role in all political behaviors (Alford and Hibbing 2008), and we have reason to expect a difference in the heritability of strength versus direction of partisanship. As measured in both our sample and in Hatemi (2007) and Hatemi et al. (2007), identifying oneself as a "strong" partisan is a reflection of the propensity toward group attachment. Research on the heritability of religion has found that while religiosity is strongly heritable, denominational affiliation is not (Eaves 1977; Martin et al. 1986; Bouchard and McGue 2003; Koenig et al. 2005; Bouchard et al. 1999; Beer, Arnold, and Loehlin 1998; Bouchard et al. 2004). We conjecture that the relationship between strength of partisan attachment and party identification is analogous to the relationship between religiosity

and denominational affiliation (Hatemi et al. 2007; Jennings, Stoker, and Bower 2001). That is, the intensity of one's attachment to a group may be shaped by genetic predispositions, but the selection of the group to which one attaches is largely shaped by parental and environmental exposures. We expect a different pattern for ideology, which is best viewed as an orientation toward the social world and its organization (Jost 2006; Alford and Hibbing 2008). Ideological orientations may be heritable because of their social psychological role (Jost et al. 2003; Jost 2006; Alford and Hibbing 2008), but the intensity of these beliefs may not have a strong heritable component since the underlying psychological factors associated with liberalism (like openness) and conservatism (like conscientiousness) are directional.

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

## Twin Studies

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

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

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

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

Some scholars argue that the identical nature of MZ twins causes them to be more strongly affiliated

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

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

## Twins Days Festival Data Collection

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

Participants were asked a series of demographic questions and a question about their partisan affiliation, using the traditional 7-point partisanship scale. This scale was then "folded over" to produce a 4-point, directionless scale of partisan attachment. This is a variation of the standard National Election Studies (NES)/Michigan party identification scale,[1] the most frequently used scale of partisanship in the literature (Weisberg 1999). This scale has been found to tap into respondents' general partisan tendencies and may be less sensitive to transitive or election-specific partisan preferences than other measures (Whiteley 1988; Green and Palmquist 1990; W. Miller 1991; Abramson and Ostrom 1991, 1992, 1994a, 1994b; W. Miller and Shanks 1996). Moreover, this measure has been used frequently as an explanatory variable in models of political participation (Timpone 1998; Verba, Schlozman, and Brady 1995). Because we are most interested in the long-term aspects of partisan strength, the wording of this question makes it most appropriate for our purposes.

Table 1 shows summary statistics for study participants. One method of evaluating whether MZ and DZ twins are drawn from comparable environments is to examine the distributions of relevant covariates between the two groups. If there are any significant differences between the MZ and DZ twins, we can interact the covariate in question with A (the additive genetic component) in the ACE model to see if it impacts the heritability estimate. For example, there is a statistically significant difference in age between the MZ and DZ twins. However, when we add a variable for age to the ACE model, we do not find any evidence that the heritability of partisanship strength changes with age. Similarly, we find that MZ twins

**Table 1**
**Summary Statistics**

| | MZ Mean | MZ *SD* | DZ Mean | DZ *SD* | Difference (*p*-Value) |
|---|---|---|---|---|---|
| Male | 0.19 | 0.40 | 0.20 | 0.40 | .89 |
| Age | 36.96 | 16.69 | 32.43 | 14.84 | .00 |
| Turnout | 0.69 | 0.46 | 0.66 | 0.47 | .30 |
| Education | 0.64 | 0.48 | 0.57 | 0.50 | .15 |
| Income | 0.41 | 0.49 | 0.36 | 0.48 | .45 |
| Democrat | 0.30 | 0.46 | 0.35 | 0.48 | .34 |
| Republican | 0.34 | 0.47 | 0.19 | 0.40 | .00 |
| Liberal | 0.19 | 0.40 | 0.20 | 0.40 | .66 |
| Conservative | 0.71 | 0.46 | 0.69 | 0.46 | .47 |
| Partisan intensity | 1.63 | 0.89 | 1.51 | 0.90 | .12 |
| Ideological intensity | 1.26 | 1.03 | 1.07 | 1.03 | .02 |

Note: MZ = monozygotic; DZ = dizygotic.

are somewhat more likely to be Republicans and ideologically intense.[2] However, neither of these variables significantly moderates the heritability estimates. Thus, while this sample is small and not randomly selected, it does not appear to be systematically biased in a way that affects our ability to use the data to estimate the influence of genes on partisan attachment. Finally, because the sample was self-selected (both into attendance at the Twinsburg Festival and into the study section of the festival), we may have a disproportionate share of people who "like to participate," indicative of some underlying tendency to associate or form group attachments. This could serve to increase the proportion identifying as strong partisans in our sample as compared to the general population, but it should not bias the relative contributions of genes and environment to that behavior. Any self-selection bias likely applies to DZ and MZ twins equally.

## Results

In the behavior genetics literature, a simple comparison of polychoric correlations is frequently used as a first test of the rate of twin concordance in behavior (for a detailed explanation of this method, see Alford, Funk, and Hibbing 2005). In our observations, the correlation in partisan strength is significantly different ($p$ = .0002) for MZ twins (.46, 95 percent confidence interval [CI] = .34, .57) and DZ twins (.16, 95 percent CI = –.15, .48) (see Table 2). Using the three variance equations noted in the appendix, it is easy to show that[3]

$$\sigma^2_A = 2(COV_{MZ} - COV_{DZ}).$$

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

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

The results suggest that heritability ($h^2$) generates about 46 percent of the variance in strength of partisan attachment. The 95 percent CI for the estimate is (5 percent, 57 percent), indicating that we can reject the hypothesis that genes play no role in partisanship strength. The ACE model also suggests that the environment plays a role, although this is primarily due to the unshared environmental factors ($e^2$), which account for 54 percent of the variance (CI = 43 percent, 67 percent). The shared environment ($c^2$) does not seem to play a significant role (0 percent, CI = 0 percent, 37 percent). Measures of model fit indicate that an AE model is better than the ACE model. All of the models presented include controls for age and gender. These control variables only influence the estimation of the three thresholds and do not enter into the estimation of $a^2$, $c^2$, or $e^2$.

**Table 2**
**Partisanship Strength**

| Model | A | C | E | –2 Log-Likelihood | *df* | $\Delta\chi^2$ | *p* |
|---|---|---|---|---|---|---|---|
| ACE | .46 (.05, .57) | .00 (.00, .37) | .54 (.43, .67) | 1,602.02 | 665 | | |
| AE | .46 (.33, .57) | | .54 (.43, .67) | 1,602.02 | 666 | 0.00 | 1.00 |
| CE | | .40 (.28, .50) | .60 (.50, .72) | 1,606.74 | 666 | 4.72 | .03 |
| E | | | | 1,645.04 | 657 | 43.01 | .00 |

Note: The ACE model consists of additive genetic factors (A), shared or common environmental factors (C), and unshared environmental factors (E). MZ = monozygotic; DZ = dizygotic. There were 278 MZ and 75 DZ twin pairs.

**Table 3**
**Partisanship Direction**

| Model | A | C | E | –2 Log-Likelihood | *df* | $\Delta\chi^2$ | *p* |
|---|---|---|---|---|---|---|---|
| ACE | .30 (.00, .71) | .37 (.00, .68) | .33 (.27, .41) | 2,287.26 | 662 | | |
| AE | .67 (.60, .74) | | .33 (.26, .40) | 2,289.95 | 663 | 2.69 | .10 |
| CE | | .64 (.56, .71) | .36 (.29, .44) | 2,290.05 | 663 | 2.79 | .10 |
| E | | | | 2,434.45 | 664 | 147.20 | .00 |

Note: The ACE model consists of additive genetic factors (A), shared or common environmental factors (C), and unshared environmental factors (E). MZ = monozygotic; DZ = dizygotic. There were 278 MZ and 75 DZ twin pairs.

**Table 4**
**Liberal/Conservative Intensity**

| Model | A | C | E | –2 Log-Likelihood | *df* | $\Delta\chi^2$ | *p* |
|---|---|---|---|---|---|---|---|
| ACE | .13 (.00, .51) | .32 (.27, .48) | .61 (.49, .73) | 1,754.73 | 677 | | |
| AE | .41 (.28, .52) | | .59 (.48, .72) | 1,755.78 | 676 | 1.06 | .30 |
| CE | | .38 (.26, .49) | .62 (.51, .74) | 1,754.95 | 676 | 0.22 | .64 |
| E | | | | 1,791.01 | 678 | 36.28 | .00 |

Note: The ACE model consists of additive genetic factors (A), shared or common environmental factors (C), and unshared environmental factors (E). MZ = monozygotic; DZ = dizygotic. There were 278 MZ and 75 DZ twin pairs.

Previous research indicates a modest genetic influence on vote choice (e.g., choice between Labor versus Conservative parties) but suggests that it could be attributed to an underlying genetic component in perceived attitudes and perceptions that intermediate the relationship with vote choice (Hatemi et al. 2007). Other research indicates that individual differences in political partisanship can be attributed mostly to the environment (Eaves, Eysenck, and Martin 1989; Olson, Vernon, and Jang 2001; Bouchard et al. 2003). To test these theories in our sample, we repeated the analysis using a directional measure of partisanship in Table 3. Although the ACE model was the best fit of any of the models tried, we do not find a significant role for heritability in describing the direction of partisanship. This result is consistent with prior work suggesting the heritability of party choice is low (Alford, Funk, and Hibbing 2005). In other words, although genes appear to play a role in how strongly we attach to a given party, we do not find much evidence that they influence which party will be chosen.

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

strength of ideology is not heritable. A is estimated to be 13 percent but not significantly different from 0. Therefore, our strength of partisanship result cannot be explained as merely the by-product of a genetic contribution to extreme ideological orientations.

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

## Discussion

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

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

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

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

strength could be viewed as the unshared environmental factors that combine with genetic predispositions to change partisanship strength in an individual.

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

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

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

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

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

There are several limitations in our study. First, our sample is relatively small and self-selected. While we do not think that this has biased our analysis in any way, we must keep the limitations of the sample in mind when generalizing to the population at large. Excluding those younger than age eighteen from our sample means that we are not examining the role of heritability in the initial stages of partisan attachment and instead are focusing on the strength

of attachment once it has been formed. Second, our measure of partisan attachment, while frequently employed in the literature, has been subject to the critique that it does not adequately address the theoretical differences between partisans and nonpartisans; partisanship appears to be multidimensional and nonmonotonic (Kamieniecki 1988; Petrocik 1974), and consequently, standard scales of partisanship strength may not adequately address how partisanship is correlated with political behavior. The 7-point scale used in our study may be best for measuring attitudes toward political parties in general because the main differences between subgroups of independent voters is in their orientation toward the symbols of political independence (Craig 1985). However, a scale such as that discussed in Greene (2002) or Weisberg and Hasecke (1999) may better measure the social-psychological identity aspect of partisan strength.

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

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

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

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

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

Thus, the finding that strength of party identification is heritable suggests that more work should be

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

## Appendix
## The ACE Model

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

$$\begin{aligned} \sigma^2_P &= \sigma^2_A + \sigma^2_C + \sigma^2_E \\ COV_{MZ} &= \sigma^2_A + \sigma^2_C \\ COV_{DZ} &= 1/2\sigma^2_A + \sigma^2_C. \end{aligned}$$

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

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

The structural equation specification of our model is as follows:

$$\begin{aligned} \text{VAR(Partisan Strength)} &= a^2 + c^2 + e^2 \\ \text{COV(MZ)} &= a^2 + c^2 \\ \text{COV(DZ)} &= 0.5a^2 + c^2. \end{aligned}$$

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

$$\begin{pmatrix} a^2 \\ c^2 \\ e^2 \end{pmatrix} = \begin{pmatrix} 1 & 1 & 0 \\ 0.5 & 1 & 0 \\ 1 & 1 & 1 \end{pmatrix}^{-1} \begin{pmatrix} COV_{MZ} \\ COV_{DZ} \\ \sigma_{Partisan\ Strength} \end{pmatrix}.$$

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

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

## Notes

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

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

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

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