

## **Is There a “Party” in Your Genes?**

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## **ABSTRACT**

Utilizing quantitative genetic models, we examine the sources of party identification and the intensity of that identification. The results indicate genes exert little, if any, influence on party identification, directly or indirectly through covariates. However, we find that genes appear to play a pivotal role in shaping the strength of an individual's party identification. Together with recent examinations of political attitudes and vote choice, these findings begin to provide a more complete picture of the source of partisanship and the complex nature of the political phenotype.

Party identification is among the most studied concepts in the history of modern political science, particularly for those scholars focusing on political behavior, elections, public opinion and voting. Given the centrality of political parties in structuring vote choice and generally organizing representative democracies (Schattschneider 1957), it is not surprising that scholarly interest in the origins of public feelings toward political parties has been high. *The American Voter* (1960) is only the best known of the scholarly works detailing the nature and sources of party identification (PID). Its publication half a century ago popularized the belief that party identification is acquired early in life, well before children comprehend the policy content of the competing parties. Scholars have long assumed that the early presence of PID was the result of strong parental socialization (Niemi and Jennings 1991). The possibility that partisan identification could be transmitted genetically rather than socially was not considered and left untested. However, science is not built on perceptions alone, particularly when technological advances allow for empirical examination. In this article, we expand upon Alford, Funk and Hibbing's (2005) and Hatemi's (2006, 2007) exploration of genetic influences on partisanship, and test the hypothesis that, in addition to environmental forces, genes may play a role in the direction and intensity of party identification.

### **The Direction and Intensity of Political Affiliation**

Political affiliations of family members including parents, offspring, siblings and spouses are highly correlated (Eaves et al. 1999; Niemi and Jennings 1991). Parental-offspring similarity for political preferences is more than would be expected due simply to shared familial

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environment or socio-economic status (Glass, Bengston and Dunham 1986). This similarity is held up as evidence of familial socialization (Hyman 1959; Campbell et al. 1960; Page and Jones 1979). Jennings, Stoker and Bowers (2001) find “Children are more likely to adopt the partisan orientations of the parent than any other political trait...The high levels of concordance found for partisan orientations compare favorably with those for the religious attributes of church attendance and interpretations of the Bible.” They and others (see Achen 2002) recognize the importance of familial transmission, but do so without considering the possibility that part of the transmission may be genetic.

This is unfortunate because research outside of political science finds a wide variety of social traits, behaviors, and attitudes to be genetically influenced, including church attendance, issue positions, and political ideology (Eaves, Eysenck and Martin 1989). Party Identification (PID) has not played much of a role in this research stream to date (for an exception see Hatemi 2006, 2007). Alford et al. (2005) reported PID was weakly heritable but did not examine the nature of the relationship between political affiliation and important covariates such as policy positions or social indicators, nor model fit to test if genetic influences could be excluded. In addition, the methodology did not include opposite sex twins, roughly 1/3 of the sample. Thus, whether PID is the product of familial socialization alone or in part genetically transmitted, and whether transmission is direct or indirect through related social and political traits is unknown.

Clarifying if inherent sources influence the *direction* of partisanship is important, but equally important and much less researched is the individual variation in the *strength* of partisan attachments (i.e., “partisan intensity”, see Hatemi 2006). Much attention is given to the vote choice of strong and weak partisans, as well as independents (e.g., Keith et al. 1992). However, influences on strength of party identification are largely ignored. Whether Democratic of

Republican, why is it that some people cling to their partisanship with great vigor, but the partisan attachments of others are tepid at best? The general concept of intensity applies to substantive areas spanning many disciplines, and much attention has been directed at it. This is particularly apparent in the personality psychological literature. Personality scales are used to measure the internal affective, cognitive, and motivational processes that provide consistency and continuity in behavior, which constitutes one's personal identity (Caprara and Cervone 2000; Eysenck 1990). It is reasonable to hypothesize that political intensity may come from some component of personality intensity or the same constructs as the tendency to be intense about other identities and affiliations such as religious groups.

### **Behavior Genetics and Biometric Theory**

Behavior genetic techniques have been developed in an attempt to understand why individuals in a population differ from one another (Medland and Hatemi 2009). Analyses are concerned with accounting for variation around a population mean, thereby providing information on *individual differences* in a population. The underlying construct which is responsible for a specific trait value (phenotype) is due to some combination of genetic and environmental influences. Behavior genetic population samples are often centered on twins as well as the family members and peers of twins. The power of these samples is based on the knowledge that monozygotic twins (MZ) develop from a single fertilized ovum, and are genetically identical, whereas, dizygotic twins (DZ) arise from two different ova fertilized by different sperm (Holzinger 1929). As such, DZ twins share on average only 50 percent of their segregating genes, just as is the case for all non-twin full siblings. Using a sample of twin pairs raised by the same parents in the same environment and at the same time provides a natural experiment controlling for familial socialization and other shared environmental influences. This

allows researchers a means to begin to disentangle the influences of genes and the environment (Eaves 1977). If PID or partisan intensity is influenced by genes, the co-twin correlation of MZ twin pairs should be substantially higher than that of DZ twin pairs. However, the classical twin design (CTD) does more than estimate heritability in a population. By sampling individuals who differ in their genetic and environmental relatedness, it is possible to decompose variance into that which is shared between relatives and that which is unique to the individual. The shared variance may be partitioned into that which is due to genetic effects and that which is due to the family or common environment.

The variance components terminology used in behavior genetics is intuitive but is only now becoming more familiar to political scientists (e.g., Alford, Funk and Hibbing 2005; Fowler, Baker and Dawes 2008; Hatemi, Medland and Eaves 2009). “Additive genetic” (A) is simply the combined influence of all genes. “Common environment” (C) is that which is common or shared among family members, including familial and cultural socialization. “Unique environment” (E) includes idiosyncratic personal experiences and all environmental stimuli unique to the individual (in most CTD studies estimates of E also include measurement error).

Maximum likelihood (ML) structural equation modeling (SEM) is the most common form of variance components analysis used for twin samples, though Bayesian methods are also used. Among numerous other benefits, ML/SEM allows for the inclusion of opposite-sex twins pairs and provides the ability to test reduced models that assess the significance of genetic and environmental components. This technique offers a means to test the validity of theories regarding the source of partisanship including beliefs that only environmental forces influence PID, as well as alternative models which include genetic influences.

### **Model Assumptions: Addressing the Critics**

As with any methodological technique, assumptions are built into classical twin design (CTD) variance components analyses. First, the CTD assumes that there are no differences in the means (or prevalence) and variances of the different zygosity groups. These assumptions were tested during preliminary analyses of the data. However, if means differences did exist, a customized model would be needed.

It is also assumed that the magnitude and correlation of shared environmental influences are the same for MZ and DZ co-twin pairs (the “equal environments assumption”). It is well documented that MZ co-twins share more similar environments as children such as dress style, room sharing, and haircuts. However, it is also well-known that these environments have little or nothing to do with most social and psychological traits in adult life (Loehlin and Nichols 1976). So far this holds true for political preferences as well. In a longitudinal study of adolescents combined with an adult cohort study, Hatemi et al. (2008) found no difference in MZ/DZ twin pair correlations through adolescence. Rather, MZ/DZ twin pair correlations only differed in adulthood. Specifically, DZ co-twin correlations significantly dropped once leaving home. This finding is in direct contrast to the belief that unequal environments in childhood influence political preferences differently for each zygosity group as adults. Instead, it is the shared environment which appears to be responsible for greater concordance between DZ co-twins.

Substantial evidence supports the use of the CTD in general, and the implications of unequal environments have been widely discussed with numerous methods employed to test and model potential violations of the assumption (for a review see Medland and Hatemi 2009). However, regardless of wide acceptance of the role of twin methodology (with its limitations) as an initial method to identify genetic influences on behavior, the twin method has met with new resistance in political science (see Charney 2008; Suhay, Kalmoe and McDermott 2007).

Unfortunately, this present debate simply recapitulates that which was exhausted in psychology and psychiatry over 20 years ago and is seemingly unaware of the current literature. The discussion is couched purely in *a priori* terms with no empirical support or attempt to provide a set of testable predictions from any explicit alternative model. This failure appears to violate one of the fundamental criteria for any fertile research program (see Lakatos 1970; Urbach 1974). The disregard for data collection and statistical analyses was explicit (see Charney 2008). This commitment to history and environmental-only models lies in conflict with what is considered scientific progress. Regarding the empirical findings that genes influence political preferences, Charney (2008:311) concludes "... the assumption that political ideologies are genetically transmitted, rather than explaining the phenomena better than, say, traditional historical and cultural and sociological explanations, render them mysterious, if not incomprehensible." Similar views are shared by Suhay et al. (2007:26); with respect to political attitudinal variation "...there is no mystery for genetics to solve".

The CTD is but one of several elements in a research program to address the genetic and social components of individual differences. Others include the study of extended pedigrees and kinships (e.g., Eaves and Hatemi 2008), adoption studies (Mednick, Gabrielli and Hutchings 1984), genetic linkage (e.g., Hatemi 2008) and genome-wide association on non related individuals to name a few. The heuristic that genes influence behavior is unashamedly empirical because that is the nature of science. However, unlike the aforementioned critiques, the heuristic is not vested in any particular conclusion and is designed to be abandoned or modified when the fabric of discovery and new insight tears under the weight of rationalization and post-hoc qualification (e.g., Murphy 1997). The integrated gene-environmental approach is not inherently biased towards the detection of genetic effects nor does it fail to model explicitly the contribution

of a wide range of contextual and social influences. Indeed, there are many examples where there is absolutely no evidence for the influence of genes, and these are reported (e.g., Eaves et al. 2008). Even in cases where the combined evidence for genetic effects from twin, family, adoption studies, and random samples is overwhelming, the number of genes involved turns out to be extremely large and their individual effects astonishingly small (see Visscher 2006).

Apart from concerns about the role of data in science, the consideration of any reference to genetic factors in social behavior has also been dismissed on moral grounds (e.g., Beckwith 1993). Specifically, while arguing genetic methods are acceptable for exploring eating behaviors and sleeping disorders, it has been proposed that utilizing genetics to investigate social human behaviors is inappropriate. Commenting on E.O. Wilson's work, Beckwith and others claim theories that consider genetic influences for complex human behaviors "join the long parade of biological determinists whose work has served to buttress the institutions of their society by exonerating them from responsibility for social problems" (Allen et al. 1975). Such claims have no greater or less substance when applied to so-called "biological determinism" than to research grounded in social, environmental, economic or historical determinism. The problem of "determinism" arises not merely from genetics but from approaches which refuse to explore evidence that supports an alternative theory. If our purpose is to seek to understand why individuals and groups behave the way they do, then dismissal of a research paradigm because it includes endogenous factors is inconsistent with the theory and practice of science.

Ideological and moral objections aside, no single approach can stand by itself. Results from twin models must be considered in the context of their limitations and stand or fall by their consistency with other empirical studies. Since twin samples are not random, and the twin design is utilized to explore individual differences within a population, the results are population

specific and generalizations should not be based upon a single study. However, in spite of plausible *a priori* limitations such as the “equal environments assumption”, twin methods have been greatly elaborated since the early ‘70s and have provided a productive initial platform for further research in medical, behavioral and psychiatric genetics, including the study of gene-environment interaction (e.g., Purcell 2002) and the search for specific environmental factors and individual genes contributing to individual differences. The characteristic findings for different domains have been remarkably consistent across a wide range of samples from different times, populations, and cultures (e.g., combined evidence across populations and age groups has led to a general conclusion that personality is genetic and environmental, Bouchard and McGue 2003).

### **Describing the Sample and the Measurement of Concepts**

The data analyzed in this paper were collected in the mid to late 1980s as part of the Virginia 30,000 Health and Life-Style Survey for Twins (VA30K). Respondents were recruited from a combination of a Virginia population based twin registry (now the Mid-Atlantic Twin Registry) and a volunteer sample from the American Association of Retired Persons. Obtaining a sample size of over 14,000, as we do here, is considered extremely robust for twin studies. Ascertainment, survey instrument, measurement techniques, and other properties of the sample are described in detail by Maes, Neale and Eaves (1997).

PID is assessed in a somewhat different fashion than the norm in modern US studies. The question reads: “write in the number which best describes [your] political affiliation: (1) don’t know (2) always supports Republicans (3) usually supports Republicans (4) varies (5) usually supports Democrats (6) always supports Democrats (7) other (8) prefer not to answer. For direction of party identification (PID), analyses were performed utilizing a collapsed variable in which 2 or 3 = Republican, 5 or 6 = Democrat, and 4 = a middle category of “varies.” Those

who responded “other” or “prefer not to answer” (<10%) were set to missing. The second primary phenotype, partisan intensity, is assessed by folding the answer options to create a partisan intensity item: “usually” and “varies” (responses 3, 4, or 5), were combined to indicate weak/no partisanship, and “always” (2 or 6) were taken to indicate strong partisan affiliation, thus creating a dichotomous variable of high versus low intensity.<sup>1</sup>

A number of sociodemographic variables and political attitudes are related to partisanship (Campbell et al. 1960; Fiorina 1981; Popkin 1991). We employed many of these, including age, income, education, religion, occupation, marital status and church attendance. Political attitudes were assessed with a modified 28-item version of the Wilson-Patterson (1968) Attitudes Inventory (see Table 1 for a complete list of attitude items available in the VA30K and for previously reported heritability estimates of those items). The Wilson-Patterson Attitude Inventory is administered by presenting subjects with a short stimulus phrase such as “death penalty” and asking them to provide a simple “agree,” “disagree,” or “uncertain” response.

(Table 1 about here)

Exploring the relationship between sociodemographic traits, political attitudes, and PID requires selecting the appropriate covariates. Using all issue positions available in a multivariate design would be problematic both for empirical and theoretical reasons. In addition, selecting only those traits identified in the literature as the most significant covariates with PID assumes the VA30K respondents are similar to a nationally representative sample. In order to identify the traits that best correlate with PID and to verify that the relationship between political attitudes and partisanship in this sample is somewhat representative of the voting public during the time of the survey, discriminant function analyses were performed on the 28 items from the political attitude inventory. The standardized discriminant function coefficients serve the same purpose as

beta weights in linear regression and indicate the relative importance of the covariate in predicting the dependent variable (PID).

On the other hand, selection of covariates for partisan intensity was based on established indicators of general intensity common in the personality psychology scholarship. The measures available in our survey include religious service attendance and Eysenck's Personality Quotient (EPQ). There are three main personality factors in the EPQ: psychoticism (versus impulse control), extraversion (versus introversion), and neuroticism or emotional stability (versus instability). Two additional sub factors, impulsivity and social conformity (the "lie" scale), are also available in the sample and included in the analyses. Given their role as covariates, a brief explanation of Eysenck's personality dimensions is warranted.

*Psychoticism* is associated with risk-taking, impulsivity, manipulateness, sensation-seeking, irresponsibility, tough-mindedness and practicality. Psychoticism correlates highly with magical ideation and is a very strong predictor of religious fervor and extreme beliefs. At the extremes, a person with high psychoticism would be troublesome, uncooperative, hostile, and socially withdrawn, whereas a person with low psychoticism would be altruistic, socialized, empathic, and conventional (Eysenck 1990).

*Extraversion* is related to social interest and positive affect which includes activity, sociability, expressiveness, assertiveness, ambition, dogmatism and aggressiveness. Extraversion has two central components: affiliation, which includes valuing close interpersonal bonds, being warm and affectionate, and agency, which includes social dominance, leadership, assertiveness, and tendency to accomplish goals (Depue and Collins 1999). Endogenous contributors of extraversion include cortical arousal stimulating the cerebral cortex and dopamine responsivity, which contributes to people being highly sensitive to incentives (Eysenck 1990).

*Neuroticism* is related to feelings of inferiority, unhappiness, anxiety, dependence, hypochondria, guilt, emotional stability and obsessiveness. Neuroticism is in part based on activation thresholds in the limbic system which regulate emotional states such as sex, fear, aggression, and fight-or-flight responses. Those with high neuroticism experience a fight-or-flight response in the face of minor stressors, while those with low neuroticism require major stress to illicit a fight-or-flight response (Eysenck and Eysenck 1985).

*Impulsivity* originated as a sub factor of extraversion and is best characterized by venturesomeness, low anxiety, lack of inhibition (impulsive behavior), sensation seeking, risk-taking, novelty seeking, adventuresomeness, boldness, boredom susceptibility, and unreliability (Moeller et al. 2001). Finally, *Social Desirability* is characterized by a two-fold nature, 1) social acquiescence or conformity and 2) lack of self insight (Francis, Brown and Pearson 1991).

### **Hypotheses**

The distinction between the concepts of PID and partisan intensity is analogous to the concepts of religious affiliation and religiosity. The former defines the group with which an individual affiliates; the latter, the strength of that affiliation. This is important because previous research on religion discovered that identification with a religious denomination is heavily influenced by environmental forces and influenced hardly, if at all, by genes (Eaves et al. 2008). If a child's parents are Zoroastrian, it is highly likely that the child will also be Zoroastrian for no other reason than that it is the denomination the child sees the parents observing. However, the intensity of those religious attachments (religiosity) is found to have strong genetic precursors (Eaves et al. 1989). Once children leave home, individual differences in religiosity are largely a function of personal experience and genetic influence. Since the underlying construct of party affiliation is similar to denominational affiliation (Jennings et al. 2001), our hypothesis is that

PID is primarily the result of familial socialization and not other latent social or genetic influences. However, this is unlikely to be the case for partisan intensity. Based upon examinations of religiosity, it seems likely that partisan intensity is influenced by genes as well as the environment.

Several of the personality scales appear to be strong candidates for significant relationships with partisan intensity (see McClosky 1958). Extraversion's affiliation and agency aspects, along with dogmatism would appear to influence partisan intensity. The most intriguing scale is psychoticism. Psychoticism's relationship to magical ideation and religious fervor make it an ideal candidate to explain variations in political intensity if the intensity element is the same element as that in religious intensity. Conversely, the only component of neuroticism likely to influence political intensity is obsessiveness, but this would require obsession with politics specifically, which neuroticism does not measure. Thus, neuroticism should account for little of the variance in partisan intensity (unless the majority of the sample is obsessed with politics). Impulsivity appears to run counter to stable intensity and thus should account for little of the variance. Lastly, the relationships of social desirability with partisan intensity would depend heavily on the environment of the individual. With a large and unbiased sample, social desirability is unlikely to have a significant effect on partisan intensity. Thus the more explicit hypothesis is that if personality intensity is related to political intensity, psychoticism and extraversion should have significant and positive relationships with variance in partisan intensity.

### **Procedures**

Polychoric correlations by twin pair zygosity were calculated for each of the traits. Correlations between PID, partisan intensity, socio demographic items, selected personality traits, and political attitudes were also calculated for males and females separately. Significant

twin correlations establish a familial relationship, but they do not distinguish between genetic and environmental effects, or separate the environment into that which is common to the family and that which is unique to the individual. Similar to variance components analyses of voter preference (e.g., Hatemi et al 2007b), using structural equation modeling the variance of the phenotypes is decomposed into additive genetic (A), common environmental (C), and unique environmental (E) influences.

Univariate genetic models using raw data were fit to PID, partisan intensity, sociodemographic indicators, selected political covariates, religiosity, and the personality traits. These models, which adopt a liability threshold that assumes each trait has an underlying normal distribution and at least one cut-point, were fit to the observed frequencies for each of the ordinal traits (e.g., Medland and Hatemi 2009). Thresholds expressed as  $z$  values discriminate between categories that correspond to the frequency of the PID and partisan intensity correlates. Thresholds were tested for similarity across sex and zygosity and corrected for age effects. Mx 1.60 (Neale et al. 2003) was used for genetic model fitting. Correlations between the latent additive genetic factors were 1.0 for monozygotic twins (MZ) and .5 for dizygotic twins (DZ), including opposite sex pairs (OS). Correlations between the latent common environment factors were 1.0 for both MZ and DZ twin pairs (see Figure 1). Because the data of opposite sex DZ twin pairs were available, quantitative sex-limitation models were used to analyze the data. These models assume the same sources of genetic and environmental influence for males and females, but allow for differences in the magnitude of these effects.

(Figure 1 about here)

Multivariate analyses permit determination of both the sources of covariation and the structure by which the related phenotypic traits influence PID and partisan intensity. Covariance

matrices between variables were computed by PRELIS 2 on twin pairs with complete data. Multivariate designs often use correlation matrices due to the numerical problems and extremely long run-times of analyses with large numbers of categorical variables (e.g., Hatemi et al. 2007b). Cholesky decomposition was performed on the correlation matrices to assess the extent to which the genetic and environmental components of PID were explained by (1) the genetic and environmental influences shared with the selected sociodemographic indicators and political attitudes and (2) the genetic and environmental influences specific to PID or partisan intensity (depending on the model). The Cholesky is a fully saturated factorization of the data which has as many latent factors per variance component as there are variables. The first factor loads on all variables in the analysis. The second variable in the model is assumed to be caused by a second latent factor that also explains part of the variance of all variables except the first, and so on, with the last factor loading only on the last variable (Loehlin 1996). PID and partisan intensity are entered as the last variable in each of the separate Cholesky decompositions, thereby defining the model to explain the genetic and environmental variance of these two primary phenotypes. The final latent factor explains any remaining variance of PID (or partisan intensity) not accounted for by their covariates (see Figure 2).

(Figure 2 about here)

In order to determine the importance of the ACE components, the full models in both the univariate and multivariate analyses were tested against reduced models in which the A or C matrices of factor loadings were fixed to 0. Nested models equating the path coefficients for males and females were also examined and compared to the full model in order to test whether sex-specific differences in the magnitude of the variance components provide a better model fit than when sex differences are ignored (e.g., Hatemi et al. 2009). Model fitting provides a means

to hypothesis test. Specifically, in order to test for genetic influences on PID and partisan intensity, the fit of the full Cholesky (ACE) was compared to the fit of a model in which all genetic influences on PID or partisan intensity were set to 0 (CE) and to a model where all common environmental influences were set to 0 (AE).

The nested or reduced models were compared to the saturated model using likelihood ratio tests ( $\Delta-2LL$ ), where a significant increase in  $-2LL$  indicates a worsening of model fit. As the resulting  $-2LL$  is chi-square distributed, the goodness of fit of the model can be assessed by comparing the  $-2LL$  with the degrees of freedom being equal to the difference between the number of parameters estimated in the different models. A non-significant difference in chi-square is indicative that the more parsimonious model is a better fitting model.

## **Results**

Responses to the partisan affiliation item were offered by 13419/14761 (91%) of the twins in the sample. Because of missing values for covariates, and the use of only Republican and Democrat affiliation for the PID analyses, the numbers for the PID analyses will be smaller. Using only twins, Republicans accounted for 40% of the sample, “varies” 34% and Democrats 26% (numbers are reflective of the respondents being concentrated in the more conservative Southern region during the Reagan era, and of the older AARP cohort). Demographics within the dependent categories are presented in Table 2 and indicate the sample that is more female, older, Republican, religious and Protestant than national averages. However as implied, variance components analyses focus on variances, not means. Thus, while population-based samples may be biased for mean differences compared to other populations, they are remarkably robust for variance differences within populations (see Neale et al. 1989).

(Table 2 about here)

The phenotypic correlations by twin pair zygosity are shown in Table 3. Correlations are higher for MZ pairs than DZ pairs in all traits—though as expected the differences are small for PID. There are some substantial differences in the correlations of opposite sex pairs compared to same sex DZ pairs, giving cause to suspect that sex differences in the magnitude of genetic and environmental influences may be present.

(Table 3 about here)

Attitudes toward unions, school prayer, nuclear power, gay rights, death penalty, federal housing, Moral Majority, women’s liberation, socialism and busing had the lowest Wilks’ Lambdas and the highest standardized function coefficients, indicating they were the strongest discriminators between Republican and Democratic supporters in our sample. Republican supporters tended to be in favor of the death penalty, school prayer, and Moral Majority, and against unions, gay rights, and federal housing. Democratic supporters were more favorable to women’s liberation, socialism, busing, and gay rights and opposed to school prayer. However, discriminant analyses did not provide reliable covariates distinguishing Democrats and Republicans from those who responded “varies.” Based upon these results, the following six variables were identified as most strongly associated with partisan support: church attendance, attitudes on school prayer, gay rights, death penalty, unions, and federal housing. Correlations between these covariates and the dependent phenotypes are provided in Tables 4a and 4b.<sup>2</sup>

(Tables 4a and 4b about here)

The findings in the preliminary and discriminant analyses add support to the notion that for political attitudes the VA30K respondents are in many ways similar to the general voting public in the 1980s. The major issue differences between the parties in the 1980s (e.g., Popkin 1991) are also those identified by the statistical analyses performed on this sample. Initially, we

included those who responded “varies” in both the PID and partisan intensity analyses; however, while the “varies” respondents make up a significant portion of the sample, the combination of the question wording (“varies”) with the lack of issue differentiation from the two major parties does not allow for a consistent ordinal positioning of the group in the direction of party affiliation (PID) theme. Since the “varies” category includes members that cannot be distinguished from either party, even through covariates, variance components analyses for PID using the “varies” respondents introduce theoretical and empirical concerns, and would be difficult to interpret. Therefore all further analyses for the PID phenotype (direction of affiliation but not the strength of partisan intensity phenotype) were performed only on those respondents who placed themselves in one of the two major party categories.

Univariate models containing additive genetic, common environmental, and unique environmental variance components were fitted to determine which model best explains PID, correcting for age (Table 5). As expected, the full model indicates only modest genetic influence (.08 and .12 in females and males respectively), with confidence intervals that include a zero bound. The model containing only common environmental and unique environmental components (CE) for both females and males was not significantly different ( $p=.31$ ,  $\Delta X^2=2.34$ , 2 *d.f.*) from the fit of the full model (ACE) and provided a more parsimonious model, demonstrating that no significant additive genetic influence for PID is significantly present. Rather, common environmental influences are sufficient to explain familial resemblance (.81). These results further elucidate Alford et al’s (2005) analyses which, using only same sex twin pairs, found that the genetic influence on PID was minor (.14) in comparison to common and unique environmental influences. The saturated model employed here utilizing the full sample of twins provides even lower additive genetic estimates. Once model fitting is taken into account,

the results suggest genetic sources of variance need not be considered to explain individual differences in party identification.

(Table 5 about here)

Whether familial socialization factors are acting indirectly on PID through sociodemographic and political attitudes, or whether genetic influences from related covariates indirectly affect PID is further clarified through multivariate analysis. The results from the Cholesky show that all genetic influences on PID can be dropped without significantly worsening model fit ( $\Delta X^2 = 16.54$  for 14 *d.f.*). However, dropping the genetic or common environmental effects on all 6 covariates (school prayer, gay rights, death penalty, Unions, federal housing and religiosity) significantly worsened model fit. Standardized principal components from the model with no genetic effects on PID are shown in Table 6.

(Table 6 about here)

Previous research reported that each of the covariates contained significant genetic effects (see Table 1). However, in the fully saturated model these covariates account for little of the overall variance as all genetic influences on PID can be dropped. Equally important, the covariates also account for little of the common environmental variance in PID. It appears that PID is largely due to familial socialization and that this socialization is directed toward PID itself ( $C^2 = .747^2$  or 56%). A final “unique” environmental component for PID is also present. After all other unique environmental variance is accounted for by correlates, the unique environment (E) specific to PID accounts for 25% ( $.507^2$ ) of the variance.

Whereas a majority of the political attitudes are moderately influenced by genes, those attitudes are only able to explain a small amount of the variance in PID. This finding is quite different from Hatemi et al’s (2007b) examination of vote choice. That is, people are socialized

to favor one party or the other specifically. Both the socialization and personal experience variance related to PID are not a byproduct of some other common experience, unique experience, or latent genetic factor. The attitude items also have a weak common environmental (C) relationship to PID as none of the covariates explains more than 10% of the variance for C. Thus, the data do not support the inference that salient issue positions provide a significant amount of the familial influence on party identification (e.g., Achen 2002; Fiorina 1981). Rather, the common environment (C) is by far the greatest source of variance (81%) and C is largely specific to PID itself (56%) and not to key issue positions. This also suggests that PID's influence on issue positions through socialization is minor.

Shifting to partisan intensity, the univariate results confirmed our expectations; individual differences in partisan intensity are genetically influenced. The preferred model for partisan intensity is an additive genetic and unique environmental model (Table 5). Dropping the common environmental variance and equating male and female paths provided a better fitting and more parsimonious model ( $\Delta X^2 = 3.08$  for 1 *d.f.*). Half of the variance is accounted for by genes and half by unique experience, but in contrast to PID, no significant amount is accounted for by familial socialization.

Multivariate analyses clarified if the genetic variance was specific to partisan intensity or if the genetic variance was merely measuring a latent construct expressed through partisan intensity. The best fitting multivariate model for partisan intensity was a reduced ACE model. Removing the additive genetic (A) or common environmental (C) influence for all items significantly worsened model fit for both males and females. However, removing the C path specific to partisan intensity did not provide a significantly worse fit and was more parsimonious ( $\Delta X^2 = 18.99$  for 14 *d.f.*).

Standardized principal components for the reduced Cholesky are shown in Table 7. In the reduced model, the remaining genetic influence specific to partisan intensity (A7) accounts for 27% and 22% ( $.516^2$  and  $.467^2$ ) of the genetic variance in females and males respectively, and roughly 70% of the unique environmental variance. Surprisingly, neither religiosity nor all personality traits combined shared more than 5% of the genetic variance with partisan intensity. Indeed, the strongest influence outside of the residual variance due to partisan intensity itself is extraversion in males, accounting for a paltry 2% of the genetic variance. However, the unique environmental influences between partisan intensity and personality are partially shared.

(Table 7 about here)

A majority of the genetic variance in partisan intensity remained specific to itself, suggesting that the genetic influence of partisan intensity is not related to religiosity or the intensity elements in the personality facets used here—though it certainly could be related to other traits (e.g., traits specifically focused on intensity or the desire to belong perhaps).

## Discussion

In evaluating an early paper on the possible role of genes on behavior, an anonymous referee commented to the effect that, “it is probably alright to use the twin study to estimate the genetic contribution to variables which you *know* are genetic like stature and weight, and it’s probably alright for things like blood pressure. But it certainly can’t be used for behavioral traits which we *know* are environmental like social attitudes” (quoted in Neale and Cardon 1992). Our view is somewhat different. The twin study provides us with clues to approaches when we believe we don’t know the answer, or when there may be reason to doubt answers commonly accepted on *a priori* grounds.

Twin studies do not pretend to offer an exhaustive analysis of all the potential subtleties of the action and interaction of social and genetic influences. The CTD and associated linear structural models provide an initial decomposition of individual differences into broad genetic, common and unique environmental components. The genetic and social consequences of assortative mating, genotype x environment interaction (GxE) and genotype-environment correlation (rGE) are confounded with the estimates of the principal variance components (Eaves 1982; for an example of modeling rGE on political preferences see Eaves and Hatemi 2008). There is no single direct path from genes to behavior. Rather, they are likely to be numerous and convoluted. Any sophisticated behavior is far too complex to fully explain in discrete terms. However, estimates of variance components are no more absolute than estimates of regression coefficients in typical social science models. In the “real” world behavior is not divided into perfect buckets of genes and environment, nor is a regression coefficient a definitive predictive estimate. Interpretation of results is based on the models employed and covariates used, with accepted limitations. Our univariate and multivariate analyses are based on the initial “ACE” model widely used across disciplines. They constitute an invitation to explore a paradigm until it fails to account for significant features of future data. Other models and measures might yield more specific interpretations and predictions. Establishing general estimates of genetic and environmental influence for PID and partisan intensity paves the way to consider models in which different genes and environments influence males and females, to include non-additive genetic effects, dominance, effects of sibling imitation and contrast, GxE, rGE, as well as more complex models which try to identify the specific genes or environments involved in particular neurochemical processes that mediate the effects of genetic differences upon the behavioral

phenotypes. The analysis of the roles of genes and environment is progressive. Success is judged by the cumulative weight of coherent findings. The analyses here are only the beginning.

The findings themselves point towards an integrated model in which both social and genetic factors play their own distinct role. Here we provide evidence supporting the longstanding political science literature theorizing that party identification is based on social transmission. Until now, that assumption has never been empirically verified using a multivariate model that controlled for genetic influences. Direction of PID is almost entirely driven by familial socialization without any involvement of genetic transmission. Parents socialize their children to become Republicans or Democrats and this socialization has a lasting, though not immutable, impact on their affiliation well into adulthood, regardless of personal experiences or ideological issue positions developed later in life (e.g., Goren 2005).

The data suggest, however, that the source of partisan intensity is quite distinct and influenced in part by genetic differences comparable to, but different from, those that have long been regarded as constitutive of differences in personality. Individual differences in partisan intensity result equally from genetic and unique experiential influences while familial socialization has little significance. With regard to political party affiliation, people appear to be influenced by a biological propensity to be intense or apathetic regardless of how they were raised or which party they were raised to support. It appears clear that neither personality (as measured here) nor religious intensity is genetically, or to a large degree environmentally, related to partisan intensity. Whether there is truly a unique genetic influence on the intensity of partisan support remains to be seen. Future analyses are required to determine if intensity of attachment to a political party is truly different from all other types of intensity or group support.

It is critical to avoid over-simplification in the understanding of genetic sources of variation. There is no gene “for” being a Republican or Democrat or for voting a certain way or for an opinion on abortion, or any other complex trait. Rather, we are exploring the underlying multidimensional mechanisms and multifactorial liabilities that accumulate to influence individual responses to political aspects of contemporary society. Such behavior cannot exist without a culture any more than it can without a genome.

All social behaviors take place in a social context. In the United States, even our “fallible” twin data confirm that the origin of party identification is almost exclusively cultural. PID is a component of group identification, and in general far more socialized than any individual issue position. Party identification encompasses a social identity that lay far beyond mere political preference. People are raised to be Democrats or Republicans as much as they are raised to be Catholic or Protestant. Indeed, our results for PID are comparable to those from extensive twin data that show the origins of religious affiliation are entirely social and not in the least genetic (Eaves et al. 2008). That is, in the few cases where the answer might seem to be obvious, twin data yield results that are entirely consistent with expectations. However, the intensity of commitment to a party reveals a markedly different pattern. Partisan intensity is heavily influenced by genetic liability but influenced very little by familial socialization. Which party one chooses and how much they are willing to hold to that party may be related as far as voter outcomes, but appear to be derived from two different, possibly not unrelated mechanisms. Parents influence the party with which their children identify but have little, if any, social control over how zealous their offspring becomes.

Every complex behavior is comprised of an endless number of factors for any living organism; behavior is not pre-determined, nor is it uninfluenced by genes. Rather, the

foundations of human behavior include some function of genes, development, socialization and environmental stimuli, embedded in an evolutionary framework (Dobzhansky 1973). We are still beginning the application to political differences a heuristic and methodology that has proved productive in many other areas of human research, including social behavior and its disorders. Only time and data will tell if the situation is similar in the political arena. If these early findings prove to be incorrect, the issue must be decided by the data and not by personal preference, attachment to existing methods, political correctness, or by appeal to history and past authority. Science is self-correcting. So far, such data as we have suggest that a “partially genetic” theory accounts for many features of the data, including some that have not received much attention from the social sciences (i.e., genes are relevant to political behavior). Results from twin, family and genotypic data at least present an inconvenient truth for a purely social theory. We can choose to ignore the findings, or can explain it away by ad hoc appeals, but in doing so we might close off a whole area of quite revolutionary understanding. Bertrand Russell (2004: 78) wrote, “We cannot know in advance that the truth will turn out to be what is thought edifying in a given society.” Science allows us, and sometimes compels us, to think the unthinkable. For Darwin (1872) it took nerve to imagine the alternative to the established paradigm. Only time and data can decide whether our heuristic has lasting value. Our results imply that political behavior will not be understood fully without reference to endogenous factors. Doing so will require certain adjustments in our usual disciplinary procedures but holds the promise of creating a valuable interdisciplinary bridge between the life and the social sciences and between genes and culture, in the study of a concept which remains at the very center of modern electoral politics.

## NOTES

<sup>1</sup> In the absence of a generally approved coding procedure for the data, we opted for the coding that was both the most theoretically and psychometrically sound. The alternative of utilizing a 3-point version of partisan intensity that separated “varies” into its own category was more normally distributed, but provided minimal difference in results. In addition, we also considered analyses which combined “usually” and “always” versus all others; but this coding choice was not normally distributed and theoretically implausible.

<sup>2</sup> Nuclear Power was a strong discriminator, but loaded on the same factor as defense, draft, and military drill. Since it was intended to measure opinions on alternative energy, it was not used in further analyses.

## REFERENCES

- Achen, Christopher H. 2002. Parental Socialization and Rational Party Identification. *Political Behavior* 24(2):151-170.
- Alford, John, Carolyn Funk, and John Hibbing. 2005. Are Political Orientations Genetically Transmitted? *American Political Science Review* 99(2):153-167.
- Allen, Elizabeth, Jon Beckwith, Barbara Beckwith, Steven Chorover, David Culver, Margaret Duncan. 1975. Against 'Sociobiology.' *The New York Review of Books* 22(18).
- Beckwith, Jon. 1993. A historical view of social responsibility in genetics. *BioScience* 43:327-333.
- Bouchard, Thomas, Jr., and Matt McGue. 2003. Genetic and Environmental Influences on Human Psychological Differences. *Journal of Neurobiology* 54 (1): 4-45.
- Caprara, G. V., and Cervone, D. 2000. *Personality: Determinants, dynamics and potentials*. Cambridge: Cambridge University Press.
- Campbell, Angus, Philip E. Converse, Warren E. Miller, and Donald E. Stokes. 1960. *The American Voter*. New York: Wiley.
- Charney, Evan. 2008. Genes and Ideologies. *Perspectives on Politics* 6(2):292-319.
- Darwin, Charles. 1872. *The Expression of Emotions in Man and Animals*. Oxford: Oxford University Press.
- Depue, Richard A. and Paul F. Collins. 1999. Neurobiology of the Structure of Personality: Dopamine, Facilitation of Incentive Motivation, And Extraversion. *Behavioral Brain Science* 22(3):491-517.
- Dobzhansky, Theodosius. 1973. Nothing in Biology Makes Sense Except in the Light of Evolution. *American Biology Teacher* 35: 125-129.
- Eaves, Lindon J. 1977. Inferring the Causes of Human Variation. *Journal of the Royal Statistical Society* 140(3): 324-155.
- Eaves, Lindon J. 1982. The utility of twins. In *Genetic basis of the epilepsies*, Eds. V. E. Anderson, W. A. Hauser, J. K. Penry and C. F. Sing. New York: Raven Press.
- Eaves, Lindon J and Peter K. Hatemi. 2008. Transmission of attitudes toward abortion and gay rights: Parental socialization or parental mate selection? *Behavior Genetics* 38:247-256.
- Eaves, Lindon J, Peter K. Hatemi, E.C. Prom and E.L. Murrelle. 2008. Social and Genetic Influences on Adolescent Religious Attitudes and Practices. *Social Forces* 86(4):1621-1646.

- Eaves, Lindon, Andrew Heath, Nicholas Martin, Hermine Maes, Michael Neale, Kenneth Kendler, Katherine Kirk and Linda Corey. 1999. Comparing the biological and cultural inheritance of personality and social attitudes in the Virginia 30 000 study of twins and their relatives. *Twin Research* 2:62–80.
- Eaves, L. J., H. J. Eysenck, and Martin, N.G. 1989. *Genes, Culture and Personality; an Empirical Approach*. London, Academic Press.
- Eysenck, H. J., and Eysenck, M. W. 1985. *Personality and individual differences: A natural science approach*. New York: Plenum.
- Eysenck, H. J. 1990. Biological dimensions of personality. In *Handbook of personality: Theory and research*, ed. L. A. Pervin. New York: Guilford.
- Fiorina, Morris P. 1981. *Retrospective Voting in American National Elections*. New Haven: Yale University Press.
- Fowler, James H., Laura A. Baker, and Christopher T. Dawes. 2008. The Genetic Basis of Political Participation. *American Political Science Review* 102 (2).
- Francis, LJ, LB Brown and PR Pearson. 1991. The dual nature of the EPQ lie scale among university students in Australia. *Personality and individual differences* 12(10):989-991.
- Glass, Jennifer, Vern L. Bengtson and Charlotte C. Dunham. 1986. Attitude Similarity in Three-Generation Families: Socialization, Status Inheritance, Reciprocal Influence? *American Sociological Review* 51(5): 685-698.
- Goren, Paul. 2005. Party Identification and Core Political Values. *American Journal of Political Science* 49 (4): 881-96.
- Hatemi, Peter K. 2006. The Genetics of Party Identification and Vote Choice. Presented at the Hendricks Conference on Biology, Evolution, and Political Behavior, Lincoln, NE.
- Hatemi, Peter K. 2007. *The Genetics of Political Attitudes*. PhD Dissertation, University of Nebraska-Lincoln
- Hatemi, Peter K. 2008. The pathway from biology to political behavior: initial findings of genetic association and linkage studies for political preferences. Presented at the Annual Meeting of the American Political Science Association, Boston.
- Hatemi, Peter K., Sarah E. Medland and Lindon J. Eaves. 2009. Genetic Sources for the Gender Gap? *Journal of Politics* (Forthcoming).
- Hatemi, Peter K., Cary L. Funk, Sarah E. Medland, Hermine H. Maes, Judy Silberg, Nicholas G. Martin and Lindon J. Eaves. 2008. Genetic and Environmental Transmission of Political Attitudes Across the Life-course. Presented at the Annual Meeting of the International Society of Political Psychology, Paris.

- Hatemi, Peter K., John Hibbing, John Alford, Nicholas Martin and Lindon Eaves. 2007. "We Get Opinions from Our Parents, but Not How We Think We Do: Genetic and Social Components of the Familial Transmission of Political Attitudes" paper presented at the Midwest Political Science Association, Chicago.
- Hatemi, Peter K., Sarah E. Medland, Katherine I. Morley, Andrew C. Heath, and Nicholas G. Martin. 2007b. The Genetics of Voting: An Australian Twin Study. *Behavior Genetics* 37:435–448.
- Holzinger, Karl J. 1929. The relative effect of nature and nurture influences on twin differences. *Journal of Educational Psychology* 20:241-248.
- Hyman, Herbert. 1959. *Political socialization*. Glencoe, Illinois: The Free Press.
- Jennings, Kent M., Laura Stoker and Jake Bowers. 2001. Politics Across Generations: Family Transmission Reexamined. Institute of Governmental Studies. Paper WP2001-15. <http://repositories.cdlib.org/igs/WP2001-15>.
- Keith, Bruce E., David B. Magleby, Candice J. Nelson, Elizabeth Orr, Mark C. Westlye, and Raymond E. Wolfinger. 1992. *The Myth of the independent voter*. Berkeley, California: University of California Press.
- Kendler K.S. and J. Gardner. 1998. "win studies of adult psychiatric and substance dependence disorders: are they biased by differences in the environmental experiences of monozygotic and dizygotic twins in childhood and adolescence? *Psychological Medicine* 28:625-633.
- Lakatos, Imre. 1970. Falsification and the Methodology of Scientific Research Programmes. In *Criticism and the Growth of Knowledge*, eds. Imre Lakatos and Alan Musgrave. London, UK: Cambridge University Press.
- Loehlin, John C. 1996. The Cholesky Approach: A Cautionary Note. *Behavior Genetics* 26(1).
- Loehlin, J.C., and R.C. Nichols. 1976. *Heredity, environment, and personality: A study of 850 sets of twins*. Austin, TX: University of Texas Press.
- Maes, Hermine H., Michael C. Neale and Lindon J. Eaves. 1997. Genetic and Environmental Factors in Relative Body Weight and Human Adiposity. *Behavior Genetics* 27:325-351.
- Martin, N.G., L.J. Eaves, A.C. Heath, R. Jardine, , L.M. Feingold and H.J. Eysenck. 1986. Transmission of social attitudes. *Proceedings of the National Academy of Science* 83: 4364-4368.
- Medland, Sarah E. and Peter K. Hatemi. 2008. Political Science, Behavior Genetics and Twin Studies: a Methodological Primer. *Political Analysis* (Forthcoming)

- Mednick, S.A., W.F. Gabrielli and B. Hutchings. 1984. Genetic influences in criminal convictions: Evidence from an adoption cohort. *Science* 224: 891-894.
- McClosky, Herbert. 1958. Conservatism and personality. *American Political Science Review* 52:27-45.
- Moeller, Gerard, Ernest S. Barratt, Donald M. Dougherty, Joy M. Schmitz, and Alan C. Swann. 2001. Psychiatric Aspects of Impulsivity. *American Journal of Psychiatry* 158:1783-1793.
- Murphy, Nancy. 1997. *Anglo-American Post-Modernity, Philosophical Perspectives on Science, Religion, and Ethics*. New York: Westview Press
- Neale, M., S. Boker, G. Xie, and H. Maes. 2003. *Mx: Statistical modeling* (5th ed.). Richmond, VA: Department of Psychiatry, Virginia Commonwealth University.
- Neale, Michael C. and Lon R. Cardon. 1992. *Methodology for genetic studies of twins and families*. Dordrecht, The Netherlands: Kluwer Academic Publishers.
- Neale, Michael C., Lindon J. Eaves, Kenneth S. Kendler and John K. Hewitt. 1989. Bias in correlations from selected samples of relatives. *Behavior Genetics* 19: 163-169.
- Niemi, Richard G., and M. Kent Jennings. 1991. Issues and Inheritance in the Formation of Party Identification. *American Journal of Political Science* 35:970-88.
- Page, Benjamin and Calvin Jones. 1979. Reciprocal Effects of Policy Preferences, Party Loyalties and the Vote. *American Political Science Review* 73:1071-1090.
- Popkin, Samuel L. 1991. *The Reasoning Voter*. Chicago: University of Chicago Press
- Purcell S. 2002. "Variance components models for gene-environment interaction in twin analysis." *Twin Research* 5(6):554-71.
- Russell, Bertrand. 2004. *History of Western Philosophy*. New York: Routledge.
- Schattschneider, Elmer E. 1957. Intensity, Visibility, Direction and Scope. *The American Political Science Review* 51(4): 933-942.
- Suhay, Elizabeth, Nathan Kalmoe and Christa McDermott. 2007. Why Twin Studies Are Problematic for the Study of Political Ideology: Rethinking Are Political Orientations Genetically Transmitted? presented at the International Society of Political Psychology.
- Urbach, P. 1974. Progress and Degeneration in the IQ Debate. *British Journal for the Philosophy of Science* 25:93-135.
- Wilson, G. and Patterson, J. 1968. A new measure of conservatism. *British Journal of Social and Clinical Psychology* 7: 264-269.

**Table 1: Various Estimates of the Heritability of Political and Social Attitudes from the VA30K Wilson Patterson Index.**

Item	Polychoric Correlations Method- Same Sex Twins (Alford et al 2005)		ML/SEM Method- All Twins (Hatemi 2007)	
	A <sub>m</sub>	A <sub>f</sub>	A <sub>m</sub>	A <sub>f</sub>
Death Penalty	.27	.35	.36	.34
Astrology	.49	.33	.47	.34
X-rated movies	.47	.38	.51	.43
Modern Art	.31	.21	.39	.26
Womens' liberation	.23	.35	.31	.34
Foreign aid	.25	.41	.31	.40
Federal housing	.05	.29	.01	.32
Democrats	.28	.23	--	--
Military drill	.42	.24	.38	.16
The draft	.39	.39	.41	.32
Abortion	.26	.24	.38	.26
Property Tax	.57	.32	.43	.36
Gay rights	.41	.22	.43	.24
Liberals	.08	.22	--	--
Immigration	.29	.35	.23	.45
Capitalism	.52	.33	.62	.33
Segregation	.34	.24	.36	.22
Moral Majority	.44	.38	.32	.43
Pacifism	.46	.34	.34	.30
Censorship	.55	.20	.40	.20
Nuclear Power	.51	.13	.43	.16
Living together	.11	.39	.00	.51
Republicans	.48	.30	--	--
Divorce	.20	.28	.42	.25
School prayer	.44	.40	.47	.32
Unions	.43	.34	.28	.42
Socialism	.33	.36	.28	.36
Busing	.18	.31	.12	.31

**Notes:** A<sub>m</sub>=additive genetic effects for males; A<sub>f</sub>=additive genetic effects for females

**Table 2.** Demographics by Party Identification and partisan intensity (%)

	Party Identification			Partisan Intensity		
	Republican	Varies	Democrat	Strong	Weak	No Affiliation
<b>Strong Affiliation</b>	31.3	-	23.4	-	-	-
<b>Weak Affiliation</b>	68.7	-	76.6	-	-	-
<b>Age</b>						
Mean	51	46	53	56	54	46
SD	18	16	17	19	18	17
<b>Sex</b>						
Male	44	40	38	35	37	37
Female	56	60	62	65	63	63
<b>Education</b>						
< 7 years	0.9	0.9	2.2	1.9	1.0	0.8
8 years	2.2	1.5	3.4	4.0	3.0	1.7
9-11 years	6.0	7.3	9.5	9.7	7.7	7.9
High School Diploma	26.4	30.9	28.4	34.6	30.5	34.4
College (1-3 years)	27.5	27.7	21.5	26.3	25.6	27.0
College graduate	37.1	31.8	35.0	23.5	32.3	28.3
<b>Income</b>						
< 5000	1.7	1.4	2.1	3.0	2.2	1.7
<10000	2.7	3.0	4.5	5.7	4.1	3.9
<15000	5.8	6.6	8.7	9.5	8.1	7.7
<20000	7.5	7.7	9.2	10.2	9.6	9.5
<25000	9.0	10.6	10.7	11.5	10.4	11.3
<35000	18.4	21.3	19.9	19.2	19.2	20.7
<50000	21.7	23.8	20.3	18.7	20.8	23.0
>50000	33.1	25.5	24.6	22.3	25.5	22.2
<b>Religion</b>						
Catholic	12.4	15.9	20.1	14.7	15.8	15.4
Protestant	75.6	65.6	55.4	69.4	68.0	64.3
Jewish	1.2	3.2	9.2	3.0	4.7	3.5
Other	8.0	11.0	8.4	10.3	8.0	13.0
None	2.8	4.3	6.9	2.7	3.5	3.6
<b>Church attendance</b>						
2+ per Week	17.3	14.0	13.1	17.3	16.7	14.3
Weekly	32.0	27.1	28.7	31.1	31.5	27.7
Monthly	11.5	11.5	10.2	10.3	10.7	11.2
Yearly	15.9	18.7	18.4	16.7	16.4	18.5
Rarely	17.3	21.2	19.8	17.6	18.3	21.1
Never	6.0	7.5	9.8	7.0	6.3	7.1
N	5415	4554	3450	2707	6158	4554

**Table 3:** Twin Correlations for Voting, Sociodemographic Traits, Political Attitudes and Personality Scales

	<b>MZF</b>	<b>DZF</b>	<b>MZM</b>	<b>DZM</b>	<b>DZOS</b>
Party ID (PID)	0.84	0.80	0.82	0.79	0.76
Partisan Intensity (PI)	0.48	0.41	0.48	0.24	0.24
Church Attendance	0.75	0.51	0.60	0.48	0.38
School Prayer	0.66	0.47	0.65	0.45	0.42
Gay Rights	0.61	0.49	0.58	0.38	0.36
Death Penalty	0.56	0.38	0.54	0.43	0.29
Unions	0.44	0.26	0.43	0.24	0.13
Federal Housing	0.43	0.26	0.25	0.23	0.15
Neuroticism	0.41	0.25	0.38	0.14	0.12
Social Desirability	0.54	0.32	0.44	0.31	0.25
Extraversion	0.51	0.11	0.46	0.16	0.12
Impulsivity	0.31	0.11	0.31	0.19	0.10
Psychoticism	0.35	0.13	0.30	0.18	0.13
N Pairs <sup>a</sup>	2029	1273	826	610	1397

**Notes:** (a) Correlations were estimated for full information maximum likelihood observations on incomplete pairs. Due to missingness cases range as follows: MZF (1774-2029), DZF (1054-1273), MZM (745-826), DZM (527-610), DZOS (1219-1397).

**Table 4 A:** Correlations<sup>a</sup> between Party ID, Attitudes, and Religiosity Covariates; Males Upper Triangle, Females Lower Triangle.

	<b>Males</b>							
	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>	<b>5</b>	<b>6</b>	<b>7</b>	<b>8</b>
1. Party ID	.	.16	.39	-.39	.46	-.47	-.35	.09
2. Religiosity	.15	.	.35	-.26	-.03	-.07	.03	-.20
3. School Prayer	.39	.37	.	-.55	.39	-1.06	-.19	-.13
4. Gay Rights	-.34	-.38	-.55	.	-.43	.24	.36	.10
5. Death Penalty	.32	-.06	.26	-.24	.	-.24	-.31	.01
6. Unions	-.41	-.11	-.11	.24	-.18	.	.32	.07
7. Federal Housing	-.30	-.04	-.20	.34	-.19	.29	.	-.04
8. Age	.03	-.22	-.15	.17	.07	.09	-.02	.
	<b>Females</b>							

**Table 4 B:** Correlations<sup>a</sup> between Strength in Party ID, Religiosity and Personality Subscales; Males Upper Triangle, Females Lower Triangle.

	<b>Males</b>							
	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>	<b>5</b>	<b>6</b>	<b>7</b>	<b>8</b>
1. Partisan Intensity	.	.19	.08	.05	.05	.07	.11	-.15
2. Religiosity	.18	.	.11	.05	.05	.10	.11	-.11
3. Neuroticism	.09	.07	.	.08	.14	.32	.18	-.07
4. Social Desirability	.02	.05	.04	.	.30	.13	.33	.14
5. Extraversion	.03	.05	.05	.33	.	.36	.31	-.02
6. Impulsivity	.05	.04	.21	.15	.35	.	.27	-.07
7. Psychoticism	.08	.09	.13	.34	.34	.24	.	-.04
8. Age	-.16	-.07	-.11	.24	.06	.01	-.05	.
	<b>Females</b>							

**Notes:** (a) casewise deletions

**Table 5.** Standardized Variance Components Sex-Limitation Model Fitting for PID and partisan intensity (95% confidence intervals); Thresholds Corrected for Age and Sex.

Model	Parameter Estimates						-2LL	$\Delta X^2$	$\Delta df$	p-value (comparison model)
	Females			Males						
	$a^2$	$c^2$	$e^2$	$a^2$	$c^2$	$e^2$				
<b>Party Identification</b>										
ACE	.08 (0-.24)	.76 (.61-.86)	.17 (.12-.21)	.12 (0-.30)	.71 (.54-.71)	.18 (.15-.26)	8736.41	-	-	-
ACE (M=F)	.10 (0-.21)	.74 (.63-.82)	.17 (.13-.21)	.10 (0-.21)	.74 (.63-.82)	.17 (.13-.21)	8736.80	0.39	3	.94 (ACE)
<b>AE</b>	.34 (0-.50)	-	.66 (0-.50)	.34 (0-.50)	-	.66 (0-.50)	8862.35	125.55	1	<.001 (ACE)
CE	-	<b>.81 (.78-.84)</b>	<b>.19 (.16-.22)</b>	-	<b>.81 (.78-.84)</b>	<b>.19 (.16-.22)</b>	<b>8738.75</b>	<b>2.34</b>	<b>2</b>	<b>.31 (ACE)</b>
E	-	-	1	-	-	1	9700.73	964.31	3	<.001 (ACE)
<b>Partisan Intensity</b>										
ACE	.15 (.15-.41)	.33 (.11-.50)	.52 (.47-.60)	.44 (.24-.57)	.03 (0-.20)	.53 (.42-.64)	10750.24	-	-	-
ACE (M=F)	.34 (.31-.53)	.14 (0-.27)	.52 (.52-.59)	.34 (.31-.53)	.14 (0-.27)	.52 (.52-.59)	10755.78	5.54	3	.14 (ACE)
<b>AE</b>	<b>.50 (.44-.50)</b>	-	<b>.50 (.44-.54)</b>	<b>.50 (.44-.50)</b>	-	<b>.50 (.44-.54)</b>	<b>10758.86</b>	<b>3.08</b>	<b>1</b>	<b>.08 (ACE)</b>
CE	-	.39 (0-.39)	.61 (.25-.61)	-	.39 (0-.39)	.61 (.25-.61)	10768.39	12.61	2	<.001 (ACE)
E	-	-	1	-	-	1	13809.33	3053.55	3	<.001 (ACE)

**Notes:** Best-Fitting Models in Bold. A=additive genetic, C=common environment, E=unique environment. The full model (ACE) contains all three sources of variance. Reduced models are notes as AE, CE and E. M=F references a model where males and females are equated.

**Table 6.** Cholesky Decomposition Standardized Path Coefficients for Party Identification (Males and Females Combined)

<b>Common Environment Factor</b>							
	C1	C2	C3	C4	C5	C6	C7
Church	-0.65						
School Prayer	-0.397	0.502					
Gay Rights	0.334	-0.341	0.421				
Death Penalty	0.084	0.23	-0.103	0.51			
Unions	0.083	-0.005	0.057	0.022	0.358		
Fed Housing	0.079	-0.023	0.334	-0.059	0.051	0.338	
PID	-0.107	0.146	-0.179	-0.107	-0.316	-0.05	0.747

<b>Unique Environment Factor</b>							
	E1	E2	E3	E4	E5	E6	E7
Church	0.524						
School Prayer	0.08	0.582					
Gay Rights	-0.131	-0.147	0.614				
Death Penalty	-0.029	0.102	-0.188	0.643			
Unions	-0.055	0.061	0.124	0.018	0.751		
Fed Housing	-0.06	0.02	0.177	0.002	0.07	0.785	
PID	0.001	0.013	-0.052	0.035	-0.07	-0.024	0.507

**Table 7.** Cholesky Decomposition Standardized Path Coefficients for Partisan Intensity

**FEMALES**

**Additive Genetic Factor**

	<b>A1</b>	<b>A2</b>	<b>A3</b>	<b>A4</b>	<b>A5</b>	<b>A6</b>	<b>A7</b>
Church attendance	-.668						
Neuroticism	.002	-.675					
Social Desirability	-.003	.140	.576				
Extraversion	.042	.021	-.274	.558			
Impulsivity	-.103	-.062	.080	-.059	.515		
Psychoticism	-.015	-.289	.128	-.066	.121	-.371	
Partisan Intensity	.006	.028	-.040	-.134	-.040	-.026	.516

**Unique Environment Factor**

	<b>E1</b>	<b>E2</b>	<b>E3</b>	<b>E4</b>	<b>E5</b>	<b>E6</b>	<b>E7</b>
Church attendance	.665						
Neuroticism	-.033	.720					
Social Desirability	.007	-.161	-.758				
Extraversion	.008	.004	.111	-.667			
Impulsivity	.044	.021	-.062	.056	-.734		
Psychoticism	-.012	.241	-.073	.107	-.083	-.782	
Partisan Intensity	-.008	-.015	.008	.007	.026	.034	-.832

**MALES**

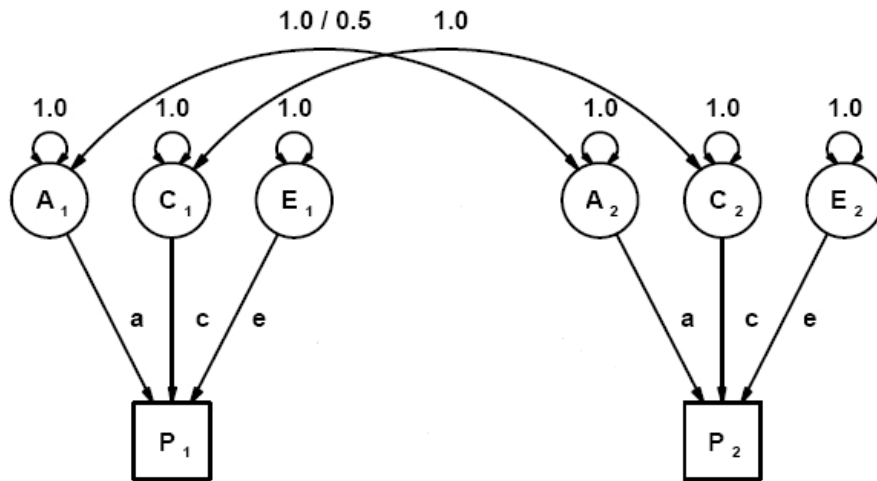
**Additive Genetic Factor**

	<b>A1</b>	<b>A2</b>	<b>A3</b>	<b>A4</b>	<b>A5</b>	<b>A6</b>	<b>A7</b>
Church attendance	-.432						
Neuroticism	-.030	-.563					
Social Desirability	-.307	.084	.456				
Extraversion	-.070	-.126	-.098	.263			
Impulsivity	.036	.006	.104	.276	.325		
Psychoticism	-.068	-.209	.131	.153	.199	-.210	
Partisan Intensity	-.020	.076	-.074	-.144	-.050	-.052	.467

**Unique Environment Factor**

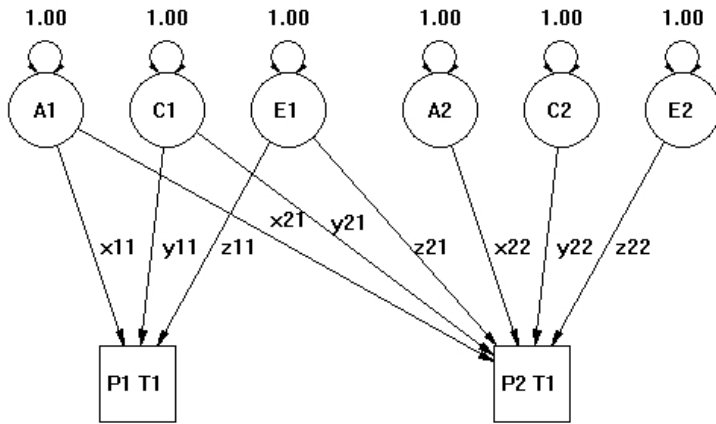
	<b>E1</b>	<b>E2</b>	<b>E3</b>	<b>E4</b>	<b>E5</b>	<b>E6</b>	<b>E7</b>
Church attendance	.763						
Neuroticism	-.023	.781					
Social Desirability	.012	-.162	.700				
Extraversion	.009	-.017	-.166	.747			
Impulsivity	.037	-.031	.073	-.126	-.898		
Psychoticism	-.006	.253	.106	-.128	-.112	-.770	
Partisan Intensity	-.019	-.027	-.013	-.045	-.003	.026	-.840

**Figure 1-** The ACE path diagram



**Notes:** P1 is the phenotype or trait value of twin 1, P2 is the phenotype or trait value of twin 2. A is the additive genetic component, C is the common environment components and E is the unique environment component. A is equated for monozygotic twins (1) and set to .5 for dizygotic twins. Common environment is equated for all twin pairs, and unique environment is not constrained.

**Figure 2-** Cholesky Decomposition



**Notes:** A is the additive genetic component, C is the common environment component and E is the unique environment component. In the Cholesky there are as many latent factors as variables. For explanatory purposes, only two variables were included in this diagram; however, the Cholesky used in the analyses was extended to seven variables. This diagram contains a model with two traits or phenotypes for twin 1 (T1). The model shows P1 and P2, where P2 only influences the second variable and P1 influences both variables allowing for a correlation between the variables due to shared genetic effects. The same rules apply to C and E. What the Cholesky offers is the correlation between traits and whether this correlation is determined by shared genetic or shared environmental effects will be indicated by the cross-twin cross-trait covariance.