Partisanship, Voting, and the Dopamine D2 Receptor Gene

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Previous studies have found that both political orientations (Alford, Funk, and Hibbing 2005) and voting behavior (Fowler, Baker, and Dawes 2008; Fowler and Dawes 2008) are significantly heritable. In this article we study genetic variation in another important political behavior: partisan attachment. Using the National Longitudinal Study of Adolescent Health, we show that individuals with the A2 allele of the D2 dopamine receptor gene are significantly more likely to identify as a partisan than those with the A1 allele. Further, we find that this gene's association with partisanship also mediates an indirect association between the A2 allele and voter turnout. These results are the first to identify a specific gene that may be partly responsible for the tendency to join political groups, and they may help to explain correlation in parent and child partisanship and the persistence of partisan behavior over time.

The early work of the Michigan School, most notably the *The American Voter*, argues that party identification is an affective attachment that is the product of socialization (Campbell et al. 1960). Party identification is generally weak or nonexistent until individuals reach their formative years, at which point their partisanship ties strengthen as a result of becoming active members of their communities and forming close associations with social groups, some of which have ties to political parties (Campbell et al. 1960). More recent scholarship, building on this social psychology view emphasizes the notion of party identification as a social attachment, arguing that the identification with a particular party is primarily based on an image of that party as a social group (Gerber and Green 1998; Green, Palmquist, and Schickler 2002).

In contrast to the social psychology theory of partisanship, instrumental theories characterize partisan attachment as an information shortcut that is continually updated and adjusted based on rational evaluation (Fiorina 1981; Popkin 1991). For example, Achen (1992) argues that voters act as Bayesian updaters, prospectively judging parties based on their observations of the party's past performance and information received from a campaign. Voters receive ''noisy'' signals about party performance, and this noise originates at the individual-level and/or system-level of the information environment. 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).

There are many examples of careful empirical studies of these theories; however, nearly all have focused exclusively on *environmental* explanations. In contrast, recent work has shown that *genetic* factors account for a significant proportion of variation in social attitudes (Martin et al. 1986) and political attitudes (Alford, Funk, and Hibbing 2005; Hatemi et al. 2007) related to the *direction* of partisanship (e.g., Republicans vs. Democrats). Two twin studies have recently shown that the *strength* of partisanship is significantly heritable (Settle, Dawes, and Fowler in press; Hatemi et al. in press). Moreover, genetic factors are also important for political behaviors like voting (Fowler, Baker, and Dawes 2008; Fowler and Dawes 2008; Fowler and Schreiber 2008) that are known to be influenced by the tendency to *attach* to a given party (e.g., partisans vs. nonpartisans).

While no studies to date have considered a link between specific genes and partisanship, previous association studies have identified genes that are important in shaping personality traits and behaviors

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integral to instrumental and social psychology theories of partisanship. The social psychology theory of partisanship suggests variation in partisanship can be explained in part by variation in social attachments, whereas instrumental theories suggest that differences in information processing, as well as the level of individual-level noise, are important determinants. Although there are likely to be dozens of genes involved in complex political behavior, here we identify one, the DRD2 gene, that is believed to play an important role in *both* the development of social attachments and cognitive functions that may be critical to the formation of partisan ties.

Based on the political science and behavior genetics literature, we hypothesize that the DRD2 gene influences whether or not a person will identify with a political party. Using both case-control and family-based gene association tests, we find that the A2 allele of the DRD2 dopamine receptor gene is significantly associated with partisanship. Specifically, individuals who have two A2 alleles of the DRD2 gene are 8% more likely to become partisans than those who have no A2 alleles. Furthermore, this increase in the likelihood of partisan attachment also mediates a significant positive association between the A2 allele and voter turnout.

These results suggest that inherent biological variation from person to person helps to explain variation in political behavior. In particular, since gene variants like DRD2 are inherited from parents, they may help to explain the well-known correlation in strength of partisanship between parent and child (Campbell, et al. 1960; Niemi and Jennings 1991). They may also help to explain why partisan attachments are long-lasting and stable over time (Converve 1969; Miller and Shanks 1996).

Genetic Concepts

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

At conception individuals inherit two copies of each gene, with one copy coming from the mother and one copy from the father. Some genes come in different versions, known as *alleles*—for example, sickle cell disease results from a particular allele coding for abnormal rather than normal hemoglobin. A gene is said to be a *polymorphism* when there is more than one type of allele that exists in the population. Each parent has two separate copies of an allele at each *locus*, or location, on the chromosome, but each sperm or egg cell contains only one of these alleles. Thus a child has a 50% chance of receiving a particular allele from each parent. For example, suppose that at a given locus there are two possible alleles, A1 and A2. If both parents are *heterozygous* at that locus, meaning they each have an A1 and an A2 allele (A1A2), then a given offspring has a 25% chance of being *homozygous* for A2 (A2A2), a 25% chance of being homozygous for A1 (A1A1) and a 50% chance of being heterozygous (A1A2 or A2A1—order is irrelevant). An individual's *genotype* at a particular locus is the combination of alleles that they have at a particular locus; A2A2, A2A1, and A1A1 are the possible genotypes in our example.

Many of the observable traits and behaviors of interest, referred to as *phenotypes*, are far downstream from the original genotype present in the DNA. While in some cases one allele can single-handedly lead to a disease (such as Sickle Cell Anemia, Huntingtons disease, and cystic fibrosis), the vast majority of phenotypes are *polygenic*, meaning they are influenced by multiple genes (Mackay 2001; Plomin 2008). Moreover, phenotypes are typically shaped by a multitude of environmental forces. Even the brain can be shaped in both its structure and function by internal environments (e.g., the presence of certain hormones) and external environmental conditions.

Most genes specify the composition of proteins (Hartl 2000). This process is known as *gene expression*. The first step in the process is *transcription* where strands of DNA that make up a gene are copied into the code of RNA. Not all of the information transcribed is used to code for proteins. *Exons* are sequences of DNA used to code for proteins, whereas *introns*, which lie between exons, do not contain information used for protein synthesis. In the second step of gene expression, *RNA processing*, introns are removed from the RNA code and exons are spliced together. The end result of this two-step process is messenger RNA. The messenger RNA carries the copied instructions to the ribosome where RNA undergoes *translation*. Translation entails combining the instructions provided by messenger

RNA with raw materials to make proteins. Subunits of messenger RNA, called *codons*, provide specific instructions for the construction of protein chains.[1]

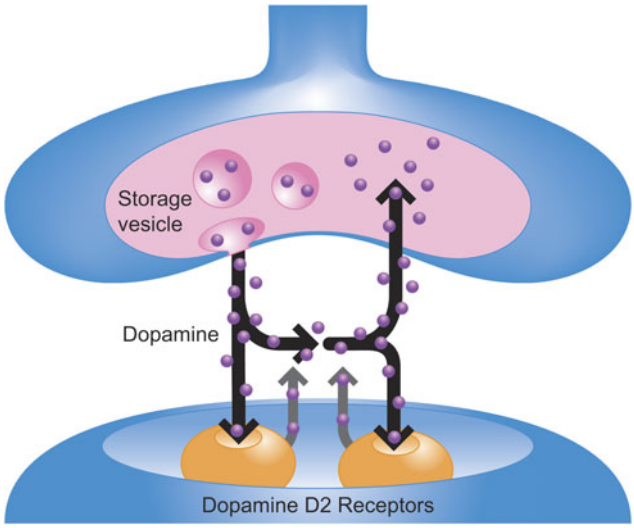
Dopamine and the D2 Receptor Gene

Neurons are nerve cells in the brain that are responsible for sending, receiving, and processing information. In order for this information to be sent from one neuron to another, signals must cross a small gap called a synapse that exists between the axon of a sending neuron and dendrite of the target neuron. Neurotransmitters, released by the axon of the sending neuron cross the synaptic gap and bind with "receptors" on the dendrite of the postsynaptic (receiving) neuron, triggering changes in the postsynaptic neuron's metabolic activity. The neurotransmitter is then released and reabsorbed into the presynaptic neuron. Signals are carried throughout the brain by the sequential firing of one neuron after another across these synapses. Figure 1 illustrates the neurotransmitter cycle.

The brain is made up of many different types of neurons that rely on different neurotransmitters, each with different functions. Dopamine, a member of the catecholamine family, is one such neurotransmitter. The dopamine system is responsible for the control of locomotion, cognition, emotion, positive reinforcement, appetite, and endocrine regulation (Missale et al. 1998) and also plays a strong role in human attachment (Fisher et al. 2002). Dopamine influences these physiological processes by activating at least five different dopamine receptors (D_1, D_2, D_3, D_4, and D_5) located throughout the brain, including the striatum, amygdala, caudatus, and putamen (Missale et al. 1998).[2] There is strong evidence that impairments of the dopamine system are implicated in neurological, psychiatric and drug addition disorders, and mental illness (Hurd and Hall 2005).

The D2 receptor has been the subject of intense scrutiny because of its role in modulating dopamine

FIGURE 1 Simple representation of the release, reception, and recycling of dopamine in neuron.



synthesis, cell firing, and release (Hurd and Hall 2005). Differences in the number and function of D2 receptors have been linked to the gene that codes for it, the dopamine D2 receptor (DRD2) gene. Individuals with the A1 allele of the DRD2 gene exhibit a greater than 30% reduction in the density of D2 receptors, leading to weaker dopamine signaling in the brain (Jonsson et al. 1999; Noble 2003; Pohjalainen et al. 1998), and central nervous system (Berman and Noble 1995), as well as reduced glucose metabolism in the brain (Noble et al. 1997).

Several studies have found a significant relationship between the dopamine D2 receptor density and social attachment (Breier et al. 1998; Farde, Gustavsson, and Jonsson; 1997; Jonsson et al. 1999) as well as an association between the A1 allele and social alienation (Hill et al. 1999), antisocial personality disorder (Ponce et al. 2003), and avoidant personality types (Blum, Sheridan, Chen, Wood, Braverman and Cull 1997). Research on nonhuman animals also supports a connection between the D2 receptor and the formation of social attachments (Curtis et al. 2006). For example, Gingrich et al. (2000) found the D2 receptor mediated social attachments in prairie voles, and Shively et al. (1997) found decreased D2 receptor binding in socially isolated cynomolgus monkeys.

The D2 receptor has also been linked to differences in cognitive function. Neuroimaging studies have found D2 receptor binding to be correlated with

[1]DNA is made up of subunits called nucleotides. There are four such nucleotides: adenine (A), cytosine (C), thymine (T), and guanine (G), named based on the nitrogenous base that they contain. The base uracil (U) replaces thymine in RNA. Codons are groupings of three adjacent nucleotides. These groupings correspond to specific amino acids are subunits of the protein chain.

[2]These receptors are classified into two families, D1-like receptors (D_1 and D_5) and D2-like receptors (D_2, D_3, and D_4).

attention, working memory, planning, and visual processing (Backman et al. 2000; Cropley et al. 2006; Reeves et al. 2005; Takahashi et al. 2007; Volkow et al. 1998). Drugs stimulating D2 receptors have been shown to improve cognitive function (Berthier 2005; Kimberg, D'Esposito and Farah 1997; McDowell, Whyte, and D'Esposito 1998), while antagonist drugs impair them (Mehta et al. 1999). However, results based on the DRD2 gene are mixed. An early study linked the A1 allele to reduced cognitive ability (Berman and Noble 1995) but subsequent work has failed to corroborate these findings (Moises 2001, Petrill 1997). Studies of nonhuman subjects have shown activation of D2 receptors improves acquisition and retention of working memory tasks (Missale et al. 1998). For example, Glickstein, Hof, and Schmauss (2002) found mice lacking D2 receptors exhibited impaired working memory.

Based on this large literature linking the dopamine D2 receptor and DRD2 gene to the formation of social attachments and cognitive functions, two prominent variables in theories of partisanship, we theorize that individuals with the A2 allele of the DRD2 gene are significantly more likely to identify themselves as a partisan. However, a note of caution is necessary at the outset. Finding a clear link between particular genes and behaviors has been notoriously difficult and any true causal story is likely to be complex. Although the literature we cite here illustrates plausible causal mechanisms, it does not offer a clear roadmap for determining specifically how DRD2 may influence partisanship. If we find an association, it is only the first step.

Add Health

The National Longitudinal Study of Adolescent Health (Add Health) is a study that explores the causes of health-related behavior of adolescents in grades 7 through 12 and their outcomes in young adulthood.[3] The first wave of the Add Health study was collected in 1994–95 when subjects were between 11 and 19 years old, the second wave in 1996, and the third wave in 2001–02 when subjects were between 18 and 26 years old. The third wave was made up of 15,170 of the original Wave I participants. The Add Health study is a nationally representative study. Women make up 49% of the study's participants, Hispanics 12.2%, Blacks 16.0%, Asians 3.3%, and Native Americans 2.2%.[4] Participants in the Add Health study also represent all regions of the country: the Northeast makes up 17% of the sample, the South 27%, the Midwest 19%, and the West 17%.

In Wave I of the Add Health study, researchers created a genetically informative sample of sibling pairs based on a screening of the in-school sample of 90,114 adolescents. These pairs include all adolescents that were identified as twin pairs, siblings, half siblings, or unrelated siblings raised together. The Wave I sibling-pairs sample has been found to be similar in demographic composition to the full Add Health sample (Jacobson and Rowe 1998). Genetic markers are available for 2,574 individuals,[5] including markers that identify alleles of the DRD2 gene.

This study focuses on the Taq1 A polymorphism of the DRD2 gene. The DRD2 gene is located on chromosome 11 and the Taq1 A polymorphism is located just beyond the DRD2 gene.[6] This polymorphism has recently been discovered to be residing in the neighboring ANKK1 gene (Dubertret 2004; Neville, Johnstone, and Walton 2004). Therefore, it remains unclear how exactly Taq1 A affects DRD2 expression since it is not located in a protein-encoding region of the DRD2 gene. However, the Taq1 A is believed to be in linkage disequilibrium[7] with a polymorphism (or polymorphisms) residing in the DRD2 gene (Fossella, Green, and Fan 2006). This means that while the Taq1 A allele may not play a role in DRD2 expression, it is likely highly correlated with a polymorphism that does. Therefore, Taq1 A serves as a proxy for that yet undetermined polymorphism. Better understanding the Taq1 A

[3]The Add Health study has been extensively described elsewhere. Details about the study can be found at www.cpc.unc.edu/addhealth.

[4]A breakdown for those providing DNA samples is presented in the appendix, along with a variety of summary statistics.

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

[6]Specifically, it is approximately 10.5 kb, or 10,500 nucleotides, beyond exon 8 of the DRD2 gene. Exon 8 contains the termination codon, so Taq1 A is not in a protein-encoding region of DRD2. Complete details of the genotyping protocol can be found at www.cpc.unc.edu/addhealth/files/biomark.pdf.

[7]A detailed explanation of linkage disequilibrium is beyond the scope of this paper. Interested readers can find such a discussion in introductory texts on population genetics, such as Hartl (2000).

allele's precise role is an area of intense ongoing research.

There are two DRD2 alleles, A1 and A2. In our Add Health sample, 54% of the subjects have two A2 alleles (no A1 alleles), 37% have one A2 allele and one A1 allele, and 8% have no A2 alleles (two A1 alleles). The dependent variables in our study are subject responses to the questions "Do you identify with a specific political party? [Yes or No]" and "Did you vote in the most recent (2000) presidential election? [Yes or No]," both of which were collected as part of the study's Wave III.

Genetic Association

A genetic association study indicates whether an allele is found more frequently than can be attributed to chance in a group exhibiting a particular trait than those without the trait. In our case, is the frequency of a particular allele higher among partisans than nonpartisans? However, a significant association can mean one of three things: (1) The allele itself influences partisanship; (2) the allele is in linkage disequilibrium with an allele at another locus that influences partisanship; or (3) the observed association is a false positive signal due to population stratification.[8]

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

There are two main research designs employed in association studies: case-control designs and family-based designs. Case-control designs compare the frequency of alleles or genotypes among subjects that exhibit a trait of interest to subjects who do not.[9] As a result, they are vulnerable to population stratification. A typical way to control for this problem is to include controls for the race/ethnicity of the subject or to limit the analysis to a specific racial or ethnic group. Family-based designs eliminate the problem of population stratification by using family members, such as parents or siblings, as controls. Tests using family data compare whether offspring exhibiting the trait receive a risk allele from their parents more often than would be expected by chance.[10] A major limitation of family-based studies is that they tend to be underpowered, thus prone to Type I error (Xu and Shete 2006). In this study we employ both case-control and family-based designs.

Case-Control Design

The first approach we use to test for genetic association is a mixed-effects logistic regression model (Guo and Zhao 2000; Xu and Shete 2006):

$$logit(P[Y_{ij} = 1|Z_{ij},\ U_j]) = \beta_0 + \beta_X X_{ij} + \beta_Z Z_{ij} + U_j$$

where i and j index subject and family respectively, X is the number of A2 alleles (0,1,or 2), Z is a matrix of variables to control for underlying population structure of the Add Health samples as well as other variables that may influence party attachment, U is a family random effect that takes into account the fact the observations are not independent because siblings come from the same family. The random effect controls for genetic and environmental correlation among family members.

To control the effects of the underlying population structure, we use indicator variables for whether a subject self-reported as black, Hispanic, Asian, or Native American (omitted category is white). Following the policy of the United States Census, Add Health allows respondents to mark more than one

[8]Given our data, we cannot differentiate between 1 and 2. In order to do so we would need additional genetic information about loci in close proximity to the locus of interest. In future work, we intend to utilize additional genetic data that will become available in Add Health wave (IV) to perform this test. The important point for us here is that a true signal of association means that either a particular allele, or one likely near it on the same gene, significantly influences partisanship.

[9]Controls may be randomly selected from the population or from groups known not to exhibit the trait.

[10]If there were no association between the trait and the risk allele, offspring would get the same number of alleles from their parents as predicted by chance alone.

race. In our sample, 108 subjects chose two races, nine subjects chose three races, and 37 subjects chose no race. For those 117 subjects choosing more than one race, we assign a single race category based on their response to the Add Health follow-up question of "which one category best describes your racial background?"[11]

Table 1 presents the results for the test of association between the A2 allele of the DRD2 gene and partisanship. The first model (baseline) only includes controls for age, gender, and race. The second model includes income, marital status, and homeownership since these measures of socioeconomic status may influence whether one identifies with a party. The odds ratio of the A2 allele parameter estimate is an individual's odds of being a partisan if he or she has one A2 allele compared to having no A2 alleles (or having two A2 alleles compared to one A2 allele). A significant odds ratio implies that the dopamine D2 receptor gene is associated with partisanship. The baseline model shows that the A2 allele is significant ($p = 0.03$) and the odds of an individual with one A2 allele being a partisan are 1.2 times greater than for someone with no A2 alleles and the odds of an individual with two A2 alleles being partisan are 1.4 times greater than for someone with no A2 alleles. The A2 allele remains significant ($p = 0.04$) in the model with SES measures and the odds ratios are nearly identical. Figure 2 presents the simulated first differences for the baseline model. Holding the control variables at their means and changing the number of A2 alleles from zero to one increases average partisanship by about 4 percentage points and from zero to two by about 8 percentage points. Both simulated first differences are significantly different from zero.

It is important to note that DRD2 is associated with the likelihood a person will identify as a partisan, but it does not say anything about *which* party a person will identify. In the appendix (Appendix 5) we present results that show Democrats and Republicans do not have significantly different distributions of the A2 allele, suggesting we will need to look elsewhere for genes that may be associated with political orientations. One such possiblity has been suggested by Settle et al. (2008).

[11]We also employ two additional approaches. First, we create a separate group for those identifying themselves as multirace and included an indicator variable in the regression for that group along with the other categories. Second, we omit the 117 multirace individuals. The results based on these two approaches, presented in the appendix (Appendix 5), are nearly identical. Individuals who do not choose a race might also prefer not to choose a political party, therefore we included a dichotomous variable for no race in first first model in Appendix 5. The results are identical to those reported so we did not include them. Finally, when we restrict the analysis to only single-race white respondents (67% of the sample), the association remains significant ($p = 0.02$).

Family-Based Design Results

In order to ensure population stratification is not driving our results, we also use a family-based test of the association between the A2 allele of DRD2 and partisanship. Spielman and Ewens (1998) constructed a sibling-based test of association, known as the sib TDT, for binary traits.[12] In this test, sibling members of a nuclear family are compared with one another to determine whether the allele frequency among those "affected" siblings is significantly different from "unaffected siblings" (partisans and nonpartisans in our case). For the sib TDT to be a valid test of association, sibships must be made up of exactly two siblings, one affected and one unaffected, with different genotypes.[13] If there is no association, which is the null hypothesis, then each genotype is equally likely for affected and unaffected sibs in these sibships (Spielman and Ewens 1998). Based on this criteria, we have 91 sibships (182 individuals) in our sample.

The sib TDT supports the finding of an association between the A2 allele and partisanship ($\chi^2 = 4.55$, $p = 0.03$) that was found using the case-control approach.[14] However, the simple sib TDT does not allow the inclusion of potentially relevant covariates like age and gender. An alternative approach is to model the sib TDT using a retrospective logistic regression model (Waldman, Robinson, and Rowe 1999; Zou 2006). Table 2 shows that the logistic sib TDT also yields a significant association with the inclusion of age and gender as covariates ($p = 0.02$).

Both specifications of the sib TDT test support an association between the A2 allele and partisanship. Although the family-based design reduces our sample size, it supports the results from the case-control design, giving us greater confidence that population stratification is not driving the relationship between DRD2 and partisan attachment.

[12]This test is a variant of the *McNemar Test* (McNemar 1947).

[13]Specific to our study, this means both siblings must have a different number of A2 alleles.

[14]The z-score test constructed by Spielman & Ewens (1998) is known to be overly conservative, therefore we use the score test proposed by Zou (2006).

TABLE 1 Case-control test of association between DRD2 and partisan attachment.

	Base Model				*w/ SES Controls*			
	Coef	**OR**	**SE**	**P value**	**Coef**	**OR**	**SE**	**P value**
Intercept	−2.63	0.07	0.78	0.00	−2.74	0.06	0.80	0.00
A2	0.21	1.23	0.10	0.03	0.20	1.23	0.10	0.04
Black	0.76	2.14	0.16	0.00	0.78	2.18	0.17	0.00
Asian	−1.11	0.33	0.27	0.00	−1.06	0.35	0.27	0.00
Native American	0.13	1.13	0.40	0.75	0.09	1.09	0.41	0.83
Hispanic	−0.62	0.54	0.20	0.00	−0.65	0.52	0.20	0.00
Age	0.07	1.08	0.03	0.03	0.08	1.09	0.04	0.02
Male	−0.23	0.80	0.11	0.05	−0.21	0.81	0.12	0.07
Income					−0.02	0.98	0.02	0.36
Homeowner					−0.03	0.97	0.19	0.88
Married					0.12	1.12	0.16	0.48
N	2534				2493			
LL	−1576				−1551			
LL(*constant*)	−1615				−1590			

Notes: The model is a mixed-effects logit which estimates a random intercept for each family (not shown). The dichotomous dependent variable is Partisan, which is the answer to the question "Do you identify with a specific political party? [1 = yes, 0 = no]." Logit coefficients (Coef), odds ratios (OR), standard errors (SE), and p-values are presented. Detailed variable descriptions are provided in the appendix.

Voting

Given that the dopamine D2 receptor gene predicts partisan attachment, a natural question to ask is whether it is also associated with voter turnout since we know that partisans are more likely to vote (Bartels 2000). To explore this question we follow the steps of testing for mediation laid out by Baron and Kenny (1986). A variable *M* mediates the relationship between an independent variable *X*, in our case a genotype, and a dependent variable *Y*, in our case voter turnout, if (1) *X* significantly predicts *Y*, (2) *X* significantly predicts *M*, and (3) *M* significantly predicts *Y* controlling for *X* (Baron and Kenny 1986). This relationship is illustrated in Figure 3.

A formal test of mediation, known as the *Sobel Test*, determines whether the indirect effect, the product of the coefficient on the number of A2 alleles in (2) and the coefficient on partisanship in (3), is significantly different from zero (Baron and Kenny 1986; Sobel 1982). An assumption of the test is that the indirect effect is distributed normally; however, this assumption has been shown to be problematic (Bollen and Stine 1990; Lockwood and MacKinnon 1998; Mackinnon et al. 2002; Shrout and Bolger 2002). A superior approach, which we take, is to bootstrap the estimated indirect effect and construct confidence intervals based on the bootstrapped values (Bollen and Stine 1990; Shrout and Bolger 2002).[15]

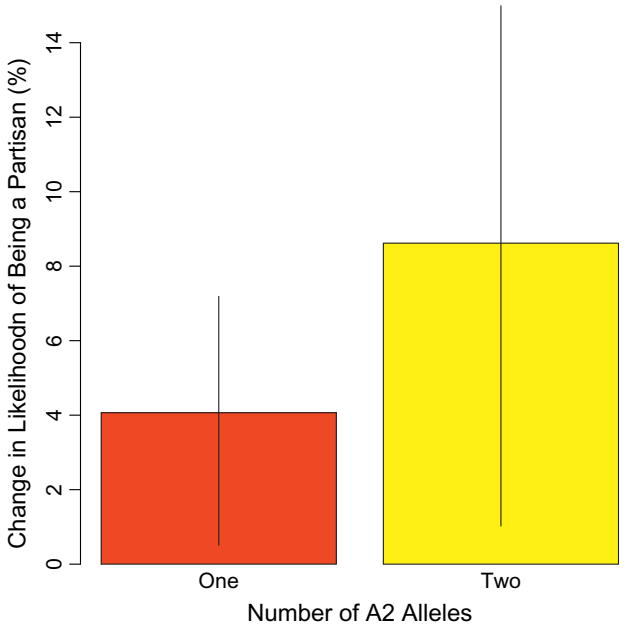
Figure 4 presents a histogram of the bootstrapped values for the indirect and direct effects of the dopamine D2 receptor gene on voting. The mean of the bootstrap distribution for the indirect effect is 0.09 (0.01, 0.18). Meanwhile, the mean of the direct effect is zero (−0.12, 0.12). Therefore, the evidence suggests that the relationship between the DRD2 gene and voter turnout is more likely to be indirect, mediated by DRD2's effect on partisanship.

Discussion

We find that an extensively studied gene that regulates the dopamine system is associated with the tendency to identify with a political party. We arrived at this result using two different design approaches, one of which guards against a false positive due to underlying population structure instead of a true association.

[15]When the dependent variable, independent variable, and mediator are all continuous, the product of the coefficients can be taken directly from the regression estimates. However, when they are all dichotomous, as they are here, this is no longer true because the scale differs when a variable is a predictor versus an outcome variable due to the fact that the error variances are fixed. Therefore, all of the coefficients must be re-scaled so that they are comparable across equations (Mackinnon and Dwyer 1993).

FIGURE 2 Changing the number of A2 alleles yields significantly higher partisanship.



Notes: First differences, based on simulations of *Table 1* (baseline) parameters, are presented along with 95% confidence intervals. All other variables in the model are held at their means.

TABLE 2 Family-based test of association between DRD2 and partisan attachment.

	Retrospective Logit sib TDT			
	Coef	**OR**	**SE**	**P value**
Intercept	1.39	4.03	1.37	0.31
Partisan	0.50	1.65	0.22	0.02
Age	−0.05	0.95	0.06	0.39
Male	0.11	1.11	0.22	0.64
N		91		
LL		−239		
LL(*constant*)		−242		

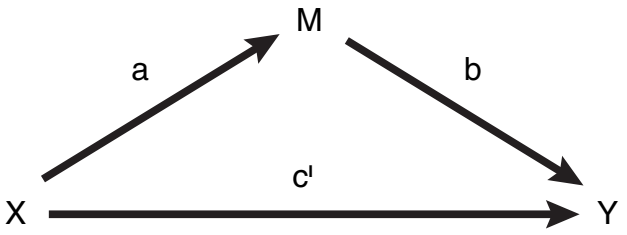
Notes: The model is a mixed-effects logit which estimates a random intercept for each family (not shown). The dichotomous dependent variable is whether or not the *k*th allele ($k = 1, 2$) is an A2 allele [A2 allele = 1, A1 allele = 0]. Logit coefficients (Coef), odds ratios (OR), standard errors (SE), and p-values are presented. Detailed variable descriptions are provided in the appendix.

It must be emphasized that we have only found an association and cannot make any causal claims about the relationship between the DRD2 gene and either partisanship or turnout. However, the empirical link between the D2 dopamine receptor and DRD2 gene, as well as the known functions of dopamine in the brain, suggest at least two channels through which the A2 allele may influence partisanship. We hypothesize that improved ability to form social attachments and/or improved cognitive function, both of which have been shown to be associated with the A2 allele, increases the likelihood an individual with the A2 allele will form and/or maintain an attachment with a political party.

One might wonder why we go all the way down to the genetic code to study partisan attachment. The fact that genes are fixed is helpful in better understanding the constraints some individuals face in developing political behavior. Specifically, genes may help us to explain two well-known features of partisanship. First, parental party identification has been shown to be one of the strongest predictors of partisanship in young adults (Campbell et al. 1960; Niemi and Jennings 1991). Our results do not contradict research suggesting that identification with a specific party is the product of socialization; however, it is possible that the tendency to attach to a party in general is due in part to the inheritance of a particular allele of a gene like DRD2. This could help to explain why parents who attach to a party have children who also attach. Second, partisan attachments are long-lasting and stable (Converse 1969; Miller and Shanks 1996). This has been interpreted as the product of reinforced behavior or loyalty (Brader and Tucker 2001), however it may also be due to genes like DRD2 since they are fixed. Future longitudinal and family studies of partisanship should investigate what role DRD2 plays in the transmission of political attitudes and behavior over time within individuals and between parents and children in order to establish the relevant roles for socialization and heritability. In particular, given that Settle, Dawes, and Fowler (in press) and Hatemi et al. (in press) show that the strength of attachment is significantly heritable while specific attachments (Republican/Democrat) are not, the full spectrum of partisan attachments may well be best represented by an interaction between genes and parental socialization.

A relatively recent extension of this literature argues that emotion also plays an important role in determining when individuals use partisanship as an information shortcut (Brader 2005; Marcus and Mackuen 1993; Marcus, Neuman, and Mackuen 2000). This research agenda is based on the finding that an individual's affective state alters the way he or she processes information; those in a positive mood are more likely to rely on heuristics whereas those in a negative mood eschew these heuristics and pay closer attention to the details (Schwarz 2000). This suggests that since anxiety is negatively related to the use of heuristics, individuals less equipped to deal

FIGURE 3 The mediation relationship described by Baron and Kenny (1986).



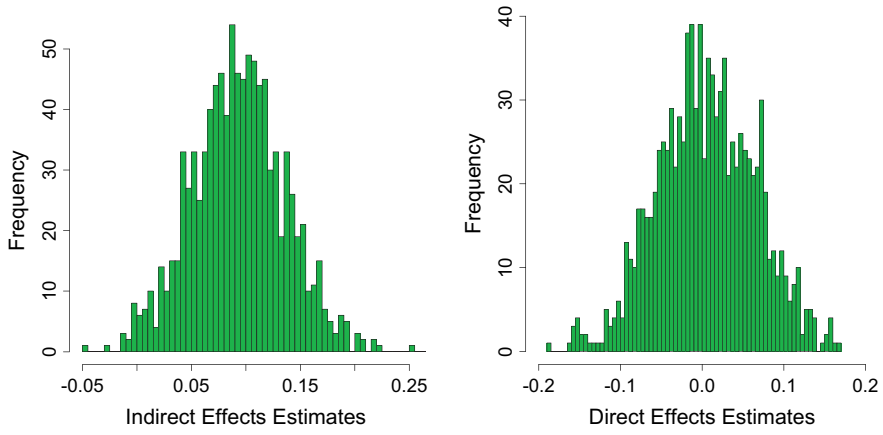
Notes: Path c' is called the direct effect and the product of paths a and b is the indirect effect. To estimate the direct and indirect effects run regressions (2) and (3): (2) $logit(P[Mediator_{ij} = 1|Z_{ij}, U_j]) = \beta_0 + aA2_{ij} + \beta_Z Z_{ij} + U_j$ and (3) $logit(P[Partisanship_{ij} = 1|Z_{ij}, U_j]) = \beta_0 + c'A2_{ij} + bMediator_{ij} + \beta_Z Z_{ij} + U_j$ where Z is the matrix of control variables and U is the family random effect.

with stress and anxiety in general are less likely to develop or consistently rely on them. We conjecture that this is a possible explanation for the positive association between the A2 allele and partisanship. The dopamine D2 receptors have been implicated in social anxiety and stress due to the fact that dopamine's stimulation of D2 receptors in the brain results in a feeling of well-being and the reduction of stress and negative feelings (Bowirrat and Oscar-Berman 2005; Kreek and Koob 1998; Pani, Porcella and Gessa 2000). This quality has resulted in dopamine being dubbed the "antistress molecule" (Blum et al. 2000). The A1 allele of the DRD2 gene has been associated with sensitivity to stress and anxiety (Bau, Almeida, and Hutz 2000; Jonsson et al. 2003) and personality traits related to stress and anxiety (Hill et al. 1999).

The association we find between the DRD2 gene and partisanship also suggests another connection between partisanship and the punishment of free-riders observed by Smirnov et al. (2007). Their study found that in an experimental setting partisans were more likely than nonpartisans to cooperate with anonymous individuals in a public goods game, and they were also more willing to spend their own money to punish free-riders. Reputations could not be formed in these experiments, meaning neither cooperation nor punishment was consistent with rational self-interested behavior. The authors were not able to identify a causal link between partisanship and punishment, but our results suggest that part of the variation in punishment behavior they observed may be attributed to genetic factors. This is because the A2 allele of the DRD2 gene is associated with a significant increase in D2 dopamine receptor density in the striatum (Jonsson et al. 1999; Pohjalainen et al. 1998), an area of the brain that has been implicated in derived satisfaction from punishing free-riders (de Quervain et al. 2004). It is possible that the reason partisans are able to overcome collective action problems through the punishment of defectors is because they are more likely to feel pleasure or relief from doing so, which is in part a function of their genes. This would be consistent with other studies that show cooperative behavior is significantly heritable (Cesarini et al. 2008).

Our results also add to the recent finding that voting behavior is significantly heritable (Fowler, Baker and Dawes 2008) and that specific genes responsible for regulating serotonin are associated with turnout (Fowler and Dawes 2008). The fact that dopamine and serotonin are both implicated in turnout is not surprising given that both the dopaminergic and serotonergic neurotransmitter systems are believed to play a vital role in the regulation of

FIGURE 4 Bootstrap distributions of the direct and indirect effect estimates of the A2 allele on voting.



emotion and mood (Bowirrat and Oscar-Berman 2005). In addition, MAOA, one of the genes the authors found to be associated with voting is responsible for degradation of both serotonin and dopamine neurotransmitters. While the relationship between these genes and voting remains unclear, this additional piece of evidence strengthens the case that turnout is significantly heritable and that these genes in particular merit further investigation.

Genopolitics, the study of the role of genes in political attitudes and behavior, is a newborn field of inquiry in political science. It is therefore especially important to highlight the limitations of studies like ours. First, the age range of our sample is between 18 and 26 years old and many of our subjects may not have had an opportunity to develop strong partisan attachments (Alwin, Cohen, and Newcomb 1991; Jennings and Markus 1984; Jennings and Niemi 1981). Therefore, our results may only apply to the initial adoption of party attachments in general. Future studies should attempt to replicate our work on an older sample. Second, our measure of turnout is self-reported, which is susceptible to overreporting (Karp and Brockington 2005). However, Fowler, Baker, and Dawes (2008) show that a substantial genetic component exists for both validated and self-reported turnout, and they do not find a statistically meaningful difference in the size of the component for the different measures. Third, genetic analyses are vulnerable to producing a spurious association due to population stratification. Our main results are strengthened by the fact that they were replicated by a family-based test; however, all of our findings remain only suggestive until they can be replicated elsewhere. Finally, we must strongly restate that all we have found thus far is a correlation between partisanship, voting, and the Taq1 A polymorphism. While the correlation is consistent with our hypothesis that genetic differences in the dopamine neurotransmitter system cause differences in political behavior, it remains an open question exactly how we get from genes to partisanship and voting.

The American Voter argued that younger adults do not have strong party attachments, since their interests in politics are limited and they are not politically active in general (Campbell et al. 1960). However, once they move beyond their ''egocentric years,'' political issues become more salient due to social attachments, resulting in the formation or strengthening of partisan ties. This narrative suggests that individuals who struggle in forming social attachments or who generally feel uncomfortable in social settings are less likely to form partisan ties. Therefore, there should be a positive relationship between prosocial behavior and the likelihood of being a partisan (Gerber and Green 1998; Green, Palmquist, and Schickler 2002). Since the DRD2 gene is known to affect these behaviors, they may be the intermediate link between the gene and partisanship.

Another potentially important determinant of partisanship, originally suggested by Downs (1957), is the cost associated with gathering information about issues. Downs theorized that rational voters with information constraints use parties as a cost-saving information shortcut. Following this line of reasoning, Shively (1979) argued that there is an inverse relationship between the ability to pay costs associated with being informed on issues and the development of party identification. The ability to pay is equated with possessing the cognitive resources necessary for learning, managing, and recalling relevant political information. Those with the fewest cognitive resources view partisanship as a lower-cost alternative and therefore are the most likely to identify as a partisan.

However, this argument directly contradicts a later argument by Huber, Kernell, and Leoni (2005) who suggest that possessing fewer cognitive resources makes it more difficult to differentiate between parties and thus choose one of them to use as a shortcut. Therefore, they theorize we should observe a *positive* relationship between cognitive ability and identification with a party. Recent empirical studies also support this argument; partisans tend to be well informed and do not appear to have lower levels of cognitive ability (Green, Palmquist and Shickler 2002; Miller and Shanks 1996), and cross country studies have found a positive relationship between cognitive ability and the likelihood of being a partisan (Huber, Kernell, and Leoni 2005; Norris 2004).

The data available currently does not permit a test of mediation that would indicate whether DRD2 affects partisanship via its effect on cognitive abilities or its effect on social behavior. However, the fact that cognitive ability leads to two distinct and mutually exclusive predictions renders it, in our view, the less plausible alternative. Moreover, while cognitive ability may speak to the question of *how* people make decisions (e.g., via bounded rationality), social attachment seems to speak more directly to the question of *why* (motivation). Thus, better understanding the role of DRD2 and other genes potentially offers insight to a variety of behaviors central to political science like voting, coalition formation, minority representation, deliberation, race relations, nationalism, participation, social movements, and a host of other topics that deal with the question how

individuals relate to other individuals and/or groups. We therefore urge scholars to design tests that will help us to understand better the intermediate steps in the process by which human biology constrains and shapes partisan behavior.

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Appendix

Variable Definitions

Partisan is the answer to the question "Do you identify with a specific political party? [1 = yes, 0 = no]" *Democrat* and *Republican* are based on the answer to the question "With which party do you identify? [1 = Democrat, 2 = Republican]" *A2* is the number (0,1, or 2) of 178bp length repeat polymorphisms of the DRD2 gene. *Black*, *Hispanic*, *Asian*, and *Native American* are indicator variables based on the questions "Are you of Hispanic or Latino origin?" and "What is your race? [white, black or African American, American Indian or Native American, Asian or Pacific Islander]" For those reporting more than one race, a single race is selected based on the answer to the question "Which one category best describes your racial background? [white, black or African American, American Indian or Native American, Asian or Pacific Islander]" *Age* is self-reported age and *Male* is an indicator taking the value of 1 if the respondent is a male and 0 for a female. *Income* is based on the response to the question "Including all the income sources...what was your total personal income before taxes in [2000/2001]?" Those who failed to respond were asked the follow-up question "What is your best guess of your total personal income before taxes? [less than $10,000, $10,000 to $14,999, $15,000 to $19,999, $20,000 to $29,999, $30,000 to $39,999, $40,000 to $49,999, $50,000 to $59,999, $60,000 to $74,999, $75,000 or more]. We recoded Income into those ranges and combined it with the response to the follow-up question. *Homeowner* is the response to the question "Do you own a residence such as a house, condominium, or mobile home [1 = yes, 0 = no]", and *Married* is and indicator taking the value of 1 if the subject reported being married.

Summary Statistics

Appendix 1 Sample means, standard errors, and 95% confidence intervals.

	Mean	Std. Err.	L 95% CI	U 95% CI
Vote	0.44	0.01	0.42	0.46
White	0.70	0.01	0.68	0.71
Black	0.19	0.01	0.17	0.20
Native American	0.03	0.00	0.02	0.03
Hispanic	0.15	0.01	0.13	0.16
Male	0.48	0.01	0.46	0.50
Partisan	0.36	0.01	0.34	0.38
Homeowner	0.11	0.01	0.10	0.13
Married	0.18	0.01	0.16	0.19

Notes: Those subjects identifying themselves as more than one race are assigned a single race category based on the follow-up question "which one category best describes your racial background?"

Appendix 2 Sample means and standard deviations.

	Mean	Std Dev
A2 alleles	1.47	0.64
Age	21.9	1.7
Income	3.25	0.05

Appendix 3 Sample means, standard errors, and 95% confidence intervals by racial and ethnic group.

		Mean	Std. Err.	L 95% CI	U 95% CI
White					
	Vote	0.44	0.01	0.41	0.46
	Partisan	0.35	0.01	0.33	0.37
	A2 alleles	1.55	0.01	1.52	1.58
Black					
	Vote	0.52	0.02	0.47	0.56
	Partisan	0.48	0.02	0.44	0.53
	A2 alleles	1.33	0.03	1.27	1.39
Hispanic					
	Vote	0.34	0.02	0.29	0.39
	Partisan	0.26	0.02	0.21	0.30
	A2 alleles	1.22	0.04	1.14	1.29
Asian					
	Vote	0.33	0.03	0.26	0.40
	Partisan	0.19	0.03	0.13	0.24
	A2 alleles	1.17	0.05	1.07	1.26
Native American					
	Vote	0.28	0.05	0.17	0.39
	Partisan	0.29	0.06	0.18	0.40
	A2 alleles	1.31	0.07	1.17	1.46

Notes: Those subjects identifying themselves as more than one race are assigned a single race category based on the follow-up question "which one category best describes your racial background?"

Appendix 4 Alternate Race Specifications.

	Multi-race Group				*Multi-race Excluded*			
	Coef	OR	SE	P value	Coef	OR	SE	P value
Intercept	−2.67	0.07	0.78	0.00	−2.75	0.06	0.79	0.00
A2	0.21	1.23	0.10	0.03	0.21	1.23	0.10	0.04
Black	0.76	2.15	0.17	0.00	0.75	2.12	0.16	0.00
Asian	−1.21	0.30	0.28	0.00	−1.21	0.30	0.27	0.00
Native American	0.42	1.53	0.43	0.32	0.42	1.52	0.43	0.33
Multi-race	−0.53	0.59	0.29	0.07				
Hispanic	−0.69	0.50	0.28	0.00	−0.68	0.51	0.21	0.00
Age	0.08	1.08	0.03	0.02	0.08	1.09	0.03	0.02
Male	−0.23	0.80	0.11	0.05	−0.16	0.85	0.12	0.16
N	2534				2380			
LL	−1572				−1490			
LL(*constant*)	−1615				−1529			

Notes: The model is a mixed-effects logit which estimates a random intercept for each family (not shown). The dichotomous dependent variable is *Partisan*, which is the answer to the question "Do you identify with a specific political party? [1 = yes, 0 = no]" Logit coefficients (Coef), odds ratios (OR), standard errors (SE), and p-values are presented.

APPENDIX 5 Test of Association for Democrat and Republican.

	Democrat				*Republican*			
	Coef	**OR**	**SE**	**P value**	**Coef**	**OR**	**SE**	**P value**
Intercept	−3.10	0.04	0.91	0.00	−4.87	0.01	1.32	0.00
A2	0.16	1.18	0.11	0.15	0.22	1.24	0.17	0.21
Black	2.18	8.84	0.20	0.00	−3.10	0.04	0.44	0.00
Asian	0.08	1.08	0.29	0.79	−2.98	0.05	0.61	0.00
Native American	0.35	1.42	0.43	0.42	−0.56	0.57	0.78	0.48
Hispanic	0.27	1.31	0.22	0.23	−1.77	0.17	0.38	0.00
Age	0.03	1.03	0.04	0.49	0.10	1.10	0.06	0.09
Male	−0.57	0.56	0.14	0.00	0.23	1.25	0.19	0.00
N		2557				2557		
LL		−1101				−947		
LL(*constant*)		−1198				−1014		

Notes: The model is a mixed-effects logit which estimates a random intercept for each family (not shown). The dichotomous dependent variables is *Democrat* and *Republican* are based on the answer to the question "With which party do you identify? [1 = Democrat, 2 = Republican]" Logit coefficients (Coef), odds ratios (OR), standard errors (SE), and p-values are presented.

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