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

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Abstract:

Scholars in many fields have long noted the importance of social context in the development of political ideology. Recent work suggests that political ideology also has a heritable component, but no specific gene variant or combination of variants associated with political ideology have so far been identified. Here, we hypothesize that individuals with a genetic predisposition toward seeking out new experiences will tend to be more liberal, but only if they are embedded in a social context that provides them with multiple points of view. Using data from the National Longitudinal Study of Adolescent Health, we test this hypothesis by investigating an association between self-reported political ideology and the 7R variant of the dopamine receptor D4 gene (*DRD4*), which has previously been associated with novelty seeking. Among those with *DRD4*-7R, we find that the number of friendships a person has in adolescence is significantly associated with liberal political ideology. Among those without the gene variant, there is no association. This is the first study to elaborate a specific gene-environment interaction that contributes to ideological self-identification, and it highlights the importance of incorporating both nature and nurture into the study of political preferences.

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

Mannheim's work would influence several generations of scholars (e.g., Huntington 1957; Bell 1959; Rapoport 1974; North 1978; Lipset 1983; Jackman and Muha 1984; Haas 1992). While many of these scholars disagreed with parts of Mannheim's argument, they all agreed with the premise: that the social and institutional environment is paramount for explaining a person's political attitudes and beliefs. When individuals say they are "liberal" or "conservative," they are referring to their ideas about the issues of the day that are specific to a place and a moment in history. Remove them to another context and their ideology will change.

This literature contrasts with a growing body of work that suggests ideology is not purely a product of the social environment or historical moment. Increasingly, it is becoming apparent that political ideology also has a "core" element that is rooted in innate predispositions, personality, and 'motivated social cognition' (Jost et al. 2003). In fact, psychologists have asserted for many years that *social* conservatism is heritable (Eaves and Eysenck 1974; Cloninger et al. 1993; Bouchard and McGue 2003; Bouchard 2004) and that genetic factors account for a significant proportion of variation in social attitudes (Martin et al. 1986; Tesser 1993). Alford, Funk, and Hibbing (2005) were among the first to present these findings to the political science discipline, showing that genetic variation helps to explain both the direction (liberal vs. conservative) and strength of ideological opinions in a very large sample of twins.

Since then, scholars have found that political attitudes related to vote choice are also heritable (Hatemi et al. 2007) as is the strength of partisan attachment (Hatemi et al. 2009a; Settle, Dawes, and Fowler 2009). Likewise, genetic variation is important for explaining variation in political *behaviors* that are known to be influenced by ideology, like voting and other forms of political participation (Fowler, Baker, and Dawes 2008; Fowler and Dawes 2008; Dawes and Fowler 2009). These findings suggest that we should revise our environment-only understanding of political attitudes and ideology. Political ideology is rooted in general social psychological tendencies and has heritable and durable components that may be constrained or exacerbated by the influence of the social context.

Social and genetic theories about the nature and origin of political ideology need not be at odds with one another. In fact, it is likely that genes influence political ideology by partially regulating the way we react to the total social context. If so, then one way forward in our understanding of the biological and social bases of political attitudes and beliefs is to search for specific gene-environment interactions that may play a role in the development and maintenance of political ideology. Behavior geneticists note that complex social behaviors are *polygenic* (Mackay 2001; Plomin 2008); likewise there are probably many gene-environment interactions that play a role in the acquisition of political ideology. A logical way to start our search is to examine gene variants that are already known to contribute to variation in social behaviors that are related to political ideology. One such gene is the D4 dopamine receptor gene (*DRD4*), which regulates dopamine activity in the brain (Cloninger et al. 1993; Wiesbeck et al. 1995). The 7R allele of this gene has been associated with novelty-seeking behavior (Benjamin et al. 1996; Ebstein et al. 1996; Ebstein et al. 1997b; Nobel et al. 1998; Ebstein et al. 1998; Auerbach et al. 1999; Tomitaka et al. 1999; Strobel et al. 1999; Benjamin et al. 2000; De Luca et al. 2001; Auerbach et al. 2001; Schmidt et al. 2002), which is a tendency that is related to *openness* (De

Fruyt, Van De Wieleb and Van Heeringen 2000), a psychological trait that has been associated with political liberalism (Jost et al. 2003; Pratto et al. 1994; Jost and Thompson 2000; Peterson et al. 1997; Peterson and Lane 2001). This prior research suggests one possible pathway from genes to personality to ideology, but it does not consider the important role of social context. We therefore investigated how the 7R allele might *interact* with an important social variable: the number of friendships a person forms.

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

Ideology: Past Research and Support for a Genetic Basis

We define ideology as a general belief system that encompasses a wide set of idea-elements that come together in a non-random fashion (Gerring 1997, Converse 1964). Here, we refer more specifically to the liberal-conservative continuum commonly understood as organizing American politics (Treier and Hillygus 2005). It has been long debated whether ideology is rooted in issue preferences (Converse 1964) or whether ideological labels are symbolic and affect-oriented (Conover and Feldman 1981). Early studies suggest that Americans have little constraint in their ideology, that they have few opinions about public policy or

political parties, that their opinions on policy change frequently, dramatically, even randomly, and that they have limited capacity to process political information (Campbell et al. 1960; Converse 1964). These early findings have been critiqued from both a methodological and conceptual perspective, and the general consensus has been that the public is not particularly ideological or sophisticated, but neither is it totally unreasoning nor unopinionated (Sniderman et al. 1991). Ideological labels are more salient (Treier and Hillygus 2005; Holbrook 1996; Hinich and Munger 1997) and more meaningful as party leaders and elites polarize (McCarty, Poole, and Rosenthal; Hinich and Munger 1997; Abramowitz and Saunders 1998; Hetherington 2001; Schreckhise and Shields 2003; Jacobson 2003). They are also useful (Lau and Redlawsk 1997, Jacoby 2004), even if not everyone precisely agrees on what the terms "liberal" and "conservative" mean.

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

Dopamine and *DRD4*-7R

Many different types of neurotransmitters can be found in the brain, each with different functions. Dopamine, a member of the catecholamine family, is one such neurotransmitter. The dopamine system affects the control of locomotion, cognition, emotion, positive reinforcement, appetite, and endocrine regulation (Missale et al. 1998). The D4 dopamine receptor DRD4 (one of five subtypes of dopamine receptors) is a protein transcribed by a gene with the same name (*DRD4*). This gene is commonly described by at least three polymorphic variations in its coding sequence (Van Tol et al. 1992), including the allele of interest in this study, the long form allele (7R). These receptors are denser in the limbic system of the brain and the anterior cortex (Golimbet et al. 2007).

DRD4 is associated with brain reward and reinforcement mechanisms (Swift et al. 2000, Golimbet et al. 2007), exploratory approach behavior in animals (Swift et al. 2000), attention disorders (Ashghari et al. 1995, Jovanovic et al. 1999, LaHoste et al. 1996, Swanson et al. 1998, Rowe et al. 1998, Smalley et al. 1998; Langley et al. 2004. McCracken et al. 2000), and novelty-seeking (Benjamin et al. 1996, Ebstein et al. 1996, Ebstein et al. 1997b, Nobel et al. 1998, Ebstein et al. 1998, Auerbach et al. 1999, Tomitaka et al. 1999, Strobel et al. 1999, Benjamin et al. 2000, De Luca et al. 2001, Auerbach et al. 2001, Schmidt et al. 2002). In particular, novelty-seeking is thought to be mediated by genetic variability in dopamine transmission (Cloninger et al. 1993, Wiesbeck et al. 1995). There are several proposed mechanisms, most of which are related to the manner and frequency with which dopamine binds to its receptors.

Studies of animals also indicate that *DRD4* is involved in cortical excitability and behavioral sensitization. These alterations in cortical arousal affect “approach traits” such as novelty-seeking and sensation-seeking, which in turn affect personality and behavior (Eichhammer et al. 2005). People who score high on measures of novelty-seeking have less

tolerance for monotony and constantly seek the new and unusual (to them) in order to alter dopamine levels to affect mood; at the extremes, they are characterized as impulsive, exploratory, fickle, excitable, quick-tempered, and extravagant (Puttonen et al. 2005). People who score low on this measure tend to be more inclined to follow the rules (Golimbet et al. 2007). Those who score lower also tend to be more reflective, rigid, loyal, stoic, slow-tempered, and frugal.

A wide variety of genetic association studies have tested the link between polymorphisms of *DRD4* and novelty-seeking behavior with generally positive results (Kluger et al. 2002; Schinka et al. 2002; Savitz and Ramesar 2004). In particular, Savitz and Ramesar (2004) conduct a meta-analysis of 37 studies, finding that 18 of them show higher than average novelty-seeking test scores to be significantly associated with the long allele in *DRD4*. In 94% of the cases where statistically significant results were found, the implicated allele was the 7R allele or a longer version. Savitz and Ramesar (2004) suggest that the relationship might not always show up because of periodic latency due to interactions between different genes, gene-environment interactions, variation in genetic background, or the presence of other variables. Overall, the association between *DRD4* and novelty-seeking has been shown to be independent of ethnicity, culture, sex, or age (Ebstein et al. 1996; Benjamin et al. 1996; Tomitaka et al. 1999).

Novelty-Seeking, Friends, and Liberalism

Certain situational and dispositional factors may contribute to a cognitive-motivational orientation toward the social world that is either closed and invariant or open and exploratory (Kruglanski and Webster 1996). In fact, "openness to experience," a construct conceptually related to novelty-seeking, is the personality trait most commonly linked to political orientations (Cornelis et al. 2009; Jost et al. 2003; McCrae 1996; Mondak and Halperin 2008) and has been

found to be negatively related to political conservatism generally (Van Hiel et al. 2000) and socio-cultural conservatism specifically (McCrae 1996; Peterson, Smirles and Wentworth 1997; Trapnell 1994; Van Hiel, Kossowska and Mervielde 2000). The relationship between openness to experience and ideology holds when ideology is measured either as support for ideological political parties (Caprara, Barbaranelli & Zimbardo, 1999; Van Hiel et al., 2000) or as ideological self-placement (Carney, Jost, Gosling, Niederhoffer, & Potter, in press; Van Hiel et al., 2000). The connection between openness to experience and ideology may even stem from the same genetic constructs: Verhulst, Eaves and Hatemi (2009) suggest that the relationship between personality and political preferences involves common causal origins as well as gene-environment covariation, whereby personality traits lead an individual to seek out certain environments which in turn affect the development of political orientations. Thus, one possibility is that genes like *DRD4* influence ideology via their effect on personality.

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

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

Finally, it is well-known that peers and friends exert an influence on political preferences (Berelson et al. 1954) and recent work indicates that informal components in the school environment, such as the influence of peer attitudes, contribute to political socialization (Settle 2009). Interestingly, friends may exert even more influence on political ideology if a young person is resentful of parental control (Maccoby, Matthews, and Morton 1954). This is consistent with previous analyses that found significant associations between dopamine and adolescent conduct disorders, which include measures of disobedience (Kirley et al. 2004). Thus, we speculate that people with the 7R allele might also be more inclined to resist parental influence and may be more influenced by their peers, provided they have them.

For these reasons, we hypothesize that the combination of an innate desire for novel experience and many friends may contribute to the activation of a liberal ideology. People who

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

Add Health and Network Properties

We use data from the National Longitudinal Study of Adolescent Health (Add Health) to examine the moderating influence of friendships on the effect of *DRD4* on political ideology. Add Health is a large publicly available study started in 1994-1995 that explores the causes of health-related behavior of adolescents in grades 7 through 12 and their outcomes in young adulthood. In addition to health-related information, a large amount of information has been collected about the personality, attitudes, relationships, religious beliefs, civic activities, and political beliefs and behaviors of the respondents. The initial wave of the study utilized a sampling design that resulted in a nationally representative study. The study has been described elsewhere (Fowler, Baker, and Dawes 2008) and more detailed description of the study can be found on the study web page (http://www.cpc.unc.edu/projects/addhealth).

In Wave I of the Add Health study, researchers created a genetically informative sample of sibling pairs based on a screening of the in-school sample of 90,114 adolescents. These pairs include all adolescents that were identified as twin pairs, half siblings, or unrelated siblings raised together. Twins and half biological siblings were sampled with certainty. The Wave I sibling-pairs sample has been found to be similar in demographic composition to the full Add Health sample (Jacobson & Rowe 1998). Genetic markers are available for a sample of 2,574 individuals, including markers that identify alleles of *DRD4*. Details on access to the study, DNA collection, and genotyping process are available at the Add Health website (Add Health

Biomarker Team 2007).

The analysis of the *DRD4* 48 bp VNTR (variable number tandem repeat) in exon 3 resulted in detection of alleles with base-pair (bp) length of 379, 427, 475, 523, 571, 619, 667, 715, 763 and 811. The two most common alleles were the 475 bp (with four repeats of the 48-bp VNTR), and the 619 bp (with seven repeats of the 48-bp VNTR). Following Hopfer et al. (2005) we group the 379, 427, 475, 523, and 571 bp alleles to form the 4R grouping and 619, 667, 715, and 763 bp alleles into the 7R grouping. In our sample, 62% have no 7R alleles, 33% have one copy of the allele and 5% have two copies of the allele.

Information was gathered in the first wave about a subject's social network. Students were allowed to nominate up to five female and five male friends and were then asked more specific details about those friendships, such as whether or not that friend went to the same school, what grade they were in, if they spent time together after school or over the weekend, and whether the friend was consulted about a problem in the previous week. This information can be used to create a variety of different measures about the respondent's social network, but in this paper we will focus on a simple measure, the number of non-familial friends named by the respondent in the first wave.

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

cited surveys (Jost 2006).

Genetic Association

Genetic association studies test whether an allele or genotype occurs more frequently within a group exhibiting a particular trait than those without the trait (e.g., is the frequency of a particular allele or genotype higher among liberals than conservatives?). However, a significant association can mean one of three things: 1) The allele itself influences ideology; 2) the allele is in "linkage disequilibrium" (LD) with an allele at *another* locus that influences ideology; or 3) the observed association is a false positive signal due to population stratification.[1] Population stratification occurs because groups may have different allele frequencies due to their genetic ancestry. For example, two groups may not have mixed in the past. 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 that are not influenced by allele 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 (Carey 2002). Case-control designs compare the frequency of alleles or genotypes among subjects that exhibit a trait of interest to subjects who do not (controls may be randomly selected from the population or from groups known not to exhibit the trait). As a result, case-control designs are vulnerable to population stratification if either group is especially prone to selection effects. A typical way to deal with this problem is to include controls for the race or

[1] 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. Thus, a significant association means that either a particular allele, or one (likely) *near* it, significantly influences ideology.

ethnicity of the subject or to limit the analysis to a specific racial or ethnic group. Because we know that the 7R allele is found with substantially different frequencies in different ethnic groups (Harpending and Cochran 2002, Ding et al. 2002), we have reason to expect that population stratification could be a problem in our study. Thus, we chose to employ a family-based design, which eliminates the problem of population stratification by using family members, such as parents or siblings, as controls. Tests using family data compare whether offspring exhibiting the trait receive a particular allele from their parents more often than would be expected by chance. They do *not* rely on twins to study genetic variation; any kind of close family relation can be used (siblings, parents, etc.).

Family-Based Design Methods and Results

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

$$y_{ij} = \beta_0 + \beta_b b_i + \beta_w w_{ij} + \beta_E E_{ij} + \beta_{wE} E_{ij} w_{ij}$$
$$w_{ij} = g_{ij} - b_i$$
$$b_i = \frac{\sum_j g_{ij}}{n_i}$$

where y_{ij} is the ideology of individual j in family i, n_i is the number of family members, g_{ij} is the genotypic score which equals the number of 7R alleles (0, 1, or 2), b_i is the expected genotypic score, w_{ij} is an individual's deviation from the expected genotypic score, and E_{ij} is the number of friendships an individual self-reports. A positive value for w_{ij} means that a child inherited an excess number of copies of the 7R allele from his or her parents than expected and thus a significant positive value for β_w indicates that an excess transmission of 7R alleles is associated

with holding a more liberal ideology. A significant positive value for β_{wE} suggests that this association is moderated by the number of friendships one has.

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

We include individuals from the same family in the analysis, and thus the observations are not independent. Therefore, we use a generalized estimating equations approach (Liang and Zeger 1986), with an independent working correlation structure for the clustered errors, to estimate the model. Only siblings that have different genotypes, in this case a different number of 7R alleles, are informative for the within-family component of variance since w_{ij} equals zero otherwise. However, families that share the same genotype are also included in our analysis for improved estimation of the between-family component. We have also included controls in the model for both age and gender, as there are numerous instances of age effects in gene-environment interactions and there are sex-specific genetic influences on political preferences (Hatemi, Medland and Eaves 2009). As is described in the behavior genetics literature (Eaves 1984), a statistical interaction between a gene and the environment will only be present when there is variation in both the gene and the environment. From a statistical perspective, the magnitude of the gene-by-environment interaction can be considered the extent to which the average performance of the gene and the environment fail to predict the response of individual combinations of genotypes and environments. When using genetic analysis, however, it may be best to view the interaction as the genetic control of sensitivity to the environment (Eaves 1984).

The results of the model are presented in Table 1.

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

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

To address further the nature of the friendship interaction, we report additional analyses in the Appendix that demonstrate our results are not being driven by gendered patterns or interactions of friendship and that the results hold when using a dichotomous measure of friendship. We also report the results of the interaction model, but without the interaction term.

The residual deviance for this model is higher than the model with the interaction, indicating that the interaction model fits better. Standard statistical tests of fit do not apply in GEE models (Pan 2002). To be sure the improvement in fit resulting from adding the interaction term to the model was not due to chance, we randomly shuffled the values in the interaction term (leaving the values for all other variables and observations intact) and reran the model, repeating this 1000 times, and measuring the residual deviance each time. The lowest deviance (best fit) out of 1000 tries was 1120.40. By comparison, the null model produces a deviance of 1123.28 and the interaction model produces a deviance of 1116.97, suggesting the interaction model's improvement in fit has less than a 1 in 1000 chance of being caused by random variation.

Discussion

Numerous findings over the last decade have led to wide acceptance that, for most traits, the effects of individual genes are too small to stand out against the combined influence of all other genes and environmental factors (e.g., Hill et al 2008; Visscher 2008). Thus, our *p*-value of 0.02 on a sample of 2000 individuals should be treated cautiously. The expectation in genetics is that only repeated efforts to replicate associations will verify initial findings like these. Thus, perhaps the most valuable contribution of this study is not to declare that "a gene was found" for anything, but rather, to provide the first evidence for a possible gene-environment interaction for political ideology.

Many large-scale analyses of political behaviors ignore the potential for genetic effects. Of those that do not, few offer a model which builds a hypothesis based on social and cultural influences that interact with a specific neurotransmitter that is regulated by a specific genetic marker (for an exception see Hatemi 2008). It is our hope that more scholars will begin to explore the potential interaction of biology and environment, thus leading to the development of

consortiums for social and political traits that will allow for the replication or combination of findings across samples.

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

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

Geneticists are sometimes skeptical of associations where an interaction effect exists and a main effect does not. The reason for this skepticism is a concern with the potential to produce false positive results. If we were testing 100 genes and 100 environmental factors, there would be 10,000 possible interactions, and many of these would yield significant results. However, in our case, the Add Health data contains only five genes, greatly reducing the number of possible interactions one could test. This does not necessarily reduce the possibility of false positive for a given test, but does offer protection from unreported multiple testing. Political scientists also typically guard against false positives by requiring ex-ante theorizing. In our case, we were only

interested in *DRD4* for political ideology because of its association with novelty-seeking behavior, and we developed an explicit theory that suggests the interaction between *DRD4* and friendships—and not the main effects—would be significant. Since our test was not able to contradict the theory, the way forward is to seek replication in different populations and age groups.

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

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

reliable measures in Add Health. All of these limitations suggest that we should develop datasets that include genes, psychological questions to create valid constructs, and political data.

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

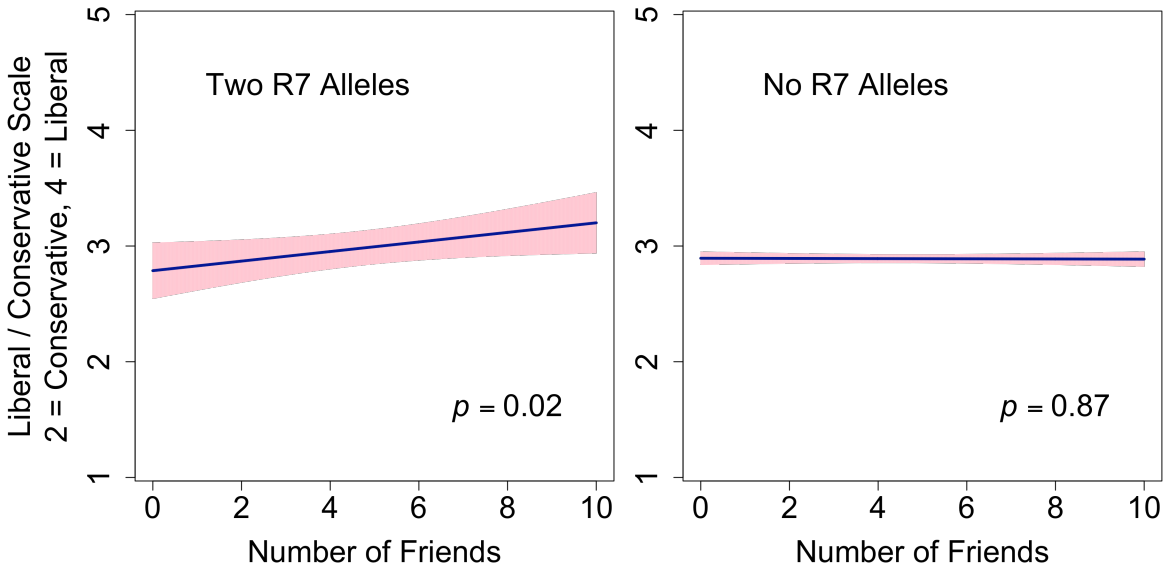
Finally, the results here suggest that, contrary to Mannheim's assertion and the body of work that followed him, the social and institutional environment cannot entirely explain a person's political attitudes and beliefs. We must take into account the role of genes and gene-environment interactions in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

Table 1. Quantitative Disequilibrium Transmission Test of an Association Between *DRD4* and Political Ideology, Including an Interaction with Number of Friendships

	Estimate (standard error)	p-value
Intercept	2.84 (0.23)	0.00
*Between-family component of DRD4-*7R *(b)*	-0.05 (0.04)	0.12
*Within-family component of DRD4-*7R *(w)*	-0.11 (0.11)	0.35
Friendships	-0.00 (0.00)	0.87
Age	0.00 (0.01)	0.67
Male	-0.05 (0.04)	0.16
*w*Friendships*	0.04 (0.02)	0.02
Deviance	1116.97	
N	1941	

Note: This table can be read like an ordinary regression except that the variable coding for *DRD4* is divided into two variables to control for population stratification. The between-family component represents the average number of *DRD4*-7R alleles among all observed members of the subject's family, while the within-family component indicates the excess number of *DRD4*-7R alleles relative to the family average. Null deviance = 1123.28.

Figure 1: Increasing the Number of Friends in People with Two Copies of *DRD4*-7R is Associated with Increased Self-Reported Liberalism



Note: These results are simulated from the model in Table 1 (King, Tomz, and Wittenberg 2000), holding all variables at their means and fixing the within-family component of DRD4-7R to 2 alleles (left) or 0 alleles (right). Dark line indicates mean relationship while shaded areas indicate 95% confidence intervals. Fraction of subjects with each number of friends is shown in the appendix

Appendix

Table A1 shows summary statistics. Tables A2 and A3 show that *DRD4* is not significantly associated with either self-reported ideology or the number of friendship nominations. We also performed the QTDT test for main effects of ideology and number of friends with the *DRD4* marker within an allelic and total association model (Abecasis et al., 2000). Sib-pair identity by descent (IBD) probabilities were estimated in Merlin (Abecasis et al., 2002) by using the *DRD4* marker and included in the QTDT. Linkage and association analyzes for main effects of both ideology and friends were performed on the genotyped sample of DZ twins and siblings using the QTDT program. The combined gene effect on sib-pair differences (the within family component) and the gene effect on the sib-pair means (the between family component) was estimated and no significant main effect was identified.

Table A1. Summary Statistics

Variable	*Mean / Incidence*
Very Liberal	1.7%
Liberal	15.4%
Moderate	57.4%
Conservative	21.9%
Very Conservative	3.5%
White	70.9%
Male	47.8%
0 7R Alleles	62.0%
1 7R Allele	33.1%
2 7R Alleles	4.9%
Average Age	21.9

Variable	*Incidence*
0 Friends	27.6%
1 Friend	4.1%
2 Friends	4.7%
3 Friends	5.3%
4 Friends	7.3%
5 Friends	9.4%
6 Friends	7.2%
7 Friends	7.3%
8 Friends	6.8%
9 Friends	9.6%
10 Friends	10.7%

Table A2. Quantitative Disequilibrium Transmission Test of a Direct Association Between *DRD4* and Political Ideology

	Estimate (standard error)	p-value
Intercept	2.84 (0.23)	0.00
*Between-family component of DRD4-*7R *(b)*	-0.05 (0.04)	0.12
*Within-family component of DRD4-*7R *(w)*	0.09 (0.07)	0.18
Age	0.00 (0.01)	0.66
Male	-0.05 (0.04)	0.15
Deviance	1119.55	
N	1941	

Note: This table can be read like an ordinary regression except that the variable coding for *DRD4* is divided into two variables to control for population stratification. The between-family component represents the average number of *DRD4*-7R alleles among all observed members of the subject's family, while the within-family component indicates the excess number of *DRD4*-7R alleles relative to the family average. The results show that *DRD4*-7R is not directly associated with political ideology. Null deviance = 1123.28.

Table A3. Quantitative Disequilibrium Transmission Test of a Direct Association Between *DRD4* and Number of Friends

	Estimate (standard error)	p-value
Intercept	9.21 (1.09)	0.00
*Between-family component of DRD4-*7R *(b)*	-0.23 (0.17)	0.16
*Within-family component of DRD4-*7R *(w)*	0.16 (0.33)	0.63
Age	-0.20 (0.05)	0.00
Male	-0.39 (0.17)	0.02
Deviance	25125.3	
N	1941	

Note: This table can be read like an ordinary regression except that the variable coding for *DRD4* is divided into two variables to control for population stratification. The between-family component represents the average number of *DRD4*-7R alleles among all observed members of the subject's family, while the within-family component indicates the excess number of *DRD4*-7R alleles relative to the family average. The results show that *DRD4*-7R is not directly associated with the number of friends. Null deviance = 25455.3.

Table A4. Quantitative Disequilibrium Transmission Test of an Association Between *DRD4* and Political Ideology, Including an Interaction with Dichotomous Friendship

	Estimate (standard error)	p-value
Intercept	2.82 (0.24)	0.00
*Between-family component of DRD4-*7R *(b)*	-0.05 (0.04)	0.12
*Within-family component of DRD4-*7R *(w)*	-0.19 (0.13)	0.14
Any Friends? (1 = Yes, 0 = No)	-0.01 (0.04)	0.74
Age	0.00 (0.01)	0.65
Male	-0.05 (0.04)	0.16
*w*Any Friends*	0.37 (0.16)	0.02
Deviance	1116.54	
N	1941	

Note: This table can be read like an ordinary regression except that the variable coding for *DRD4* is divided into two variables to control for population stratification. The between-family component represents the average number of *DRD4*-7R alleles among all observed members of the subject's family, while the within-family component indicates the excess number of *DRD4*-7R alleles relative to the family average. The results show that the interaction effect is robust to different specifications of the friendship variable. Null deviance = 1123.28.

Table A5. Regression Model Testing the Direct Association Between Number of Friends and Political Ideology

	Estimate (standard error)	p-value
Intercept	2.82 (0.24)	0.00
Friendships	-0.00 (0.00)	0.93
Age	0.00 (0.01)	0.69
Male	-0.05 (0.04)	0.17
Deviance	1122.01	
N	1941	

Note: This table can be read like an ordinary regression except that the variable coding for *DRD4* is divided into two variables to control for population stratification. The between-family component represents the average number of *DRD4*-7R alleles among all observed members of the subject's family, while the within-family component indicates the excess number of *DRD4*-7R alleles relative to the family average. The results show that there is no direct relationship between number of friends and ideology. Null deviance = 1123.28.

Table A6. Quantitative Disequilibrium Transmission Test of an Association Between *DRD4* and Political Ideology, Including an Interaction with Number of Male Friendships

	Estimate (standard error)	p-value
Intercept	2.77 (0.24)	0.00
*Between-family component of DRD4-*7R *(b)*	-0.05 (0.04)	0.13
*Within-family component of DRD4-*7R *(w)*	-0.07 (0.11)	0.53
Male Friendships	0.02 (0.01)	0.11
Age	0.01 (0.01)	0.54
Male	-0.06 (0.04)	0.09
*w*Male Friendships*	0.09 (0.04)	0.04
Deviance	1116.97	
N	1941	

Note: This table can be read like an ordinary regression except that the variable coding for *DRD4* is divided into two variables to control for population stratification. The between-family component represents the average number of *DRD4*-7R alleles among all observed members of the subject's family, while the within-family component indicates the excess number of *DRD4*-7R alleles relative to the family average. The results show that the interaction term remains positive and significant when we restrict the analysis to male friendships. Null deviance = 1123.28.

Table A7. Quantitative Disequilibrium Transmission Test of an Association Between *DRD4* and Political Ideology, Including an Interaction with Number of Female Friendships

	Estimate (standard error)	*p*-value
Intercept	2.91 (0.24)	0.00
*Between-family component of DRD4-*7R *(b)*	-0.05 (0.04)	0.12
*Within-family component of DRD4-*7R *(w)*	-0.03 (0.10)	0.80
Female Friendships	-0.01 (0.01)	0.15
Age	0.00 (0.01)	0.78
Male	-0.06 (0.04)	0.09
*w*Female Friendships*	0.05 (0.04)	0.15
Deviance	1117.40	
N	1941	

Note: This table can be read like an ordinary regression except that the variable coding for *DRD4* is divided into two variables to control for population stratification. The between-family component represents the average number of *DRD4*-7R alleles among all observed members of the subject's family, while the within-family component indicates the excess number of *DRD4*-7R alleles relative to the family average. The results show that the interaction term remains positive when we restrict the analysis to female friendships, and the coefficient on this term does not differ significantly from the coefficient for male friendships shown in the model in Table A6. Null deviance = 1123.28.

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