

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

Jaime E. Settle¹
Christopher T. Dawes¹
Peter K. Hatemi²
Nicholas A. Christakis³
James H. Fowler¹

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 associated with political ideology has so far been identified. In this article we hypothesize that individuals with a genetic predisposition towards 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. We find that the number of friendships a person has in adolescence is significantly associated with liberal political ideology among those with *DRD4-7R*. Among those without the gene variant there 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.

¹*Political Science Department, University of California, San Diego, 9500 Gilman Drive 0521, La Jolla, CA 92093-0521, USA*

²*Virginia Institute for Psychiatric and Behavioral Genetics, Virginia Commonwealth University 800 East Leigh Street Biotech 1, office 130-F Richmond, VA 23298-0126, USA*

³*Department of Health Care Policy, Harvard Medical School, and Department of Sociology, Harvard University, Cambridge, MA 02138, USA*

Contact author James Fowler, email: jhfowler@ucsd.edu. Most recent version available at <http://jhfowler.ucsd.edu>. This research was supported by National Institute on Aging grant P-01 AG-031093 and National Science Foundation grant SES-0719404.

In his influential collection of essays, *Utopia and Ideology*, 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 society. While Marx focused particularly on class relations, Mannheim observed that political ideology is a product of the *total* social context of each individual: “there is a correspondence between a given social situation and a given perspective [or] point of view....” (p.51). In other words, to understand fully a person’s political ideology, we need only examine his or her social 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. 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 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 (Dawes and Fowler 2009; Hatemi et al. 2009a; Settle, Dawes, and Fowler 2009). Likewise, genetic factors are important for 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 precisely by 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 genes and gene-environment interactions that play a role in the acquisition of political ideology. A logical way to start our search is to examine genes that are already known to contribute to 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 extraversion and *openness* (De Fruyt, Van De Wieleb and Van Heeringen 2000), the latter being 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 article is the first to identify a specific gene-environment interaction that is associated with the direction of a person's ideological self-identification. For those who carry the 7R allele of *DRD4*, the number of friends a person nominates in adolescence moderates political ideology in early adulthood, with a greater number of friends associated with a more liberal self-identification. Those who do not carry the 7R allele are not similarly affected by their friendship ties. In other words, people who are exposed to a greater number of individual social perspectives as teenagers are likely to become more liberal as adults if they have a gene variant associated with an innate tendency to seek out novel experiences. This finding is an important bridge in our understanding of the formation and transmission of political ideology. For decades, scholars have built their theories of political ideology on the premise that it is a purely social construct shaped only by the social environment, political institutions, and contemporary events. Taking a systems view, noting the importance of factors at multiple levels and their interactions, our findings provide additional evidence that both genes and environments matter, and they point the way towards new theories about how we acquire and maintain our political attitudes and beliefs. Of additional importance, this article offers a guide on how to incorporate molecular genetics methods into the study of political traits.

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 belong together in a non-random fashion (Gerring 1997, Converse 1964). In this paper, we refer more specifically to the liberal-conservative continuum that organizes 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, and randomly, and that they have limited capacity to process political information (Campbell et al. 1960 and 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.

Recent evidence suggests that ideological labels are now more salient than at any point in recent history (Treier and Hillygus 2005, Holbrook 1996, Hinich and Munger 1997). As party leaders and elites continue to polarize ideologically (McCarty, Poole, and Rosenthal, Hinich and Munger 1997, Abramowitz and Saunders 1998, Hetherington 2001, Schreckhise and Shields 2003, Jacobson 2003), the terms “liberal” and “conservative” become more meaningful. Most Americans, approximately 75-80% in recent years, can place themselves on an ideological scale (ANES Table 3.1). Lau and Redlawsk (1997) find that about 75% make the “right” vote based on their expressed preferences and interpretation of those preferences in five elections between 1972 and 1988. Most people can also accurately place parties and candidates along a liberal-

conservative continuum (Jacoby 2004) even though not everyone precisely agrees on what the terms “liberal” and “conservative” mean.

Furthermore, there is a strong association between parental and offspring political attitudes, but past scholars have attributed this to the environmental influences and political socialization to which parents expose their children (Jennings and Niemi 1968). 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). These studies employ variants of the classical twin design, which is a powerful tool for identifying the relative degree to which genetic and environmental factors influence an observed outcome (Evans et al. 2002; Neale and Cardon 1992). The reasoning behind this model relies on the principle that the variance of a political behavior can be partitioned into three distinct components: additive genetic factors (A), shared or common environmental factors (C), and unshared environmental factors (E). 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. This approach can better control for assortative mating as well as account for special twin environmental effects and gene-environment correlation (r_{GE}). In this more precise model, they find that additive genetic influences account for at least 40% of the variance in political and social attitudes. Here, we link our understanding of the genetic basis of political ideology with environmental factors known to influence political attitudes. We suggest that

individuals with the 7R allele of *DRD4*, given certain environmental stimuli, are more likely to have a liberal ideology.

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). The 7R allele of *DRD4* is known to be associated with several characteristics, such as increased extraversion, novelty-seeking, and sensation-seeking (Eichhammer et al. 2005). In turn, these characteristics have been correlated with a number of behavioral and personality traits, such as openness to new experiences (Savitz and Ramesar 2004), drug use, impulsivity, risk-taking (Golimbet et al. 2007), intolerance to monotony, and exploratory behavior (Puttonen et al. 2005). These traits could influence individuals to associate with a more liberal world view based on their social and cognitive predispositions.

In a landmark piece synthesizing five decades of research on conservatism and social-cognitive motives, Jost et al. (2003) assert a motivational basis for the stable, definitional core of conservative ideology, claiming that conservative ideologies are adopted in part to satisfy a variety of social, cognitive, and psychological needs. The authors write that people's response to threatening environmental stimuli, such as fear and uncertainty, affects the development and expression of political beliefs concerning the core components of conservative ideology, such as resistance to change and acceptance of inequality (p. 366).

We would expect to find a negative correlation between traits associated with *DRD4-7R* and a conservative ideology. For example, if those with a more conservative world view perceive new experiences as a threat, they would avoid novelty and sensation seeking in order to

satisfy their psychological needs. This has been demonstrated in the literature with sensation seeking (Kish and Donnenwerth 1972, Kish 1973), a taste for broad-mindedness (Feather 1979, Feather 1984), and openness to experience (Pratto et al. 1994, Jost and Thompson 2000, Peterson et al. 1997, Peterson and Lane 2001). Conservatives are less likely to seek out strong external stimulation in the form of social and nonsocial stimuli (Wilson 1973b). A meta-analysis conducted by Jost et al. found consistent evidence that people who hold politically conservative attitudes are generally less open to new and stimulating experiences (page 356, see Table 4 in Jost et al. 2003 for a summary of these findings).

Certainly, the *DRD4-7R* allele alone does not predispose someone to a liberal ideology. Genetic influences seem to be more strongly expressed as a person ages and may not stabilize until an individual reaches their early 20's and leaves home. Hatemi et al. 2009 use a genetic model to verify earlier claims (Hyman 1959, Jennings and Niemi 1968) that shared environmental factors like parental socialization account for the largest share of the variance in political attitudes and beliefs during adolescence. However, the role of the unshared environment increases over time and plays a bigger role in shaping ideology among adults. Thus, as people move from adolescence into young adulthood, the nonshared components of the environment—such as one's friends—play an important role in the shaping of political ideology. As a result, we might expect environmental factors to *moderate* genetic predispositions for any complex social behavior.

In our study, friendships are modeled as an environmental influence unique to each subject's individual experience. It is important to note that novelty-seeking should not necessarily lead one to make more friends—those with the 7R allele often exhibit certain asocial behaviors—and in fact, using the same sample Fowler, Dawes, Christakis (2009) show that the

number of friendship nominations is not significantly heritable. Rather, the influence of a large number of friends could serve to develop social cognition (Staub 1995), create a better understanding of other's needs (Neibrzydowski 1995) and greater consideration in regard to the society in which they live (Selman 1990, White et al. 1987), as well as increase expression of prosocial behaviors (Hartup 1993). If friends are known to exert influence on political thinking, why should more friends in adolescence make a person more liberal? Unlike the influence of friends at an older age, political influences in childhood aren't explicitly political. Older adults may recruit their friends to volunteer for an electoral campaign, send out political emails, or pass out bumper stickers or buttons. Children, however, are not typically engaged in such activities. Instead, for people 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.

Thus, a combination of innate openness and many friends may contribute to activating a more liberal ideology by exposing a teenager to diversity in the social world. People who have many friends may remain uninterested in their experiences. Alternatively, people who crave new experiences may have limited access to new points of view because they have only a few friends. It is the *combination* of the desire for new experience and many different pathways to these experiences that we hypothesize has an impact on political ideology.

The idea that genetic influences are moderated by the number of friendships a person has is in keeping with the role that friends are known to exert on both social development and political ideology. Political scientists have known since at least the 1950s that ones' peers and friends exert an influence on political preferences (Berelson et al. 1954). Interestingly, friends may exert 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 could speculate that people with the 7R allele might also be more inclined to resist parental influence. It is also possible that people with a predisposition to a liberal ideology may be drawn to each other, reinforcing the development of their shared ideology. A persistent question in the literature has been the causal origin of the association between one's ideology and the ideology of one's friends. Like-minded people may choose to be friends with each other, so the political beliefs friends share may be a result, not a cause, of one's own political ideology. The Add Health data does not permit us to analyze this question specifically because it did not include questions about attitudes towards specific policies or longitudinal ideology measures pre and post "friendship". However, the finding that people with a certain gene variant are more liberal if they have more friends suggests that it is not simply whom one associates with, but the number of people one associates with that can be an equally important influence on political ideology. The amount of information or diversity of sources is just as important as the source of information.

This leads us to believe that other mechanisms besides shared ideology may better explain the moderating effect of friendships. Having many friends increases one's exposure to new experiences (Heiman 2000). For people with the 7R allele who have an innate predisposition to novelty-seeking, there may be something inherent in the process of experiencing new things that promotes a liberal ideology. If novelty is psychologically fulfilling to them, in part due to the way they regulate dopamine, then exposure to new ideas and experiences may come to be associated with positive feelings. Thus, the development of a

personality that is open and extroverted could make a liberal ideology more satisfying to their social-cognitive motives and reward mechanisms (Jost et al. 2003).

Perhaps most importantly, a large number of friendships may change the way a person relates to his or her social environment. Psychologists have in fact found that the number of friendships has important developmental impacts for children and adolescents. Friendships can 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 support prosocial behavior (Hartup 1983). For people who like new experiences, friendships serve to expose a person to the socio-political world, activating a political ideology that is consistent with the open-minded reception to change that characterizes liberalism.

Some Basic Genetics Concepts

Genes are distinct regions of human DNA that form the blueprint for molecules that regulate the development and function of the human body. There are an estimated 25,000 genes (many of which exist in multiple copies) contained in the 46 DNA chains, or 23 chromosome pairs. Almost all cells in a person's body contain the same inherited DNA which remains relatively fixed throughout life, except for random mutations. DNA stability is an important feature, rendering it one of the purest available measures of biological inheritance. While the environment can impact DNA via transcriptional and epigenetic modifications to influence gene expression, and the products of genes are certainly affected by the environment, the primary

DNA sequence remains virtually unaffected and is able to be collected at any point throughout a person's life.

Many genes have multiple variants, called “alleles”—for example, sickle cell disease results from a particular allele coding for abnormal rather than normal hemoglobin. In somatic cells, each parent has two separate alleles at each “locus,” or location on each chromosome pair, but each sperm or egg cell (gametes) contains only one of these alleles. At conception, a single gamete from each parent fuse, resulting in a cell whose DNA code is composed in equal parts of DNA from the mother and DNA from the father. Thus a child has a 50% chance of receiving a particular allele from a particular parent. For example, suppose that at a given locus there are two possible alleles, A and B. If both parents are “heterozygous” at that locus, meaning they each have an A and a B allele (AB), then a given offspring has a 25% chance of being “homozygous” for A (AA), a 25% chance of being homozygous for B (BB), and a 50% chance of being heterozygous (AB or BA—order is irrelevant).

Genes have their information transcribed into RNA, which is subsequently translated into amino acids, which are then used to form proteins. Proteins are the end products of genetic expression and are the molecules responsible for regulating bodily structure and function. Most observable traits and behaviors, referred to as “phenotypes,” are far downstream from the original “genotypes” present in the DNA. While in very rare cases one allele can single-handedly lead to a disease (such as sickle cell anemia, Huntington disease, and cystic fibrosis), the vast majority of phenotypes are “polygenic,” meaning they are influenced by multiple genes (Mackay 2001, Plomin 2008) and are shaped by a multitude of environmental forces.

Neurobiology and *DRD4-7R*

Genes encode information that affects every system in the body and new pathways linking genotype and phenotype are constantly being identified. One of the areas in which this search has been most fruitful is in neurobiology, where much progress has been made in our understanding of how genes affect development and biochemical processes that in turn affect behavior.

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, between the axon of a sending neuron and the 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. Signals are carried throughout the body by the sequential firing of one neuron after another across these synapses.

The brain is made up of many different types of neurotransmitters, 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). Dopamine influences these physiological processes by activating at least five different dopamine receptors (D1, D2, D3, D4, and D5) located throughout the brain, including the striatum, amygdale, caudatus, and putamen. There is strong evidence that impairments of the dopamine system are implicated in neurological, psychiatric and drug addiction disorders, and mental illness (Hurd & Hall 2005).

The D4 dopamine receptor, or *DRD4*, is expressed in the dorsolateral prefrontal and entorhinal cortex, the hippocampus, hypothalamus, globus pallidus, substantia nigra, and dorsal-

media thalamus (Paterson et al. 1999, Savitz and Ramesur 2004) and is considerably homologous to other dopamine receptors (Van Tol et al. 1991). At least three polymorphic variations in the coding sequence of the human D4 receptor exist (Van Tol et al. 1992), including the allele of interest in this study, the long form allele (7R) which has a seven-fold repeat of a 48-base pair sequence (“variable number tandem repeats,” or VNTR) in exon 3 of the gene which encodes the third cytoplasmic loop of the receptor. The location of the polymorphism on that loop suggests that the variation in the alleles of the gene differ in function related to the mediation of intracellular signaling (Savitz and Ramesar 2004). These receptors are denser in the limbic system of the brain and the anterior cortex, which implies a relationship to the investigative activity in relation to new stimuli (Golimbet et al. 2007).

DRD4 is associated with the brain reward and reinforcement mechanism (Swift et al. 2000, Golimbet et al. 2007), the 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 the novelty-seeking personality trait (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). 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”—

extraversion, novelty-seeking, and sensation-seeking—which in turn affect personality and behavior (Eichhammer et al. 2005). These traits are linked to the openness and life activity of an individual and his or her tendency to take risks and alter views. People who score high on measures of novelty-seeking have less tolerance for monotony and constantly seek the new and unusual in order to alter dopamine levels to affect mood; 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 conservative in nature and are 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).

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; women comprise 49% of the study's participants, Hispanics 12.2%, Blacks 16.0%, Asians 3.3%, and Native Americans 2.2%. Participants in Add Health also represent all regions of the country: the Northeast makes up 17% of the sample, the South 27%, the Midwest 19%, and the West 17%. Wave I included participation from 145 middle, junior high, and high schools; from those schools, 90,118 students completed a 45-minute questionnaire. This process generated descriptive information about each student, the educational setting, and the environment of the school.

From these respondents, a core random sample of 12,105 adolescents in grades 7-12 were drawn plus several over-samples, totaling more than 27,000 adolescents. These students and their parents were administered in-home surveys in the first wave. Wave II (1996) was comprised of another set of in-home interviews of more than 15,000 students from the Wave I sample. Finally, Wave III (2001-2002) consisted of an in-home interview of 15,170 Wave I participants.

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 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

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.

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 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 solely 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:

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

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.

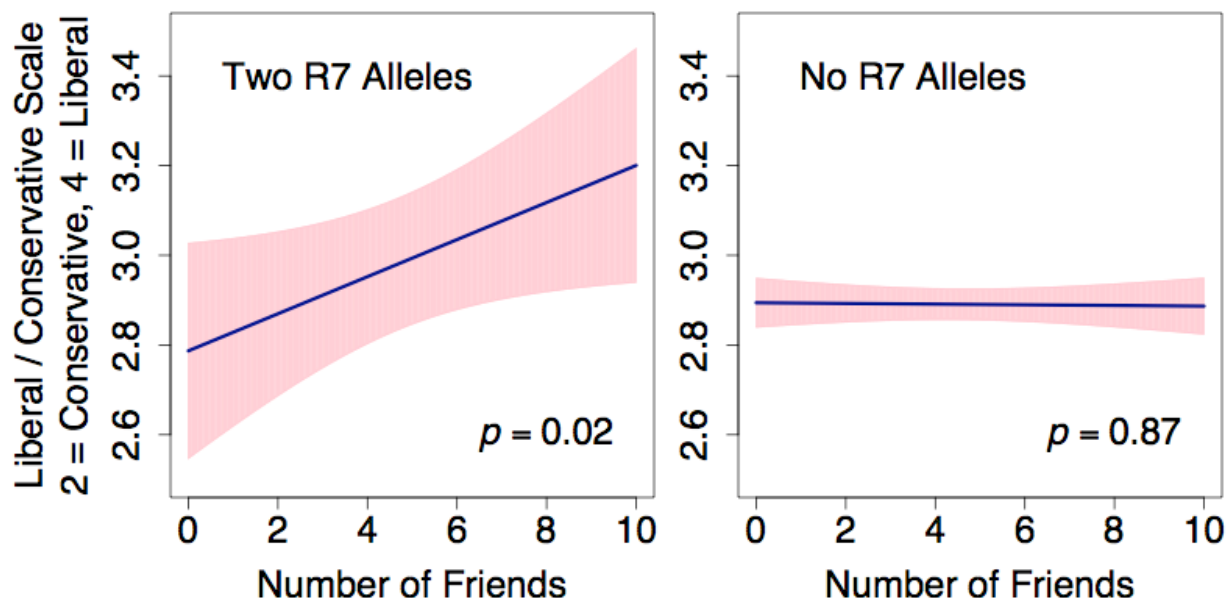
The parameters of primary interest are β_w and β_{wE} because they represent formal tests of association. The estimate of β_w is not significant ($p=0.35$) suggesting that there is not a direct association between the 7R allele and ideology. However, the estimate of the interaction β_{wE} is significant ($p=0.02$), 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 of the significant result, 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

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
<i>Intercept</i>	2.84 (0.23)	0.00
<i>Between-family component of DRD4-7R (b)</i>	-0.05 (0.04)	0.12
<i>Within-family component of DRD4-7R (w)</i>	-0.11 (0.11)	0.35
<i>Friendships</i>	-0.00 (0.00)	0.87
<i>Age</i>	0.00 (0.01)	0.67
<i>Male</i>	-0.05 (0.04)	0.16
<i>w*Friendships</i>	0.04 (0.02)	0.02
<i>Log Likelihood</i>	-2233.9	
<i>N</i>	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 log likelihood = -2246.6.

Figure 1: Increasing the Number of Friends in People with Two Copies of *DRD4-7R* Makes Subjects More Liberal



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

that the interaction is not the result of a direct association between 7R alleles and ideology or friendships, we conduct tests without the interaction. The results of these tests are presented in the appendix and show that 7R alleles have no significant effect on the number of friends, and no direct impact on ideology. Moreover a likelihood ratio test suggests the interaction term significantly improves the fit of the model (ratio=10.2, $p=0.001$).

Figure 1 is a graphical representation of the interaction effect between the presence of the alleles and the number of friendships. For those without any 7R alleles, the number of friends has no effect on 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 would have the effect of 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.

Discussion

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 Appendix 3), and the number of friends one has does not appear to be heritable (Fowler, Dawes, Christakis 2009). Thus, we can rule out a spurious relationship between the allele and ideology. Rather, it is the crucial interaction of the 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.

The Adolescent Health study was not intended to focus on political socialization or any political trait. Thus, there are a number of limitations to our study. The model requires the assumption that the greater number of friends one has, the more viewpoints to which one is exposed. As adults, individuals tend to affiliate with other individuals who are similar to them, and as adolescents members of social groups are pressured to conform to the group (Engles,

Drop and de Haan 1997). Without a sample that assesses adolescent values before and after friendships take place we cannot test this assumption. This is of particular importance when assessing the robustness of the GxE interaction. We tested whether the interaction was the result of gene-environment covariation (rGE) or a genetic predisposition to select more friends. If individuals have a heritable tendency to make more friends or enter situations where more friendships can be formed, rather than having a GxE interaction predicting ideology, the findings would reflect a partial gene x gene interaction (GxG). In our sample, the number of friends nominated is treated as an environmental influence, since the additive genetic variation for number of friends in this sample is not significant (Fowler, Dawes, and Christakis 2009) and there is no direct influence of *DRD4* on the number of nominated friends (see Appendix 3). However, past work has shown that genes might be associated with relevant aspects of friendship like social support and the amount of peer influence (Kendler 1997). Whether the number of friends nominated is associated with these other tendencies, and whether our significant interaction may also be including these effects, such that there is actually a GxG interaction underlying political ideology is an open question that should be explored in future work.

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.

There are likely many interactions between genes and the environment that affect political ideology, and different genes affect behavioral responses to different features of the environment. This expectation is borne out. While our finding is statistically significant, the effect size of the interaction is quite small. However, even in a biometric trait such as height, which would be quite difficult to argue is social in nature, less than 15% of the variation can be attributed to specific genes. Genetic effects take place in complex interaction with other genes and environments, and it will likely be the combination of hundreds if not thousands of genes interacting with each other and with external stimuli that influence behavior. The preponderance of evidence across traits provides little evidence of a general GxE interaction that affects all people in all environments at all times. Rather, it seems that there are genetic predilections that influence reactions to or interpretations of specific environmental stimuli that may only apply to certain populations or age groups (Eaves 1984). For example, novelty-seeking is known to decline with age (Savitz and Ramesur 2004), which suggests that the environment may play an increasingly important role in moderating genetic predispositions in this trait. Ideology also continues to be flexible with age. Contrary to popular thought, studies of intergenerational attitude change find that older adults do not necessarily become more conservative, indicating that one's ideology can evolve in response to social and cultural change even as one ages (Danigelis, Hardy and Cutler 2007). While we predict that the number of social ties a person has

would continue to exert an influence on a person's ideology, it is possible that the mechanism by which environmental influences affect genetic predispositions changes over the course of a lifetime. Age effects are not possible to test in this sample. The Add Health sample is restricted to individuals who are 18-26 years old during Wave III, so it is possible that our results apply only to the initiation of ideological identification in young adults and not to its subsequent development. Tests of the persistence of ideology later in life would require additional data and analysis.

There are several factors that would be instrumental for future replication studies. We do not have an explicit novelty-seeking measure in the Add Health data. If we did, we could test the extent to which the measure moderates the associations we observe here. We were also unable to test if the GxE interaction which predicted increases in liberalism would differ from a baseline before the measure of number of friends was taken. A longitudinal study is necessary for this type of exploration. Finally, 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.

Our common ancestors would have received little evolutionary benefit from a gene that bequeathed them with a liberal ideology. We do not suggest that *DRD4* in any way evolved as a "liberal" gene. However, a combination of findings has led scholars to believe that the 7R allele did have some evolutionary benefit at an earlier time in human history. The *DRD4* gene shows significant variation in human populations; the 4R allele is the most frequent, but frequencies vary greatly across different populations (Ding et al. 2002, Chang et al. 1996). Studies suggest

that the origin of the shorter alleles can be explained by simple one-step recombination or mutation events, but that the 7R allele is “younger,” originating approximately 40,000 years ago perhaps as a rare mutational event that increased to high frequency in the human population because of positive selection (Ding et al. 2002). Wang et al. (2004) confirm that the pattern of recombination is that expected for selection acting at the 7R VNTR itself, rather than at an adjacent site.

Ding et al. (2002) argue that the appearance of new technology and/or the development of agriculture could be related to the increase in *DRD4*-7R allele frequency. Novelty-seeking in this era could have been associated with reproductive success if carriers of the 7R allele were able to more easily adapt to changing environmental and societal conditions. Selection could have acted against those who were averse to change and experimentation. Furthermore, those with the 7R allele may have been more likely to migrate, leading to the wide dispersion of the allele across ethnic groups and the high prevalence of the allele in groups, such as those in South America, located long distances away from the site of human origins (Ding et al. 2002, Harpending and Cochran 2002).

We no longer face the same environmental challenges that our ancestors did, and it is argued that the presence of many alleles in our genome is a legacy to the benefit we accrued from it in the past (Gangestad and Simpson 2007; Barkow Cosmides, and Tooby 1992; Tooby and Cosmides 1990). When considering the contribution of specific genes to political and other social behaviors, it is important to keep this evolutionary perspective in focus. Population genetics do not evolve as rapidly as society, and thus the genes we inherit influence our response to modern situations, even if those genes have survived and propagated for very different reasons. The long alleles of the *DRD4* gene may have helped our ancestors explore and innovate

in the environment they faced 40,000 years ago, separating novelty-seekers from their more generally unadventurous peers. In today's environment, those same alleles may indirectly contribute to one's political ideology as a legacy of the adaptations of our past (Corning 2004; Tooby and Cosmides 1992). However, adaptations for evolutionary phylogenetic advantage were not intended for today's modern environment and no claim can be made that novelty-seeking or the 7R genotype is a positive force. Rather, novelty seekers and those with the 7R allele in today's environment are also more likely to engage in socially damaging behaviors such as drug addiction.

Our finding has important implications for how we describe and measure political ideology as well as our understanding of how people relate to the political world. 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.

First, this modifies our understanding of the definition of ideology as a pure constraint over a set of issues in one historical context. Political scientists often find that most people are not constrained in their beliefs and that a certain amount of political sophistication is required for a coherent ideology. However, we know that ideology is associated with certain dispositions and we have established an interactive association between a gene, those predispositions, and a liberal ideology. Thus, as Schreiber (2005) suggests, the measured lack of ideological sophistication may reflect a difficulty in mapping values onto the policy space, not an absence of ideology. Ideology itself is very stable and tied to complex psychological and personality characteristics. We all have ideological tendencies, but some of us are better at applying those

tendencies to the contemporary events around us. Second, this bolsters our idea of ideology both as an affective association but also as motivated social cognition (Jost et al. 2003). Our work builds upon Jost's, offering a genetic basis for the link between motivated social cognition and ideology.

Finally, this finding provides evidence for a move away from purely environmental models that were so fundamental to the field for many years. Alford, Funk, and Hibbing (2005) find that genes and the non-shared environment have the most impact on an index of conservative ideology, and our results expand upon theirs. Parents shape their children's ideology through the contribution of their genes, and it is interactions outside the home that have the greatest environmental influence. Genes may lead people to seek out certain experiences, reinforcing their predispositions (Scarr and McCartney 1983). However, our findings reinforce the notion that environmental influences without genetic precursors also play an important role in shaping political behavior. This emphasizes that both genes and the environment matter: genes affect political behavior by regulating the way we engage within our total social context.

Our finding makes it clear 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. However, 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. Thanks to the vast literature on political behavior, political scientists have a wealth of material from which to form hypotheses

about potential gene-environment interactions. Our finding will be just the first of many fascinating results to come.

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

Very Liberal	1.7%	0 Friends	27.6%
Liberal	15.4%	1 Friend	4.1%
Moderate	57.4%	2 Friends	4.7%
Conservative	21.9%	3 Friends	5.3%
Very Conservative	3.5%	4 Friends	7.3%
White	70.9%	5 Friends	9.4%
Male	47.8%	6 Friends	7.2%
0 7R Alleles	62.0%	7 Friends	7.3%
1 7R Allele	33.1%	8 Friends	6.8%
2 7R Alleles	4.9%	9 Friends	9.6%
Average Age	21.9	10 Friends	10.7%

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

	Estimate (standard error)	p-value
<i>Intercept</i>	2.84 (0.23)	0.00
<i>Between-family component of DRD4-7R (b)</i>	-0.05 (0.04)	0.12
<i>Within-family component of DRD4-7R (w)</i>	0.09 (0.07)	0.18
<i>Age</i>	0.00 (0.01)	0.66
<i>Male</i>	-0.05 (0.04)	0.15
<i>Log Likelihood</i>	-2239.1	
<i>N</i>	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 log likelihood = -2246.6.

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

	Estimate (standard error)	p-value
<i>Intercept</i>	9.21 (1.09)	0.00
<i>Between-family component of DRD4-7R (b)</i>	-0.23 (0.17)	0.16
<i>Within-family component of DRD4-7R (w)</i>	0.16 (0.33)	0.63
<i>Age</i>	-0.20 (0.05)	0.00
<i>Male</i>	-0.39 (0.17)	0.02
<i>Log Likelihood</i>	-50250.6	
<i>N</i>	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 log likelihood = -50910.6.

Works Cited

- Abecasis, G., L. Cardon, and W. Cookson. 2000. A General Test of Association for Quantitative Traits in Nuclear Families." *American Journal of Human Genetics* 66: 279-92.
- Abecasis, G. R., Cherny, S. S., Cookson, W. O., & Cardon, L. R. 2002. "Merlin-rapid analysis of dense genetic maps using sparse gene flow trees". *Nature Genetics* 30:97-101.
- Abramovitz, Alan I. and Kyle L. Saunders. 1998. "Ideological Realignment in the U.S. Electorate." *Journal of Politics* 60: 634-652.
- Alford, John, Carolyn Funk, and John Hibbing. 2005. "Are Political Orientations Genetically Transmitted?" *American Political Science Review* 99(2005): 153-167.
- ANES Guide to Public Opinion and Political Behavior. "Table 3.1: Liberal Conservative Self-Identification 1972-2004." Available at http://www.electionstudies.org/nesguide/toptable/tab3_1.htm
- Asghari, V., S. Sanyal, S. Buchwaldt, A. Paterson, V. Jovanovic, H. H. Van Tol. 1995. "Modulation of Intracellular Cyclic AMP Levels by Different Human Dopamine D4 Receptor Variants." *Journal of Neurochemistry* 65: 1157-1165.
- Auerback, J., V. Geller, S. Lezer, E. Shinwell, R. H. Belmaker, and J. Levine. 1999. "Dopamine D4 Receptor (DRD4) and Serotonin Transporter Promoter (5-HTTLPR) Polymorphisms in the Determination of Temperament in 2-Month-Old Infants." *Molecular Psychiatry* 4: 369-373.
- Barkow, J., Cosmides, L. & Tooby, J., editors. 1992. *The Adapted Mind: Evolutionary Psychology and the Generation of Culture*. New York: Oxford University Press.
- Bell, Daniel. 1959. *The End of Ideology*. Glencoe: The Free Press.
- Benjamin, J., L. Li, C. Patterson, B. D. Greenberg, D. L. Murphy, D. H. Hamer. 1996. "Population and Familial Association Between the D4 Dopamine Receptor Gene and Measures of Novelty Seeking." *Nature Genetics* 12: 81-84.
- Benjamin, J., Y. Osher, M. Kotler, I. Gritsenko, L. Nemanov, R.H. Belmaker, R. P. Ebstein. 2000. "Association Between Tridimensional Personality Questionnaire Traits and Three Functional Polymorphisms: DrD4, 5-HTTLPR, and COMT." *Molecular Psychiatry* 5: 96-100.
- Berelson, Bernard, Paul F. Lazarsfeld, and William N. McPhee. 1954. *Voting*. Chicago: University of Chicago Press.
- Bishop, J. and H. Inderbitzen. 1995. "Peer Acceptance and Friendship: An Investigation." *Journal of Early Adolescence* 15: 476-489.
- Bouchard, T. J., and M. McGue. 2003. "Genetic and Environmental Influences on Human Psychological Differences." *Journal of Neurobiology* 54(1):4-45.
- Bouchard, Thomas. 2004. "Genetic Influence on Human Psychological Traits." *Current Directions in Psychological Science* 13(4): 148-151.
- Campbell, Angus, Philip E. Converse, Warren E. Miller, and Donald E. Stokes. *The American Voter*. Chicago: University of Chicago Press: 1960.

- Carey, Gregory. 2002. *Human Genetics for the Social Sciences*. New York: Sage Publications.
- Castellanos, F. X., E. Lau, N. Tayebi, P. Lee, R. E. Long, J. N. Giedd, W. Sharp, W. L. Marsh, J. M. Walter, S. D. Hamburger, E. I. Ginns, J. L. Rapoport, E. Sidransky. 1998. "Lack of an Association between a Dopamine-4 Receptor Polymorphism and Attention-Deficit/Hyperactivity Disorder: Genetic and Brain Morphometric Analyses." *Molecular Psychiatry* 3: 431-434.
- Chang, F.-M., J.R. Kidd, K. J. Livak, A. J. Pakstis, K. K. Kidd. 1996. "The World-Wide Distribution of Allele Frequencies at the Human Dopamine D4 Receptor Locus." *Human Genetics* 98: 91-101.
- Cloninger, C. R., D. M. Svrakic, T. R. Przybeck. 1993. "A Psychobiological Model of Temperament and Character." *Archives of General Psychiatry* 50: 975-990.
- Conover, Pamela and Stanley Feldman. "The Origins and Meaning of Liberal/Conservative Self-Identification," *American Journal of Political Science* 25 (1981):617-645.
- Converse, Philip E. 1964. The Nature of Belief Systems in Mass Publics. In *Ideology and Discontent*, ed. David E. Apter. New York: The Free Press, pp. 206-261
- Cook, E. H., M.A. Stein, M. D. Krasowski, N. J. Cox, D. M. Olkon, J. E. Kieffer, B. L. Leventhal. 1995. "Association of Attention-Deficit Disorder and the Dopamine Transporter Gene." *American Journal of Human Genetics* 56: 993-998.
- Corning, Peter A. 2004. "An Evolutionary Theory of Politics." In *Handbook of Evolution*. Eds. F. Wuketits and C. Antweiler. Weinheim : Wiley-VCH.
- Danigelis, Nicholass, Melissa Hardy and Stephen J. Cutler. 2007. "Population Aging, Intracohort Aging, and Sociopolitical Attitudes." *American Sociological Review* 72(5): 812-830.
- Dawes, Christopher T. and James H. Fowler. 2009. "Partisanship, Voting, and the Dopamine D2 Receptor Gene" *Journal of Politics*, forthcoming
- De Fruyt, F., L. Van De Wieleb and C. Van Heeringen. 2000. "Cloninger's Psychobiological Model of Temperament and Character and the Five-Factor Model of Personality." *Personality and Individual Differences* 29(3): 441-452
- De Luca, A., M. Rizzardi, A. Buccino, R. Alessandroni, G.P. Salvioli, N. Filograsso, G. Novelli, B. Dallapiccola. 2003. "Association of Dopamine D4 Receptor (DRD4) Exon III Repeat Polymorphism with Temperament in 3-year-old Infants." *Neurogenetics* 4: 207-212.
- De Luca, A., M. Rizzardi, I. Torrente, R. Alessandroni, G. P. Salvioli, N. Filograsso, B. Dallapiccola, G. Novelli. 2001. "Dopamine D4 Receptor (DRD4) Polymorphism and Adaptability Trait During Infancy: A Longitudinal Study in 1- to 5-Month-Old Neonates." *Neurogenetics* 3: 79-82.
- Ding, Y.-C.; H.-C. Chi, D. L. Grady, A. Morishima, J. R. Kidd, K. K. Kidd, P. Flodman, M. A. Spence, S. Schuck, J. M. Swanson, Y.-P. Zhang, R. K. Moyzis. 2002. "Evidence of Positive Selection Acting at the Human Dopamine Receptor D4 Gene Locus." *Proceedings of the National Academy of Sciences* 99: 309-314.
- Eaves LJ. 1984. "The resolution of genotype x environment interaction in segregation analysis of nuclear families." *Genet. Epidemiol.* 1:215-28

- Eaves, L. J. and H. J. Eysenck. 1974. "Genetics and the Development of Social Attitudes." *Nature* 249: 288-289.
- Ebstein, R. P., L. Nemanov, I. Klotz, I. Gritsenko, R. H. Belmaker. 1997. "Additional evidence for an Association between the Dopamine 4 Receptor (DRD4) Exon III Repeat Polymorphism and the Human Personality Trait of Novelty Seeking." *Molecular Psychiatry* 2: 472-477.
- Ebstein, R. P., O. Novick, R. Umansky, B. Pirelli, Y. Osher, D. Blaine, E. R. Bennett, L. Nemanov, M. Katz, R. H. Belmaker. 1996. "Dopamine D4 Receptor Exon III Polymorphism Associated with the Human Personality Trait of Novelty Seeking." *Nature Genetics* 12: 78-80.
- Ebstein, R.P., J. Levine, V. Geller, J. Auerbach, I. Gritsenko, R. H. Belmaker. 1998. Dopamine D4 Receptor and Serotonin Transporter Promoter in the Determination of Neonatal Temperament. *Molecular Psychiatry* 3: 238-246.
- Eichhammer, P., P. G. Sand, P. Sooertebecker, B. Langguth, M. Zowe, and G. Hajak. 2005. "Variation at the DRD4 Promoter Modulates Extraversion in Caucasians." *Molecular Psychiatry* 10: 520-522.
- Engels, R.C. , R.A. Knibbe, M.J. Drop and Y.T. de Haan. 1997. "Homogeneity of cigarette smoking within peer groups: Influence or selection?" *Health Education & Behavior* 24 (6):801-811.
- Evans, D.M., N.A. Gillespie, and N.G. Martin. 2002. "Biometric Genetics." *Biological Psychology* 61:33-51.
- Feather, N.T. 1979. "Value Correlates of Conservatism." *Journal of Personality and Social Psychology* 37: 1617-1630.
- Feather, N. T. 1984. "Protestant Ethic, Conservatism, and Values." *Journal of Personality and Social Psychology* 46: 1132-1141
- Fowler, James H., Laura Baker and Christopher T. Dawes. 2008. "Genetic Variation in Political Behavior." *American Political Science Review* 102(2): 233-248.
- Fowler, James H. and Christopher T. Dawes. 2008. "Two Genes Predict Voter Turnout." *Journal of Politics* 70(3): 579-594.
- Fowler, James H., Christopher T. Dawes, and Nicholas A. Christakis. 2009. "A Model of Genetic Variation in Human Social Networks." *Proceedings of the National Academy of Sciences* 106(5).
- Fulker, D., S. Cherny, P. Sham, and J. Hewitt. 1999. "Combined Linkage and Association Sib-Pair Analysis for Quantitative Traits." *American Journal of Human Genetics* 64: 259-67
- Gangestad, S. W. and J. A. Simpson, eds. 2007. *The Evolution of Mind: Fundamental Questions and Controversies*. New York, NY: The Guilford Press
- Gauderman, W. James. 2003. "Candidate Gene Association Analysis for a Quantitative Trait, Using Parent-Offspring Trios." *Genetic Epidemiology* 25: 327-338.
- Gerring, John. 1997. "Ideology: A Definitional Analysis." *Political Research Quarterly* 50: 957-994.

- Golimbet, V. E., M. V. Alfimova, I. K. Gritsenko, and R. P. Ebstein. 2007. "Relationship Between Dopamine System Genes and Extraversion and Novelty Seeking." *Neuroscience and Behavioral Physiology* 37(6): 601-606.
- Haas, Peter M. 1992. "Epistemic Communities And International-Policy Coordination – Introduction." *International Organization* 46 (1): 1-35.
- Harpending, H. and G. Cochran. 2002. "In Our Genes." *Proceedings of the National Academy of Sciences* 99: 10-12.
- Hartup, W. 1983. "Peer Relations." In P.H. Mussen (Series Editor) and E.M. Hetherington (Vo. Ed.), *Handbook of Child Psychology: Vol. 4. Socialization, Personality and Social Development* (pp. 103-196). New York: John Wiley & Sons
- Hatemi, P.K., S.E. Medland, K.I. Morley, A.C. Heath and N.G. Martin. 2007. "The Genetics of Voting: An Australian Twin Study." *Behavior Genetics* 37(3):435-448.
- Hatemi, Peter K., John Hibbing, John Alford, Nicholas Martin and Lindon Eaves. 2009a. "How Much of a Party is in Your Genes." *Political Research Quarterly* (Forthcoming).
- Hatemi, P.K., C. Funk, S.E. Medland, H. Maes, J. Silberg, N.G. Martin, and L.J. Eaves. 2009b. "Genetic and Environmental Transmission of Political Attitudes over the Life Course." *Journal of Politics* (forthcoming)
- Hatemi, Peter K., Sarah E. Medland and Lindon J. Eaves. 2009c. "Genetic Sources for the Gender Gap?" *Journal of Politics* 71(1):1-13.
- Hatemi, Peter K., John Hibbing, John Alford, Nicholas Martin and Lindon Eaves. 2008. "We Get Opinions from Our Parents, but Not How We Think We Do: Genetic and Social Components of the Familial Transmission of Political Attitudes" paper presented at the American Political Science Association Conference, Sept 2007, Chicago, IL
- Heiman, Tali. 2000. "Quality and Quantity of Friendship: Students' and Teachers' Perceptions." *School Psychology International* 21: 265-280.
- Hetherington, Marc J. 2001. "Resurgent Mass Partisanship: The Role of Elite Polarization." *American Political Science Review* 95: 619-631.
- Hinich, Melvin J. and Michael C. Munger. 1997. *Analytical Politics*. Cambridge: Cambridge University Press.
- Holbrook, Thomas M. 1996. *Do Campaigns Matter?* Thousand Oaks, CA: Sage Publications Inc.
- Huntington, Samuel P. 1957. "Conservatism As An Ideology." *American Political Science Review* 51 (2): 454-473.
- Hurd, Y. and H. Hall. 2005. *Handbook of Chemical Neuroanatomy: Vol 21*. Elsevier Chapter Human Forebrain Dopamine Systems: Characterization of the Normal Brain and in Relation to Psychiatric Disorders.
- Hyman, Herbert H. 1959. *Political Socialization*. Glencoe, IL: Free Press.
- Jackman, Mary R. and M.J. Muha. 1984. "Education And Intergroup Attitudes - Moral Enlightenment, Superficial Democratic Commitment, Or Ideological Refinement?" *American Sociological Review* 49 (6): 751-769.

- Jacobson, Gary. 2003. "Partisan Polarization in Presidential Support: The Electoral Connection." *Congress & The Presidency* 30(1): 1-36.
- Jacobson, K. and D. Rowe. "Genetic and Shared Environmental Influences on Adolescent BMI: Interactions with Race and Sex." *Behavior Genetics* 28(4): 265-278.
- Jacoby, William. 2004. *Ideology in the 2000 Elections: A Study in Ambivalence. Models of Voting in Presidential Elections*. Stanford: Stanford University Press.
- Jennings, M. K. and Richard G. Niemi. 1968. "The Transmission of Political Values from Parent to Child." *American Political Science Review* 62(1): 169-184.
- Jost, J.T. 2006. The end of the end of ideology. *American Psychologist* 61 (7): 651-670.
- Jost, J.T. and Glaser, J. and Kruglanski, A.W. and Sulloway, F.J. 2003. "Political conservatism as motivated social cognition." *Psychological Bulletin* 129 (3): 339-375.
- Jost, J.T. and E. P. Thompson. 2000. "Group-Based Dominance and Opposition to Equality as Independent Predictors of Self-Esteem, Ethnocentrism, and Social Policy Attitudes among African Americans and European Americans." *Journal of Experimental Social Psychology* 36: 209-232.
- Jovanovic, V., H.-C. Guan, H H. M. Van Tol. 1999. "Comparative Pharmacological and Functional Analysis of the Human Dopamine D4.2 and D4.10 Receptor Variants." *Pharmacogenetics* 9: 561-568.
- Kendler KS. 1997. "Social Support: A Genetic-Epidemiologic Analysis." *Am J Psychiatry* 154:1398-1404
- Kendler KS, Eaves LJ. 1986. "Models for the joint effect of genotype and environment on liability to psychiatric illness." *Am. J. Psychiatry* 143:279-89.
- King G, Tomz M, and Wittenberg J. 2000. "Making the most of statistical analyses: improving interpretation and presentation." *American Journal of Political Science* 44: 341-355
- Kish, G. B. 1973. "Stimulus-Seeking and Conservatism." In G.D. Wilson (Ed.) *The Psychology of Conservatism* (pp. 197-207). London: Academic Press
- Kish, G. B. and G. V. Donnenwerth. 1972. "Sex Differences in the Correlates of Stimulus Seeking." *Journal of Consulting and Clinical Psychology* 38: 42-49
- Kluger, A. N., Z. Siegfried, R. P. Ebstein. 2002. "A Meta-Analysis of the Association Between DRD4 Polymorphism and Novelty Seeking." *Molecular Psychiatry*. 7: 712-717.
- Kruglanski, A.W. and D. M. Webster. 1996. "Motivated Closing of the Mind: 'Seizing' and 'Freezing.'" *Psychological Review* 103: 263-283.
- LaHoste, G. J., J.M. Swanson, S. B. Wigal, C. Glabe, T. Wigal, N. King, J. L. Kennedy. 1996. "Dopamine D4 Receptor Gene Polymorphism is Associated with Attention Deficit Hyperactivity Disorder." *Molecular Psychiatry* 1: 121-124.
- Laitin, David D. 1986. *Hegemony And Culture: Politics And Religious Change Among The Yoruba*. Chicago: University Of Chicago Press.
- Langley, K., L. Marshall, M. van den Bree, H. Thomas, M. Owen, M. O'Donovan, A. Thapar. 2004. "Association of the Dopamine D4 Receptor Gene 7-repeat Allele with

- Neuropsychological Test Performance of Children with ADHD.” *American Journal of Psychiatry* 161: 133-138.
- Lau, Richard R. and David P. Redlawsk. 1997. “Voting Correctly.” *American Political Science Review* 91(3): 585-598.
- Leung, P. W. L., C. C. Lee, S. F. Hung, T. P. Ho, C. P. Tang, S. L. Kwong, S. Y. Leung, S. T. Yuen, F. Lieh-Mak, J. Oosterlaan, D. Grady, A. Harxhi, Y. C. Ding, H. C. Chi, P. Flodman, S. Schuck, M. A. Spence, R. Moyzis, J. Swanson. 2005. “Dopamine Receptor D4 (DRD4) Gene in Han Chinese Children with Attention-Deficit/Hyperactivity Disorder (ADHD): Increased Prevalence of the 2-Repeat Allele.” *American Journal of Medical Genetics (Neuropsychiatry Genetics)* 133B: 54-56.
- Lipset, Seymour M. 1983. “Radicalism Or Reformism - The Sources Of Working-Class Politics.” *American Political Science Review* 77 (1): 1-18.
- Liu, I. S. C., P. Seeman, S. Sanyal, C. Ulpian, P. E. B. Rodgers-Johnson, G. R. Serjeant, H. H. M. Van Tol. 1996. “Dopamine D4 Receptor Variant in Africans, D4(valine194glycine), Is Insensitive to Dopamine and Clozapine: Report of a Homozygous Individual.” *American Journal of Medical Genetics* 61: 277-282.
- Lynn, D. E., G. Lubke, M. Yang, J. T. McCracken, J. J. McGough, J. Ishii, S. K. Loo, S. F. Nelson, S. L. Smalley. 2005. “Temperament and Character Profiles and the Dopamine D4 Receptor Gene in ADHD.” *American Journal of Psychiatry* 162: 906-914.
- Maccoby, Eleanor E., Richard E. Matthews, and Anton S. Morton. 1954. “Youth and Political Change.” *The Public Opinion Quarterly* 18(1): 23-39.
- Mackay, T. 2001. “The Genetic Architecture of Quantitative Traits.” *Annual Review of Genetics* 35: 303-339.
- Mannheim, Karl. 1936. *Ideology and Utopia*. London: Routledge.
- Martin N.G., L. J. Eaves, A.C. Heath, Rosemary Jardine, Lynn Feingold and H.J. Eysenck. 1986. “Transmission of Social Attitudes.” *Proceedings of the National Academy of Sciences* 83(12): 4364-68.
- McCarty, Nolan, Keith Poole and Howard Rosenthal. *Political Polarization and Income Inequality*. Princeton University.
- McCracken, J. T., S. L. Smalley, J. J. McGough, L. Crawford, M. Del'Homme, R. M. Cantor, A. Liu, S. F. Nelson. 2000. “Evidence for Linkage of a Tandem duplication Polymorphism Upstream of the Dopamine D4 Receptor Gene (DRD4) with Attention Deficit Hyperactivity Disorder (ADHD).” *Molecular Psychiatry* 5: 531-536.
- Missale, C., R. Nash, S. Robinson, M. Jaber and M. Caron. 1998. “Dopamine Receptors: From Structure to Function.” *Psychological Reviews* 78(1): 189-225.
- Neale, M.C. and L.R. Cardon. 1992. *Methodology for Genetic Studies of Twins and Families*. Dordrecht, The Netherlands: Kluwer.
- Neibrzydowski, L. 1995. “Friendship Among Adolescents.” Paper presented at the biennial meeting of the Society for Research in Child Development, Indianapolis, OH.
- Noble, E.P., T. Z. Ozkaragoz, T. L. Ritchie, X. Zhang, T. R. Belin, R. S. Sparkes. 1998. “D2 and

- D4 Dopamine Receptor Polymorphisms and Personality. *American Journal of Medical Genetics* 81: 257-267.
- North, Douglass C. 1978. "Structure and Performance - Task Of Economic-History" *Journal Of Economic Literature* 16 (3): 963-978.
- Paterson, A. D., G. A. Sunohara, J. L. Kennedy. 1999. "Dopamine D4 Receptor Gene: Novelty or Nonsense?" *Neurophychopharmacology* 21: 3-16.
- Peterson, B. E. and M. D. Lane. 2001. "Implications of Authoritarianism for Young Adulthood: Longitudinal Analysis of College Experiences and Future Goals." *Personality and Social Psychology Bulletin* 27: 678-690.
- Peterson, B.E., K.A. Smirles, and P.A. Wentworth. 1997. "Generativity and Authoritarianism Implications for Personality, Political Involvement, and Parenting." *Journal of Personality and Social Psychology* 72: 1202-1216.
- Plomin, R. 2008. *Behavioral Genetics*. Worth Publishers.
- Pratto, F., J. Sidanius, L. M. Stallworth, and B. F. Malle. 1994. "Social Dominance Orientation: A Personality Variable Predicting Social and Political Attitudes." *Journal of Personality and Social Psychology* 67: 741-763.
- Puttonen, S., N. Ravaja, and L. Keltikangas-Jarvinen. 2005. "Cloninger's Temperament Dimensions and Affective Responses to Different Challenges." *Comprehensive Psychiatry* 46: 128-134.
- Rapoport, Anatol. 1974. *Fights, Games, And Debates*. Ann Arbor: University Of Michigan Press.
- Rowe, D. C., C. Stever, L. N. Giedinghagen, J. M. C. Gard, H. H. Cleveland, S. T. Terris, J. H. Mohr, S. Sherman, A. Abramowitz, I. D. Waldman. 1998. "Dopamine DRD4 Receptor Polymorphism and Attention Deficit Hyperactivity Disorder." *Molecular Psychiatry* 3: 419-426.
- Rubinstein, M., T. J. Phillips, J. R. Bunzow, T. L. Falzone, G. Dziewczapolski, G. Zhang, Y. Fang, J. L. Larson, J. A. McDougall, J. A. Chester, C. Saez, T. A. Pugsley, O. Gershanik, M. J. Low, D. K. Grandy. 1997. "Mice Lacking Dopamine D4 Receptors are Supersensitive to e-Ethanol, Cocaine, and Methamphetamine." *Cell* 90: 991-1001.
- Savitz, J. B. and R. S. Ramesar. "Genetic Variants Implicated in Personality: a Review of the More Promising Candidates." *American Journal of Medical Genetics (Neuropsychiatry Genetics)* 131B: 20-32.
- Scarr, Sandra and Kathleen McCartney. 1983. "How People Make Their Own Environments: A Theory of Genotype # Environment Effects." *Child Development*. 54(2): 424-435
- Schinka, J. A., E. A. Letsch, and F. C. Crawford. 2002. "DRD4 and Novelty Seeking: Results of Meta Analyses." *American Journal of Medical Genetics (Neuropsychiatric Genetics)* 114: 643-648.
- Schmidt, L.A., N.A. Fox, K. H. Rubin, S. Hu, D. H. Hamer. 2002. "Molecular Genetics of Shyness and Aggression in Preschoolers." *Perspectives on Individual Differences* 33: 227-238.

- Schreckhise, William D. and Todd Shields. 2003. "Ideological Realignment in the Contemporary United States Electorate Revisited." *Social Science Quarterly* 84: 596-613.
- Schreiber, Darren. 2005. "Political Cognition as Social Cognition: Are We All Political Sophisticates." Unpublished manuscript.
- Selman, R. L. 1990. "Fostering Intimacy and Autonomy." In W. Damon (ed.) *Child Development Today and Tomorrow* San Francisco: Jossey-Bass.
- Settle, Jaime, Christopher T. Dawes, and James H. Fowler. 2008. "The Heritability of Partisan Attachment." *Political Research Quarterly* (Forthcoming)
- Smalley, S. L.; J. N. Bailey, C. G. Palmer, D. P. Cantwell, J. J. McGough, M. A. Del'Homme, J. R. Asarnow, J. A. Woodward, C. Ramsey, S. F. Nelson. 1998. "Evidence that the Dopamine D4 Receptor is a Susceptibility Gene in Attention Deficit Hyperactivity Disorder." *Molecular Psychiatry* 3: 427-430.
- Staub, D. 1995. "Qualitative Research on School Inclusion: What do we Know? What do we Need to Find?", paper presented at the Annual Conference of the Association for Persons with Severe Handicaps, San Francisco, CA.
- Strobel, A., A. Wehr, A. Michel, B. Brocke. 1999. "Association Between the Dopamine D4 Receptor Exon III Polymorphism and Measures of Novelty Seeking in a German Population." *Molecular Psychiatry* 4: 378-384.
- Swanson, J. M., P. Flodman, J. Kennedy, M. A. Spence, R. Moyzis, S. Schuck, M. Murias, J. Moriarity, C. Barr, M. Smith, M. Posner. 2000. "Dopamine Genes and ADHD." *Neuroscience Behavioral Review* 24: 21-25.
- Swanson, J., J. Oosterlaan, M. Murias, S. Schuck, P. Flodman, M. A. Spence, M. Wasdell, Y. Ding, H.-C. Chi, M. Smith, M. Mann, C. Carlson, J. L. Kennedy, J. A. Sergeant, P. Leung, Y.-P. Zhang, A. Sadeh, C. Chen, C. K. Whalen, K. A. Babb, R. Moyzis, M. I. Posner. 2000. "Attention Deficit/Hyperactivity Disorder Children with a 7-Repeat Allele of the Dopamine Receptor D4 Gene Have Extreme Behavior but Normal Performance on Critical Neuropsychological Tests of attention." *Proceedings of the National Academy of Sciences* 97: 4754-4759.
- Swift, Geraldine. 2000. "Novelty Seeking Traits and D4 Dopamine Receptors." *American Journal of Medical Genetics (Neuropsychiatric Genetics)* 96: 222-223.
- Tesser, Abraham. 1993. "The Importance of Heritability in Psychological Research: The Case of Attitudes." *Psychological Review*. 100(1): 129-142.
- Tomitaka, M., S. Tomitaka, Y. Otuka, K. Kim, H. Matuki, K. Sakamoto, A. Tanaka. 1999. "Association Between Novelty Seeking and Dopamine Receptor D4 (DRD4) Exon III Polymorphism in Japanese Subjects." *American Journal of Medical Genetics (Neuropsychiatric Genetics)* 88: 469-471.
- Tooby, John and Leda Cosmides. 1990. "On the universality of human nature and the uniqueness of the individual: The role of genetics and adaptation." *Journal of Personality* 58:17-67.
- Tooby, J. & Cosmides, L. 1992. *The Psychological Foundations of Culture*. In J. Barkow, L. Cosmides, & J. Tooby (Eds.), *The Adapted Mind: Evolutionary Psychology and the Generation of Culture*. New York: Oxford University Press.

- Treier, Shawn and Sunshine Hillygus. 2005. "The Structure and Meaning of Political Ideology." Working Paper.
- Van Tol, H. H. M., C. M. Wu, H.-C. Guan, K. Ohara, J. R. Bunzow, O. Civelli, J. Kennedy, P. Seeman, H. B. Niznik, V. Jovanovic. 1992. "Multiple Dopamine D4 Receptor Variants in the Human Population." *Nature* 358: 149-152.
- Van Tol, H. H. M., J. R. Bunzow, H. C. Guan, R. K. Sunahara, P. Seeman, H. B. Niznik, O. Civelli. 1991. "Cloning of the Gene for a Human Dopamine D4 Receptor with High Affinity for the Antipsychotic Clozapine." *Nature* 350: 610-614.
- Wang, E., Y.-C. Ding, P. Flodman, J. R. Kidd, K. K. Kidd, D. L. Grady, O. A. Ryder, M. A. Spence, J. M. Swanson, R. K. Moyzis. 2004. "The Genetic Architecture of Selection at the Human dopamine Receptor D4 (DRD4) Gene Locus." *American Journal of Human Genetics* 74: 931-944.
- White, K. M., J.C. Speisman, D. Costos, and A. Smith. 1987. "Relationships Maturity: A Conceptual and Empirical Approach." In J. Meacham (ed.) *International Relations: Family, Peers, Friends*. Basel: Switzerland, Karger.
- Wiesbeck, G. A., C. Mauerer, J. Thome, F. Jakob, J. Boening. 1995. "Neuroendocrine Support for a Relationship Between 'Novelty Seeking' and Dopaminergic Function in Alcohol-Dependent Men." *Psychoneuroendocrinology* 20: 755-761.
- Wilson, G. D. 1973. "A Dynamic Theory of Conservatism." In G. D. Wilson (Ed.), *The Psychology of Conservatism* (pp. 257-265). London: Academic Press.
- Wilson, G. D. and J. R. Patterson. 1968. "A New Conservatism Scale." *British Journal of Social and Clinical Psychology* 7: 264-269.