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UNIVERSITY OF CALIFORNIA, SAN DIEGO

An Examination of Potential Causal Mechanisms Linking Genes and Political Behavior

A dissertation submitted in partial satisfaction of the
requirements for the degree
Doctor of Philosophy

in

Political Science

by

Christopher Dawes

Committee in charge:

Professor James H. Fowler, Chair
Professor Zoltan Hajnal
Professor Trey Ideker
Professor Daniel T. O'Connor
Professor Darren Schreiber

2011

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The dissertation of Christopher Dawes is approved, and it is acceptable in quality and form for publication on microfilm and electronically:

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Chair

University of California, San Diego

2011

DEDICATION

To Sandy (for putting up with a lot).

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PUBLICATIONS

"The Heritability of Duty and Voter Turnout" (with Peter Loewen), *Political Psychology*, Forthcoming.

"Social Preferences and Political Participation" (with Peter Loewen and James Fowler), *Journal of Politics*, Forthcoming.

"Friendships Moderate an Association Between a Dopamine Gene Variant and Political Ideology" (with Jaime Settle, James Fowler, and Nicholas Christakis), *Journal of Politics*, 72 (4): 1189-1198 (October 2010)

"The Behavioral Logic of Collective Action: Partisans Cooperate and Punish More Than Non-Partisans" (with James Fowler, Tim Johnson, Richard McElreath, and Oleg Smirnov), *Political Psychology*, 31 (4): 595-616 (August 2010)

"No Association between Oxytocin Receptor (OXTR) Gene Polymorphisms and Experimentally Elicited Social Preferences" (with Coren Apicella, David Cesarini, Magnus Johannesson, and Paul Lichtenstein, Bjorn Wallace, Jonathan Beauchamp, and Lars Westberg), *PLoS ONE*, 5 (6): e11153 (June 2010)

'Genetic Variation in Preferences for Giving and Risk Taking" (with David Cesarini, Magnus Johannesson, Paul Lichtenstein, and Bjorn Wallace), *Quarterly Journal of Economics*, 124 (2): 809-842 (May 2009)

"Experimental Game Theory and Behavior Genetics" (with David Cesarini, Magnus Johannesson, Paul Lichtenstein, and Bjorn Wallace), *New York Academy of Sciences*, 1167 (1): 66-75 (June 2009)

"The Heritability of Partisan Attachment" (with Jaime Settle and James Fowler), *Political Research Quarterly*, 62 (3): 601-613 (September 2009)

"Partisanship, Voting, and the Dopamine D2 Receptor Gene" (with James Fowler), *Journal of Politics*, 71 (3): 1157-1171 (July 2009)

"The Role of Egalitarian Motives in Altruistic Punishment" (with James Fowler, Tim Johnson, Richard McElreath, and Oleg Smirnov), *Economics Letters*, 102 (3): 192-194 (March 2009)

"Model of Genetic Variation in Human Social Networks" (with James Fowler and Nicholas Chistakis), *Proceedings of the National Academy of Sciences*, 106 (6): 1720-1724 (10 February 2009)

"Two Genes Predict Voter Turnout" (with James Fowler), *Journal of Politics*, 70 (3): 579-594 (July 2008)

"Genetic Variation in Political Participation" (with James Fowler and Laura Baker), *American Political Science Review*, 102 (2):233-248 (May 2008) '

'Heritability of Cooperative Behavior in the Trust Game" (with David Cesarini, James Fowler, Magnus Johannesson, Paul Lichtenstein, and Bjorn Wallace), *Proceedings of the National Academy of Sciences*, 105 (10): 3721-3726 (11 March 2008)

"Egalitarian Motives in Humans" (with James Fowler, Tim Johnson, Richard McElreath, and Oleg Smirnov), *Nature*, 446: 794-796, doi:10.1038/nature05651 (12 April 2007)

ABSTRACT OF THE DISSERTATION

An Examination of Potential Causal Mechanisms Linking Genes and Political Behavior

by

Christopher Dawes

Doctor of Philosophy in Political Science

University of California, San Diego, 2011

Professor James H. Fowler, Chair

Motivated by earlier work studying the genetic basis of political attitudes (Martin, Eaves, Heath, Jardine, Feingold & Eysenck 1986, Alford, Funk & Hibbing 2005), researchers found that political behaviors such as voter turnout (Fowler, Baker & Dawes 2008), broader participation (Fowler, Baker & Dawes 2008), and partisan attachment (Settle, Dawes & Fowler 2009, Hatemi, Hibbing, Alford, Martin & Eaves 2009) also have a significant heritable component. These findings prompted a great deal of discussion and debate and helped spawn the nascent field of "genopolitics". However, in order to push this area of research forward scholars must identify causal mechanisms linking genes to political behaviors. The goal of this dissertation, which is made up of three distinct chapters, is to explore poten-

tial causal pathways my testing potential mediators such as personality traits and cognitive ability as well as identifying new genetic variants that may be associated with political behaviors.

The first chapter presents a twin study testing whether the psychological traits cognitive ability, personal control, and extraversion mediate the relationship between genes and political predisposition and acts of participation. The second chapter is a candidate gene association study that tests the hypothesis that extraversion mediates a relationship between a variant of the COMT gene and partisan attachment. The third chapter presents the results from a genome-wide association study of validated voter turnout in the 2010 election. To complete this work I have utilized four novel genetically informative data sets. Two of these data sets were constructed here at UCSD, one at the University of Minnesota, and another with a research team from Sweden.

All three studies shed new light on the relationship between genes and these political behaviors. However, the mediation results from *Chapter 1* and *Chapter 2* suggest only a modest amount of the relationship between genes and political behaviors are mediated by personality traits and cognitive ability. In addition, the investigation of over 500,000 genetic variants presented in *Chapter 3* did not turn up many new significant associations. However, all three chapters provide a template for how future work in this area should be done as well as highlight the pitfalls in this burgeoning area of research.

Chapter 1

Do Psychological Traits Mediate the Relationship Between Genes and Political Participation?

Introduction

Motivated by earlier research showing a genetic basis for political attitudes (Martin et al. 1986, Alford, Funk & Hibbing 2005), researchers recently discovered that political behaviors like voter turnout and other acts of political participation are also influenced by genetic variation (Fowler, Baker & Dawes 2008). These findings prompted a great deal of discussion and debate, however the authors of that study did not explore possible causal pathways linking genes to political behaviors. This article is an attempt to identify such pathways using a uniquely assembled and comprehensive genetically informative dataset with information on personality, cognitive ability and a wide range of political attitudes, beliefs and behaviors. It represents the first test of the conjecture most recently proposed by scholars studying the relationship between personality traits and political behavior that personality mediates the influence of genes on these behaviors (Mondak, Hibbing, Canache, Seligson & Anderson 2010, Gerber, Huber, Doherty, Dowling & Ha 2010, Blais & Labbe-St-Vincent 2010, Vecchione & Caprara 2009, Gerber, Huber, Doherty & Dowling 2009, Mondak & Halperin 2008, Gerber, Huber, Raso

& Ha 2008, Denny & Doyle 2008, Gallego & Oberski 2011). We focus on three potential mediators identified by this literature: cognitive ability, personal control, and extraversion.

In order to investigate a relationship between genes, psychological traits, and behavior it is necessary to move from a univariate to a bivariate framework of analysis. This allows the decomposition of the covariation between political behaviors and psychological traits into genetic and environmental sources. If a common genetic source underlies both then it suggests these psychological traits serve as mediators. To preview our results, we demonstrate that both acts of participation and related political predispositions share a common genetic origin with psychological traits. Our results, combined with theory, provide support for the argument that psychological traits mediate the effect of genes on political behaviors and predispositions. However, due to the fact that these three psychological traits account for only a modest amount of the variation in these political traits, we only find evidence of partial mediation. Therefore, a majority of the heritable variation in political participation and predispositions is likely mediated by other factors.

Our results attempt to address a fundamental question in political science, namely why some individuals participate in politics whereas others do not. One answer to this question is that genetic variation can ultimately explain a moderate to large share of individual differences in these behaviors. However, this begs the important question of *how* genes influence political behaviors. This article tests potential mechanisms which have been hypothesized in the literature to mediated the relationship between genetic factors and various acts of political participation and demonstrates the utility of combining methods from behavior genetics and personality psychology to better understand heterogeneity in political behavior.

Theoretical and Empirical Evidence

Fowler, Baker & Dawes (2008) first established a link between genes and political participation based on their analysis of acts such as voting, volunteering in a political club or organization, contributing money to a political party or candidate,

contacting a government official regarding political or community issues, running for public office, or attending a political rally or march. The authors found that 60% of the variation in overall participation could be attributed to genetic factors. Recently a group of scholars found similar heritability estimates for measures of non-voting participation in Denmark (Klemmensen, Hobolt, Petersen, Skytthe & Norgaard 2010) and in a new US sample (Klemmensen et al. 2010, Funk, Smith, Alford, Hibbing, Hatemi & Hibbing 2010).

While the fact that individual differences in political participation can be traced to genetic differences is interesting, the influence of genes on modern behaviors such as these is likely indirect via more fundamental traits. Distinguishing between the various mechanisms which might help explain the heritable variation in a trait is an important next step in the "genopolitics" research agenda. Scholars studying the effect of personality on political participation and attitudes have argued that since personality traits are formed before political behaviors, and are known to be heritable (Bouchard & McGue 2003), they likely represent an intermediate link in the causal chain (Mondak et al. 2010).

The idea that personality traits in part shape political behavior can be traced back to *The American Voter* (Campbell, Converse, Miller & Stokes 1960), however a new generation of scholars have begun to rigorously investigate this assertion (Mondak et al. 2010, Gerber, Huber, Doherty, Dowling & Ha 2010, Blais & Labbe-St-Vincent 2010, Vecchione & Caprara 2009, Gerber et al. 2009, Mondak & Halperin 2008, Gerber et al. 2008, Denny & Doyle 2008, Gallego & Oberski 2011). While comprehensive theory linking personality traits to political behavior is being actively developed (Mondak & Halperin 2008, Mondak et al. 2010), scholars have consistently argued that traits related to personal control and a willingness to engage in social interaction are likely to influence political participation (Carmines 1980, Carlson & Hyde 1980, Guyton 1988, Milbrath & Goel 1977, Cohen, Vigoda & Samorly 2001, Mondak & Halperin 2008, Blais & Labbe-St-Vincent 2010, Gerber et al. 2008, Gerber et al. 2009, Mondak et al. 2010, Vecchione & Caprara 2009).

Campbell et al. (1960) originally argued that differences in one's general feeling of mastery over the self and environment probably translate into the politi-

cal process as well. *Competence theory*, developed by Carmines (1980), postulates that personal control promotes political participation. Personal control is the belief that outcomes are the product of one's own efforts. The incentive to engage in any kind of action is predicated on whether or not it can produce a desired outcome. If the outcome is believed to be out of one's control then there is little incentive to undertake an action, especially if it is costly to do so. Competence theory extends this thinking to political engagement. Individuals with high personal control will be motivated to become involved in the political process because their actions will be rewarded with a desired political outcome. Several studies have found empirical support for competence theory (Carmines 1980, Carlson & Hyde 1980, Guyton 1988, Milbrath & Goel 1977). Recent work has also demonstrated an empirical link between self-efficacy, a trait strongly related to personal control (Judge, Erez, Bono & Thoresen 2002, Bono & Judge 2003), and political participation. Blais & Labbe-St-Vincent (2010) reported a positive relationship between a measure of general self-efficacy and two different indicators of turnout. Self-efficacy and personal control are strongly related concepts; self-efficacy is defined as the belief in one's ability to perform across many different contexts and personal control is the belief in one's ability to control one's environment (Bono & Judge 2003). However, self-efficacy is a broader concept tapping both control over the environment and a general belief in one's competence so the two concepts are not equivalent.[1]

Mondak & Halperin (2008) theorize that since social interaction is essential for many political acts, one's general predisposition toward interacting with others should influence one's willingness to participate in politics. This is especially true for acts like attending and speaking at meetings or rallies, signing petitions, and participating in political discussions. In support of this assertion, Mondak et al. (2010) found a significant relationship between the Big Five personality trait extraversion, which is marked by sociability and an eagerness to engage with oth-

[1]The correlation between self-efficacy and personal control is 0.56 (Bono & Judge 2003). The two questions measure Blais & Labbe-St-Vincent (2010) asks whether respondents agree with the statements "I can do just about anything I really set my mind to" and "I have little control over what happens to me", therefore control is an important part of what the authors are capturing.

ers, and contacting an elected official, attending public meetings, and attending election rallies. Vecchione & Caprara (2009) also reported extraversion to be significantly related to overall participation. Finally, Blais & Labbe-St-Vincent (2010) found shyness to be inversely related to the propensity to vote.

Another possible mediator is cognitive ability. There are various mechanisms through which cognitive ability may influence political participation. One plausible hypothesis is that acts of political participation require the mobilization of costly cognitive resources, whose availability varies across individuals. To undertake a political action, an individual is usually required to acquire and then process information (Luskin 1990). If information gathering and processing is cheaper for those with higher cognitive ability, they will be more likely to participate relative to those with lower cognitive ability.

One institution through which individuals acquire information is schooling.However, one can also imagine other information acquisition channels which are relevant for political behavior. For example, individuals with higher cognitive ability may be better at absorbing the political information to which they are exposed in the press and may also endogenously select into environments with more information available. "Preference-based" channels from cognitive ability to political behavior are also plausible. Psychologists and behavioral economists have shown that cognitive ability is associated with high discount rates and other behavioral anomalies (Benjamin, Brown & Shapiro 2006, Kirby, Winston & Santiesteban 2005). On this assessment, political participation is an act which entails paying an immediate cost to enjoy instrumental benefits at some later stage (Fowler & Kam 2006).[2]

Empirical studies have begun to include more direct measures of cognitive ability in empirical models of participation. Verba, Schlozman & Brady (1995) and Nie, Junn & Stehlik-Barry (1996) found a positive relationship between a measure of verbal ability and participation. Hauser (2000) reported a positive relationship between turnout and an interviewer rating of intelligence in the American National

[2]Individuals may enjoy immediate benefits from seeing one's preferred candidate win or warm-glow utility from doing one's civic duty. However, many of the policy-related benefits are not realized until the future.

Election Study, the verbal ability score in the General Social Survey, and Henmon-Nelson Test of Mental Ability from the Wisconsin Longitudinal Study. Denny & Doyle (2008) and Deary, Batty & Gale (2008) also found a strong relationship between measures of cognitive ability and turnout based on two separate samples in Great Britain.[3] Finally, Luskin (1990) found that a measure of political sophistication correlated significantly with an interviewer's rating of the respondent's intelligence, even controlling for education and other background characteristics.

Scholars studying the relationship between personality traits and political participation have also argued that this relationship is likely to be indirect, mediated by political predispositions like political interest, feelings of civic duty (Blais & Labbe-St-Vincent 2010, Gallego & Oberski 2011), a desire to acquire political knowledge, and perceived political efficacy (Guyton 1988, Cohen, Vigoda & Samorly 2001, Mondak et al. 2010, Vecchione & Caprara 2009, Gallego & Oberski 2011). Cohen, Vigoda & Samorly (2001) and Guyton (1988) showed that personal control influences political participation via political efficacy. Gallego & Oberski (2011) found that extraversion influences both turnout and engagement in acts of protest via political efficacy and Vecchione & Caprara (2009) reported that political efficacy fully mediated the effect of extraversion on overall political participation.

There is also empirical evidence that cognitive ability exerts an indirect effect on participation. Verba, Schlozman & Brady (1995) demonstrated that interest in politics, political efficacy, and political information are all positively related to education and when they were included in a regression model of political participation, verbal ability became insignificant suggesting that these variables mediate a relationship between cognitive ability and participation.

On the genetics side, recent work has shown that political efficacy, knowledge, and interest are all heritable based on US and European samples (Klemmensen et al. 2010, Funk et al. 2010). This research suggests that psychological traits like personal control, extraversion, and cognitive ability may mediate the relationship between genes and political predispositions known to be strongly related to polit-

[3]Denny & Doyle (2008) used the 1958 National Child Development Study and Deary, Batty & Gale (2008) used the 1970 British Cohort Study.

ical participation.

In summary, there is strong theoretical and empirical evidence that the three psychological traits cognitive ability, personal control, and extraversion mediate the relationship between genes and acts of political participation as well as political predispositions. Behavior genetics scholars have long known, based on twin studies, that personality traits and cognitive ability are highly heritable (Bouchard & McGue 2003). Recent work done in political science has also established the heritability of political participation and predispositions (Fowler, Baker & Dawes 2008, Klemmensen et al. 2010, Funk et al. 2010). These relationships are illustrated by *Figure 1.1*. While the true causal relationship may go from genes to psychological traits to political predispositions and ultimately to political participation, here we test the second two relationships illustrated in *Figure 1.2*.

Sample and Measures

The Swedish Twin Registry (STR) began in the 1950s and contains nearly all twins born in Sweden between 1886 and 2000 totaling more than 170,000 twins (Lichtenstein, Sullivan, Cnattingius, Gatz, Johansson, Carlstrom, Bjork, Svartengren, Volk, Klareskog, de Faire, Schalling, Palmgren & Pedersen 2006). The STR routinely administers surveys to its members and this article uses data from a recently administered survey called *SALTY*.[4] The SALTY study was initiated in 2007 by a group of researchers in epidemiology, medicine, economics, and political science.

Beginning in early 2009, the survey was sent out to 24,914 Swedish twins born between 1943 and 1958 and the final reminders were sent out in the spring of 2010. The survey generated a total of 11,578 responses. Of these, 11,261 (97.2%) respondents gave informed consent to have their responses stored and analyzed. Subject zygosity was classified either by questionnaire items with high reliability or, when available, by analysis of biosamples (Lichtenstein et al. 2006). In total, our sample is comprised of 2,346 complete same-sex twin pairs. In *Table 1.1*, we

[4]Twins in the Swedish Twin Registry with a date of birth between January 1, 1943 and December 13, 1958 were sent the SALTY survey, provided at least one member of each twin pair had previously participated in a major phone survey administered by the registry.

report a comparison of respondents to non-respondents on a number of background characteristics.[5] As is common in twin research, there is some oversampling of women. Respondents are also better educated and earn more money than non respondents. These differences, however, are quantitatively quite modest, never amounting to more than 0.2 standard deviations.

The SALTY survey is one of the first large-scale efforts to collect detailed questions about political attitudes, predispositions, and behaviors from a genetically informative sample. In this article we focus on political predispositions and acts of participation. The acts are whether respondents reported voting in the 2006 Swedish parliamentary election, voting in the 2009 European Union election, and whether they have performed any of the following in the previous five years in order to express their political opinions: contacted a politician, contacted a public sector official, participated in a protest or demonstration, boycotted a certain good, made a financial contribution, and/or signed a petition. In terms of political predispositions, we asked subjects how interested they were in politics, questions gauging internal and external efficacy, how confident they were in Swedish politicians, how informed they were about economic policy, and whether they viewed the act of voting as a civic duty. The questions about contacting officials, protesting, boycotting, making financial contributions, signing a petition, civic duty, internal efficacy, and external efficacy are taken from the Swedish Citizenship Survey (Petersson, Westholm & Blomberg 1989). The questions about voting in the Swedish and EU parliamentary elections, interest in politics, and confidence in politicians are taken from the Swedish Election Study. Exact question wording is reported in the appendix.

As part of SALTY, we also administered batteries designed to measure personal control and extraversion. In order to measure personal control, we used the Locus of Control Scale (LOC) battery (Rotter 1966).[6] The LOC classifies individuals along a single dimension capturing the degree to which they feel like they

[5]This data is for respondents and non-respondents from same-sex twin pairs.

[6]The Rotter (1966) battery is made up of 13 questions, one of which asked about student testing was not included on the survey due to the age of the sample. Two other questions were explicitly political in nature so we excluded them from our analysis. Therefore, our LOC score is based on 10 questions and thus ranges from 0-10.

control the outcome of events. Individuals with an *internal* locus of control feel they control their own destiny and believe outcomes they realize are the product of their own effort and skills. Those with an *external* locus of control believe that outcomes are outside of their control. A low score on the scale is associated with an internal locus of control and a high score with an external locus of control. External control is also strongly related to neuroticism and internal control with self-efficacy (Judge et al. 2002). We reverse-coded the LOC so that higher scores are associated with higher internal locus of control. To measure extraversion, the survey included the 16 item Adult Measure of Behavioral Inhibition (AMBI) battery (Gladstone & Parker 2005). The AMBI is a subjective measure of general long-standing inhibition designed to capture how an individual responds to social novelty and risk stimuli (Gladstone & Parker 2005). Higher scores on the AMBI reflect a proneness for social avoidance and introversion.[7] We reverse-coded the AMBI so that higher scores are associated with extraversion.

Finally, we used social security numbers to match the men in the SALTY sample to conscription data provided by the Swedish National Service Administration. All men in our sample were required by law to participate in military conscription around the age of 18. They enlisted at a point in time when exemptions from military duty were rare and typically only granted to men who could document a serious handicap that would make it impossible to complete training. Indeed, in our sample, we were able to successfully match 95% male twins to the information in the military archives. For the men born after 1950, approximately half of our sample, the military data has been digitalized. For the remaining twins, we manually retrieved the information from the Military Archives. The first test of cognitive ability used by the Swedish Military was developed in 1943, and it has subsequently been revised and improved on a few occasions. Its basic structure has, however, remained unchanged during the study period considered in this paper. Carlstedt (2000) discusses the history of psychometric testing in the Swedish military and provides evidence that the measure of cognitive ability is a good measure of general intelligence (Spearman 1904). The male SALTY respondents studied

[7]The correlation between AMBI and the Extraversion-Introversion scale from the Eysenck Personality Questionnaire-Revised (EPQ-R) is 0.75 (Gladstone & Parker 2005).

in this paper took four subtests (logical, verbal, spatial and technical) which, for most of the study period, were graded on a scale from 0 to 40. Because there were minor changes to the test during our study period, we do not use the raw scores as our measure of cognitive ability. Instead, we transformed the subjects' test scores to percentile rank, separately by birth year, using a standardization sample of all twins for whom data was available (not just SALTY respondents). Since conscription is not mandatory for females, we only have our measure of cognitive ability for males in the SALTY sample.

Since our measures of political predispositions and participation are all ordinal, we have transformed extraversion and cognitive ability into deciles. *Table 1.2* includes summary statistics for each of these variables broken out by zygosity. We expect personal control, extraversion, and cognitive ability to be positively related to both political predispositions and participation.

Biometric Modeling

The process we follow for analyzing the SALTY data is in two steps. In the first step, we estimate how much of the variation in acts of participation and predispositions can be attributed to genetic and environmental factors. Many of these political variables have never been studied in a genetic framework, and none has been studied in a Swedish sample. In the second step, we estimate the amount of genetic and environmental variation political behaviors and predispositions share with psychological traits. This is achieved by using a bivariate twin model.

Univariate ACE Model

The power of the classic twin design is that it leverages the fact that monozygotic (MZ) twins share 100% of their genes while dizygotic (DZ) twins share on average 50% of their genes. If we are willing to assume that differences in MZ and DZ twins for a particular trait are not due to similarity in the exogenous environmental conditions facing MZ twins, known as the equal environments assumption, we can then compare the similarity for that trait among MZ twin pairs to that of DZ twin pairs in order to get an estimate of the degree to which genes

influence that trait.

More formally, the classical twin model assumes that the variance in observed behavior can be partitioned into additive genetic factors (A), and environmental factors which are shared or common to co-twins (C), and unique environmental factors (E). This is the so-called ACE model.[8] Common environment includes the family environment in which both twins were raised and any other factor to which both twins were equally exposed. In contrast, the unique environment includes influences that are experienced individually. The role of genes and environment are not measured directly but their influence is inferred via their effects on the covariances of twin siblings (Neale & Cardon 1992) described by the model:

$$y_{ij} = aA_{ij} + cC_{ij} + eE_{ij} \tag{1.1}$$

where y is the trait of interest assumed to be expressed as deviations from zero with unit variance, A is the additive genetic component, C is common environment component, and E is the unique environment component. All three components are assumed to be mutually independent and are each distributed as a standard normal. For a given twin pair in family j:

$$cov(y_{1j}, y_{2j}) = a^2 cov(A_{1j}, A_{2j}) + c^2 cov(C_{1j}, C_{2j}) + e^2 cov(E_{1j}, E_{2j}) \tag{1.2}$$

Since MZ twins share all of their genes while DZ share 50% on average, $cov(A_{1j}, A_{2j}) = 1$ for MZ twins and $cov(A_{1j}, A_{2j}) = 0.5$ for DZ twins. Since unique environment is by definition unique to each twin, $cov(E_{1j}, E_{2j}) = 0$. Finally, since both twins in a pair face the same common environment, $cov(C_{1j}, C_{2j}) = 1$. This leaves us with:

$$cov(y_{1j}, y_{2j})_{MZ} = a^2 + c^2 \tag{1.3}$$

$$cov(y_{1j}, y_{2j})_{DZ} = 0.5a^2 + c^2 \tag{1.4}$$

Since A, C, and E are assumed to be independent, the variance for all twins is:

$$var(y_{ij}) = a^2 + c^2 + e^2 \tag{1.5}$$

[8]For a primer of biometric modeling geared for political scientists see Medland & Hatemi (2009).

where a^2 is the heritability, defined as the proportion of total variation accounted for by additive genetic factors, c^2 is the proportion accounted for by common environmental factors, and e^2 the proportion accounted for by unique environmental factors. The variance-covariance matrix for MZ and DZ twin pairs respectively is then:

$$\mathbf{\Omega_{MZ}} = \begin{bmatrix} a^2 + c^2 + e^2 & a^2 + c^2 \\ a^2 + c^2 & a^2 + c^2 + e^2 \end{bmatrix} \tag{1.6}$$

$$\mathbf{\Omega_{DZ}} = \begin{bmatrix} a^2 + c^2 + e^2 & 0.5a^2 + c^2 \\ 0.5a^2 + c^2 & a^2 + c^2 + e^2 \end{bmatrix} \tag{1.7}$$

We note that the ACE model has been the object of criticism (Goldberger 1977) and requires strong functional form and independence assumptions. One way to address these concerns is to study non-twin relatives, thus giving additional moment conditions which can be used to identify richer models, thereby relaxing some of the assumptions which are often critized in the standard behavior genetic decomposition (Loehlin 1965, Goldberger 1977, Goldberger 1979). In political science, this approach was pioneered by Hatemi, Hibbing, Medland, Keller, Alford, Smith, Marks & Eaves (2010), who found that twins-based estimates were robust to the inclusion of other pedigree relationships.

Since the variables we focus on in this study are ordinal, we estimate a threshold model that assumes a continuous latent distribution underlying a given trait for which we observe thresholds demarcating categories (Posthuma, Beem, de Geus, van Baal, von Hjelmborg, Iachine & Boomsma 2003). The prevalence of a particular category c is:

$$\int_{t_{c-1}}^{t_c} \int_{s_{c-1}}^{s_c} \phi(v_1, v_2, \Omega) dv_1 dv_2 \tag{1.8}$$

where v_1 and v_2 are the trait for each member of the twin pair (twin 1 and 2), $\phi(v_1, v_2, \Omega)$ is a bivariate standard normal distribution with correlation matrix Ω_{MZ} or Ω_{DZ} depending on the zygosity of the twin pair.[9] t and s are thresholds for each member of the twin pair such that $t_{c-1}, s_{c-1} = -\infty$ for the lowest category

[9]The diagonal elements of Ω_{MZ} and Ω_{DZ} are normalized to one.

and $t_c, s_c = \infty$ for the highest category. The thresholds are typically constrained to be the same for both members of the twin pair as well as for MZ and DZ twins, both of which we do here. However, we estimate different thresholds for males and females in all of the models. The variance components in a, c, and e as well as the thresholds are estimated via maximum likelihood.

Bivariate ACE Model

In order to estimate how much of the *covariation* between political behaviors and psychological traits theorized as mediators can be attributed to the same genetic source, we must move from a univariate to a bivariate model. Evidence of a common genetic source is consistent with mediation. The bivariate ACE model can be represented as:

$$z_{ij} = a_{11}A^1_{ij} + c_{11}C^1_{ij} + e_{11}E^1_{ij} \tag{1.9}$$

$$y_{ij} = a_{21}A^1_{ij} + c_{21}C^1_{ij} + e_{21}E^1_{ij} + a_{22}A^2_{ij} + c_{22}C^2_{ij} + e_{22}E^2_{ij} \tag{1.10}$$

where z and y are the two traits of interest both assumed to be expressed as deviations from zero with unit variance. Following the same logic as in the univariate model, $cov(A^1_{1j}, A^1_{2j}) = cov(A^2_{1j}, A^2_{2j}) = 1$ for MZ twins and $cov(A^1_{1j}, A^1_{2j}) = cov(A^2_{1j}, A^2_{2j}) = 0.5$ for DZ twins, $cov(C^1_{1j}, C^1_{2j}) = cov(C^2_{1j}, C^2_{2j}) = 1$ and $cov(E^1_{1j}, E^1_{2j}) = cov(E^2_{1j}, E^2_{2j}) = 0$ for both MZ and DZ twins. If we stack z and y for each family such that $\mathbf{Y} = (z_{1j}, y_{1j}, z_{2j}, y_{2j})'$ the variance-covariance matrices become:

$$\mathbf{\Omega_{MZ}} = \begin{bmatrix} \mathbf{A}+\mathbf{C}+\mathbf{E} & \mathbf{A}+\mathbf{C} \\ \mathbf{A}+\mathbf{C} & \mathbf{A}+\mathbf{C}+\mathbf{E} \end{bmatrix} \tag{1.11}$$

$$\mathbf{\Omega_{DZ}} = \begin{bmatrix} \mathbf{A}+\mathbf{C}+\mathbf{E} & 0.5\mathbf{A}+\mathbf{C} \\ 0.5\mathbf{A}+\mathbf{C} & \mathbf{A}+\mathbf{C}+\mathbf{E} \end{bmatrix} \tag{1.12}$$

where:

$$\mathbf{A} = \begin{bmatrix} a^2_{11} & a_{11}a_{21} \\ a_{11}a_{21} & a^2_{21}+a^2_{22} \end{bmatrix} \tag{1.13}$$

with $\mathbf{C}$ and $\mathbf{E}$ analogously defined. This model, known as a Cholesky decomposition model (Martin & Eaves 1977), assumes that the latent factors underlying z

(A^1, C^1, and E^1) also influence y, however the latent factors underlying y (A^2, C^2, and E^2) do not affect z.[10] This relationship is illustrated by the path diagram in *Figure 1.3*.

The Cholesky model, as developed here, assumes a recursive ordering where genes influence a psychological trait and then the psychological trait influences the political variable, subject to a disturbance which has the familiar ACE structure. This recursive structure is of course incorrect if personality is not causally antecedent to political behavior. An alternative approach is to allow each variance component of the first trait to be correlated with the corresponding variance component in the second trait. Loehlin (1996) shows how the parameter estimates from such a model are readily transformed into the parameters from our recursive model since both models span the same model parameter space.[11] In addition, this model assumes that the latent factors underlying the two traits are uncorrelated with one another across and within individuals. These assumptions are necessary in order for the model to be identified and leaves us with nine parameters to be estimated (a_{11},a_{21},a_{22},c_{11},c_{21},c_{22},e_{11},e_{21},e_{22}). The parameters can be estimated using the following moments from the data:

$$cov(y_{1j}, z_{2j})_{MZ} = a_{11}a_{21} + c_{11}c_{21} \tag{1.14}$$

$$cov(z_{1j}, z_{2j})_{MZ} = a_{11}^2 + c_{11}^2 \tag{1.15}$$

$$cov(y_{1j}, y_{2j})_{MZ} = a_{11}^2 + c_{11}^2 + a_{21}^2 + c_{21}^2 \tag{1.16}$$

$$cov(y_{1j}, z_{2j})_{DZ} = 0.5a_{11}a_{21} + c_{11}c_{21} \tag{1.17}$$

$$cov(z_{1j}, z_{2j})_{DZ} = 0.5a_{11}^2 + c_{11}^2 \tag{1.18}$$

$$cov(y_{1j}, y_{2j})_{DZ} = 0.5a_{11}^2 + c_{11}^2 + 0.5a_{21}^2 + c_{21}^2 \tag{1.19}$$

$$var(z_{ij}) = a_{11}^2 + c_{11}^2 + e_{11}^2 \tag{1.20}$$

$$var(y_{ij}) = a_{11}^2 + c_{11}^2 + e_{11}^2 + a_{22}^2 + c_{22}^2 + e_{22}^2 \tag{1.21}$$

[10]The **A** matrix is arrived at by multiplying the lower triangular matrix (a_{11}, a_{21}, a_{22}) with its transpose.

[11]The proof of this is straightforward and consists of merely writing the variance-covariance matrices of the two models in terms of the underlying structural parameters and equating the elements from matrix 1 with the corresponding elements from matrix 2.

$$cov(z_{ij}, y_{ij}) = a_{11}a_{21} + c_{11}c_{21} + e_{11}e_{21} \tag{1.22}$$

where the last three equations are the population variance and covariances. Since both variables in our bivariate models are ordinal, estimation of the thresholds and variance components is achieved following the same approach described in the univariate case.

The parameter estimates generated by this bivariate model can be used to construct quantities of interest. The genetic correlation (r_g) is obtained by dividing the genetic covariance between the two traits by the square root of the product of their genetic variances:[12]

$$r_g = \frac{a_{11}a_{21}}{\sqrt{a_{11}^2(a_{21}^2 + a_{22}^2)}} \tag{1.23}$$

Genetic correlation quantifies the degree to which the genetic endowment of two traits covary. A correlation of 0 means that the two traits are influenced by completely different genes and a correlation of 1 means the same genes influence both traits. However, the genetic correlation is of limited usefulness without incorporating the extent to which the two traits are heritable. For example, the same genes may be influencing two traits, but these genes may not account for very much of the variation in each of the traits. A more meaningful quantity is the the percentage of the *covariation* between two traits that can be explained by additive genetic factors. The total correlation between two traits (r), based on (1.22) and (1.23) is:[13]

$$r = r_g\sqrt{a_{11}^2(a_{21}^2 + a_{22}^2)} + r_c\sqrt{c_{11}^2(c_{21}^2 + c_{22}^2)} + r_e\sqrt{e_{11}^2(e_{21}^2 + e_{22}^2)} \tag{1.24}$$

where a_{11}^2 and $a_{21}^2 + a_{22}^2$ represent the heritabilities of traits z and y respectively.[14] The product of square root of the two univariate heritabilities and the genetic

[12]The correlations for the common environment (r_c) and unique environment (r_e) are analogously defined.

[13]It is clear that (1.22) and (1.24) are equivalent, however (1.22) and (1.23) attempt to demonstrate the intuition behind the derivation of the genetic correlation and bivariate heritability.

[14]Beauchamp, Cesarini, Johannesson, Lindqvist & Apicella (2010) recently derived an extension of the standard bivariate Cholesky model which allows for assortative mating. They prove that in the presence of cross-trait assortative mating at the genetic level, the bivariate ACE model produces estimates of the genetic share of the correlation between two traits which are downward biased. This would suggest that estimates of the genetic correlation reported in this paper are conservative, though other sources of bias may work in the opposite direction.

correlation is known as *bivariate heritability*. Therefore, the percentage of the total correlation accounted for by genetic factors[15] is:

$$\% \text{ Genetic} = \frac{r_g \sqrt{a_{11}^2(a_{21}^2 + a_{22}^2)}}{r} \tag{1.25}$$

Notice that (24) implies that since genetic and environmental correlations range between -1 and 1, the point estimate for the percentage of the total correlation due to genetic factors may be less than 0% or greater than 100%. For clarity of presentation, we report values that fall outside this range at the nearest boundary.

It is important to point out that like any correlation coefficient, the genetic correlation is not in and of itself informative about the direction of causality. For example, it may be the case that genes are influencing z which in turn influences y, however it may also be the case that these genes are influencing z and y independently. The latter relationship, where the same gene affects more than one trait, is known as pleiotropy. These two possible relationships are illustrated by *Appendix Figure 1.1* and *Appendix Figure 1.2*. While both relationships are possible, the theoretical arguments made by Gerber, Huber, Doherty, Dowling & Ha (2010) and Mondak et al. (2010) point to mediation as the more likely scenario. At the very least, our estimates put an upper bound on the proportion of the heritable variation in political traits that *may* be mediated by the psychological traits we study.

Univariate Results

Political Participation

The estimates of heritability, common environment, and unique environment for self-reported vote in the 2006 Swedish parliamentary election and 2009 European Union election are presented in *Table 3*.[16] We also show estimates for other acts of participation including contacting a politician, contacting a public sector official, participating in a protest or demonstration, boycotting a certain good, making a financial contribution, and signing a petition.

[15] The percentage for common environment and unique environment are analogously defined.

[16] All results reported in this paper are based on complete twin pairs.

The heritability estimates for three of the acts are significantly different from zero. In particular, the heritability for voting in the Swedish parliamentary election is close to the estimates reported by Fowler, Baker & Dawes (2008) and Funk et al. (2010), however it should be noted that there is very little variation in our measure since 96% of the sample reported voting.[17] The heritability estimate for the sum of self-reported activities is a bit lower than the participation index reported by Fowler, Baker & Dawes (2008), however it is very close to the estimates reported by (Funk et al. 2010) for a US sample and Klemmensen et al. (2010) based on a Danish sample. While not statistically different from zero in the ACE model, heritability estimates for protesting, participating in a boycott, and making financial contributions to candidates or parties are similar in magnitude. Like voting in the EU election, signing a petition yields a heritability estimate that is small and about the same size as the estimate for common environment. The point estimates for common environment are at or near zero for contacting a politician, contacting an elected official, voting in the Swedish parliamentary, and the overall index and are insignificant for every act of participation, suggesting the common environment plays at best a small role in these outcomes.

Political Predispositions

The univariate results for political interest, internal and external political efficacy, confidence in politicians, level of information regarding economic policy, and civic duty are also presented in *Table 1.3*. The heritability estimates are significantly different from zero and the percentage of the variation accounted for by common environment is insignificant for all six variables. The heritability estimates for most of the variables are moderate in magnitude, however genetic factors account for approximately half of the variation in interest in politics and being informed about economic policy.

Our estimates for interest in politics and political efficacy are similar to those reported by Klemmensen et al. (2010) and Funk et al. (2010) and our estimate

[17] Overall turnout in Sweden was 90% in the age group we study.

for civic duty is similar to Loewen & Dawes (2011).[18] Two of these measures, confidence in politicians and level of information regarding economic policy, have never been studied in a behavior genetic context. These results suggest that genetic variation influences a wide variety of political behaviors and predispositions that are thought to influence political participation.

Bivariate Results

The purpose of the second step of our analysis is to quantify the amount of the covariation between acts of participation and psychological traits that can be attributed to a common genetic source. As a starting point, *Table 1.4* presents the polychoric correlations between each trait we found to be heritable and personal control, extraversion, and cognitive ability. The correlations are relatively moderate (ranging from 0.09 to 0.32 in absolute value) but they are significant, suggesting that each of these psychological characteristics plays a role in explaining several different acts of political engagement.

Based on the results from the univariate model, we restrict the bivariate analysis of personal control and extraversion to acts for which we found a heritability estimate statistically distinguishable from zero (voting in the Swedish parliamentary election, contacting a politician, contacting a public sector official, and the overall index). For cognitive ability, we restrict the analysis to acts for which heritability estimates are statistically distinguishable from zero for men (contacting a politician and contacting a public sector official) since we only have a measure of cognitive ability for males in the sample.[19] Additionally, since the common environment point estimates for these acts of participation and psychological traits are at or close to zero and insignificant in the univariate model, we drop parameter c_{21} from the bivariate model. This means we are assuming that the common environment correlation is zero.[20] However, while dropping insignificant parameters

[18] Klemmensen et al. (2010) did not find civic duty to be significant in a Danish sample.

[19] Heritability estimates for political participation among males are presented in *Appendix Table 1.1*.

[20] Fit statistics, provided in *Appendix Table 1.2*, confirm that this more parsimonious model is appropriate in all cases.

from the model is common practice in behavior genetics, it is not without its critics (Goldberger 2002) because the reported p-values do not reflect the model selection procedure. Therefore, we also present the results for the unrestricted models in the appendix.[21]

Political Participation

The genetic and environmental correlations for the three acts of political participation as well as the overall index are presented in *Table 1.5* and the the percentage of the total correlation due to genetic and environmental factors are presented in *Table 1.6* and are graphically illustrated in *Figure 1.4*. The only insignificant genetic correlation is between turnout and personal control. Of the acts for which the genetic correlations were significant, genetic factors make up between 65% and 97% of the total correlation. These results suggest that for contacting politicians and public sector officials, common genes account for nearly all of the correlation with the three psychological traits. There is also evidence of genetic overlap for the index of overall participation and both personal control and extraversion. The results for turnout are not as strong likely due to the limited variation in the measure.

The fact that we find significant results for contacting politicians and elected officials accords well with existing theory and empirical evidence. Verba, Schlozman & Brady (1995) classify contacting officials as a *skill-based* act because it requires individuals to be organized and write and speak effectively, skills that are correlated with cognitive ability. Our results are also consistent with competence theory. Individuals who believe that they will achieve a desired policy goal by contacting an official will be more likely to do so. A simple example is the belief that a call to a local city council member will get a pot hole filled. Finally, extraverts are more willing to interact with others in general so it is not surprising that they would be willing to initiate contact in a political setting. Also, since they tend

[21]Results for models estimating c_{21} are presented in *Appendix Table 1.3* and *Appendix Table 1.4*. In all cases r_c is insignificant in the full model. The full models produce very similar point estimates to the ones in which c_{21} is dropped but the confidence intervals are wider. One important difference is that the genetic correlations between all three acts and cognitive ability are significant in the model without c_{21} and insignificant in the full model.

to have larger social networks, they are more likely to be persuaded by others to contact elected officials. Mondak et al. (2010) report a strong positive relationship between extraversion and contacting a member of the US congress. What our results add is that the same genes appear to influence both extraversion and contacting behavior, which lends support to the hypothesis that a psychological characteristic mediates the relationship between genes and political participation.

Political Predispositions

The genetic and environmental correlations for all of the political predispositions are also presented in *Table 1.5* and the the percentage of the total correlation due to genetic and environmental factors are presented in *Table 1.6* and are graphically illustrated in *Figure 1.5*. All of the genetic correlations between political predispositions and the psychological traits are significant and genetic factors account for between 49% and 100% of the total correlation. Once again, it appears that most of the relationship can be explained by variation in the same set of genes.

As with political participation, these results are consistent with mediation when combined with existing theoretical and empirical evidence. Previous research has demonstrated that personal control is linked with political efficacy (Guyton 1988, Cohen, Vigoda & Samorly 2001) and civic duty (Blais & Labbe-St-Vincent 2010); extraversion to political efficacy (Vecchione & Caprara 2009, Blais & Labbe-St-Vincent 2010, Mondak & Halperin 2008), interest in politics (Blais & Labbe-St-Vincent 2010) and civic duty (Blais & Labbe-St-Vincent 2010); and cognitive ability to interest in politics (Verba, Schlozman & Brady 1995) and political efficacy (Verba, Schlozman & Brady 1995). There is no direct evidence of a relationship between personal control and interest in politics, however the closely related trait of openness to experience has been shown to be related to political interest (Mondak & Halperin 2008). Therefore, we expected a significant percentage of the covariation between political predispositions and psychological traits to be due to genetic factors since this would be the case if they were in fact mediators. Our results are consistent with these expectations.

Neither knowledge of economic policy nor confidence in politicians have been previously studied in the context of personality traits or cognitive ability, therefore we did not have a clear expectation about what we would find. However, knowledge of economic policy is likely related to political knowledge and confidence in politicians to political trust. Political knowledge has been linked to extraversion (Mondak & Halperin 2008) and cognitive ability (Verba, Schlozman & Brady 1995). Schoon, Cheng, Gale, Batty & Deary (2010) demonstrated a relationship between political trust and cognitive ability. While these earlier findings are suggestive, more work is needed to be sure that the significant genetic overlap we have shown for personality with knowledge of economic policy and confidence in politicians indicates a mediation relationship.

Discussion

The SALTY sample is unique in a number of ways. It is one of the first studies to ask a very large sample of twins an extensive battery of questions about engagement in well-studied political activities as well as predispositions towards politics. Typically, published studies on political behavior have been based on much smaller samples and thus may suffer from imprecise parameter estimates. Our sample also contains measures of psychological traits allowing us to test for the first time the conjecture that genes influence political behaviors indirectly through these traits. And since cognitive ability was measured in adolescence while political variables were measured later in life, it suggests that the causal arrow flows from psychological traits to politics and not the other way around.

Based on our univariate modeling, we find that the total number of self-reported participatory acts is heritable, as are specific acts like contacting a politician or a public sector employee. These heritabilities range from 0.36 to 0.52, similar to what has been found for other political behaviors (Fowler, Baker & Dawes 2008, Klemmensen et al. 2010, Funk et al. 2010). We find mixed results for turnout, however the confidence intervals for voting in the Swedish and EU elections overlap suggesting the two estimates cannot be distinguished from one another statistically. On the other hand, we fail to find significant heritability for

participating in a protest, boycotting, making a financial contribution, or signing a petition. The fact that the total number of acts is heritable, combined with similar magnitudes for the heritability estimates for protesting, boycotting, and making contributions, suggests that genetic variation explains individual differences in overall political participation.

For political predispositions, heritability estimates were significant for political interest, internal and external political efficacy, confidence in politicians, level of information regarding economic policy, and the view that voting is a civic duty. These estimates range from 0.24 to 0.50 in line with other recent studies (Klemmensen et al. 2010, Funk et al. 2010). The magnitudes for these estimates are also quite similar to what we found for acts of participation. Our result that 50% of the variation in political interest can be attributable to genetic factors potentially has implications for the finding by Prior (2010) that people tend to be either interested or uninterested in politics and that this predisposition is exceptionally stable over the life cycle. The determinants of political interest are not currently well known, however the observed relationship may be due in part to genetic variation in the population.

These results are very similar to those based on samples in the US as well as Sweden's neighbor Denmark suggesting that our findings may be informative about the heritability of political participation and predispositions in other modern Western democracies. However, it is important to point out that our heritability estimates, like any other descriptive statistic, are specific to the time and population on which they are based. Therefore, we urge caution when extrapolating to other societies and eras. The relative importance of genes crucially depends on the prevailing cultural and institutional setting.

Our bivariate results for political participation show that genetic factors account for 65% to 88% of the correlation between extraversion and the three acts of participation we examined as well as the overall number of reported acts. In addition, personal control significantly overlaps with contacting a politician and contacting a public sector official, with genetic factors accounting for 93% and 95% of the total correlation. For political predispositions, all of the genetic

correlations are significant and account for between 49% and 100% of the total correlation. These results thus present evidence that most of the relationship between psychological traits and political participation can be explained by *the same set of genes* which, when combined with theoretical arguments previously presented (Gerber, Huber, Doherty, Dowling & Ha 2010, Mondak et al. 2010), is consistent with mediation. In this light, we view the work presented here as an important step in an ambitious research agenda aimed at better understanding the role that variation in genetic endowments for personality, skills, traits and preferences plays in generating heritable variation in political behavior. However, it is important to emphasize that our results suggest only partial mediation.

Overall, genes account for the lion's share of the overlap between the psychological traits and political behaviors we focus on, but the size of this overlap is relatively modest. *Figure 1.6* illustrates this by putting the percentages presented in *Figure 1.4* and *Figure 1.5* in terms of the correlations between psychological and political traits. The overall height of each bar is the total correlation, the dark portion is the genetic component, and the light portion is environmental component based on *Equation 24* (where $r_c = 0$). It is clear that most of the correlation is due to shared genetic factors, however the height of the bars implies a modest amount of the variation in the political traits overlaps with the psychological traits. For example, nearly all of the correlation between cognitive ability and contacting a politician can be attributed to genetic factors. However, since the total correlation is 0.25 it implies that the influence of extraversion can only account for 6% of the variation in contacting a politician. If we take our results literally, 46% of the variation in contacting a politician explained by genetic factors is not mediated by cognitive ability. Therefore, while our results support the assertion that the effects of psychological traits on political behavior signal the mediated influence of genes (Mondak et al. 2010), future work must identify and test other mediators.

The goal of this article has been to move from simply demonstrating that genes matter for political behaviors towards a better understanding of *why* they matter. In order to do that in the context of a twin study we used bivariate modeling techniques to estimate the extent to which genetic factors can explain the

covariation between political traits and more basic psychological traits like personality and cognitive ability. There are limitations to this approach. First, as with a standard regression model, our results have not established a causal relationship going from genes to psychological traits to political participation (or predispositions). Our results are *consistent* with a mediation hypothesis, but it is important to remember that the relationship may be spurious. Like any other empirical investigation, we must rely on extant theory to distinguish between the two. Second, the causal pathway from genes to political behaviors and predispositions is much more complex and likely involves multiple mediators. In particular, psychological traits are believed to influence behavior through political predispositions suggesting a trivariate model from genes to psychological traits to political predispositions to political behavior. Here we have limited our analysis to bivariate relationships, but in future work we will examine more complicated pathways. Though we have applied standard methodology, it is well known that the assumptions needed to identify a bivariate model are quite strong when only twin data is available, and we suggest that future work use extended samples incorporating other sibling types and pedigrees to evaluate some of the moment restrictions assumptions in the model.

The results presented here nevertheless provide qualified support for the argument that psychological traits, since they are highly heritable, *mediate* some of the effect of genes on political behaviors and predispositions. This study has demonstrated that the set of genes related to certain acts of political participation significantly overlaps with the set related to personal control, extraversion, and cognitive ability. The same is true for political predispositions. One interpretation of this finding is that genes shape these psychological traits which then have a downstream effect on political predispositions and behaviors. However, this finding is also consistent with the hypothesis that the same genes influence both traits independently. To adjudicate these competing alternatives, we must rely on the theoretical argument made by scholars studying the relationship between personality and political behavior. Competence theory postulates that personal control promotes political participation (Carmines 1980). Individuals with high

personal control will be motivated to become involved in the political process because their actions will be rewarded with a desired outcome. Also, extraversion is clearly linked to political behavior since social interaction is necessary for many participatory political acts (Mondak & Halperin 2008).

Finally, a genome-wide association study (GWAS) is another approach to identifying and testing potential mediators. Instead of using a latent genetic model based on MZ and DZ twins, GWAS collect genetic samples from individuals and analyze hundreds of thousands of small sequences of DNA called single-nucleotide polymorphisms (SNPs) located across the entire human genome. Knowing the function of SNPs found to be associated with political traits holds the promise of suggesting presently unknown biological, psychological, and even social causal pathways that contribute to political participation.

Tables

Table 1.1: Sample background statistics. Income is defined as the sum of income earned from wage labor, income from own business, pension income and unemployment compensation. Capital income is not included. The education variable produced by Statistics Sweden is categorical (with seven categories ranging from middle school to PhD). The categorical scores are converted to years of education. Age is the respondent's age on January 1, 2010. A survey respondent is defined here as an individual who responded to the survey and gave informed consent to have his or responses analyzed. Non-respondents are all other individuals in the SALTY cohort.

	Respondents			Non-Respondents		
	Mean	S.D.	N	Mean	S.D.	N
Birthyear	1949.7	4.58	7197	1950.5	4.60	7816
1 if Female	0.55	0.50	7197	0.49	0.50	7816
1 if MZ	0.42	0.49	7197	0.38	0.49	7816
Education	11.95	2.74	7172	11.42	2.64	7774
Income	295191	191971	7192	274757	212828	7811
1 if married	0.65	0.48	7195	0.59	0.49	7815

Table 1.2: Summary Statistics for Psychological and Political Traits in Monozygotic (MZ) and Dizygotic (DZ) Twins. Note: cognitive ability measure is only available for males.

	MZ Mean	MZ SD	DZ Mean	DZ SD
Vote (Parliament)	0.96	0.21	0.96	0.21
Voted (EU)	0.63	0.48	0.63	0.48
Contacted a Politician	0.09	0.29	0.10	0.30
Contacted a Public Sector Official	0.10	0.30	0.11	0.31
Protest	0.06	0.24	0.07	0.26
Boycott	0.13	0.34	0.17	0.38
Financial Contributions	0.04	0.20	0.07	0.25
Signed a Petition	0.37	0.48	0.42	0.49
Participation Index (8 items)	2.37	1.35	2.50	1.41
Not Interested In Politics	0.10	0.29	0.09	0.29
Not Very Interested	0.46	0.50	0.44	0.50
Fairly Interested	0.38	0.49	0.40	0.49
Very Interested	0.07	0.25	0.07	0.25
Little Confidence in Politicians	0.13	0.34	0.13	0.34
Fairly Little Confidence	0.45	0.50	0.46	0.50
Fairly Large Confidence	0.40	0.49	0.40	0.49
Very Confident	0.02	0.13	0.01	0.12
Very Uninformed About Econ Policy	0.07	0.25	0.08	0.27
Somewhat Uninformed	0.11	0.31	0.11	0.32
Neither Informed nor Uninformed	0.53	0.50	0.52	0.50
Well Informed	0.27	0.44	0.25	0.44
Very Well Informed	0.03	0.17	0.04	0.19
Internal Efficacy	4.37	1.91	4.26	1.97
External Efficacy	5.35	1.83	5.34	1.84
Voting is a Civic Duty	7.46	2.51	7.52	2.51
Personal Control	5.77	1.93	5.71	1.90
Extraversion	4.69	1.74	4.52	1.74
Cognitive Ability	4.74	1.46	4.57	1.50

Table 1.3: Heritability Estimates for Psychological and Political Traits. Parameter estimates and 95% confidence intervals in brackets are shown for a univariate ACE model that allows different thresholds for males and females. Significant heritability estimates are bolded. These results suggest that several acts of political participation and political predispositions are influenced by genetic variation. Note: cognitive ability measure is only available for males.

	Heritability	Common Environment	Unique Environment
Vote (Parliament)	**0.41**	0.00	0.59
	[0.03, 0.58]	[0.00, 0.29]	[0.42, 0.79]
Vote (EU)	0.17	0.19	0.64
	[0.00, 0.43]	[0.00, 0.36]	[0.55, 0.73]
Contact Politician	**0.52**	0.00	0.48
	[0.31, 0.63]	[0.00, 0.15]	[0.37, 0.61]
Contact Official	**0.41**	0.00	0.59
	[0.15, 0.53]	[0.00, 0.20]	[0.47, 0.72]
Attend Protest	0.35	0.11	0.55
	[0.00, 0.61]	[0.00, 0.44]	[0.39, 0.73]
Boycott Participation	0.29	0.08	0.63
	[0.00, 0.49]	[0.00, 0.33]	[0.51, 0.76]
Financial Contributions	0.44	0.04	0.52
	[0.00, 0.65]	[0.00, 0.41]	[0.35, 0.74]
Sign a Petition	0.15	0.16	0.69
	[0.00, 0.39]	[0.00, 0.32]	[0.60, 0.78]
Participation Index (0-8)	**0.36**	0.05	0.59
	[0.20, 0.46]	[0.00, 0.18]	[0.54, 0.64]
Interest in Politics	**0.50**	0.06	0.44
	[0.35, 0.61]	[0.00, 0.19]	[0.39, 0.49]
Internal Efficacy	**0.36**	0.00	0.64
	[0.23, 0.41]	[0.00, 0.09]	[0.59, 0.70]
External Efficacy	**0.24**	0.10	0.66
	[0.08, 0.39]	[0.00, 0.22]	[0.61, 0.72]
Civic Duty	**0.29**	0.05	0.67
	[0.12, 0.39]	[0.00, 0.18]	[0.61, 0.73]
Informed about Econ. Policy	**0.45**	0.00	0.55
	[0.36, 0.51]	[0.00, 0.07]	[0.49, 0.60]
Confidence in Politicians	**0.37**	0.06	0.57
	[0.19, 0.49]	[0.00, 0.20]	[0.51, 0.63]
Personal Control	**0.27**	0.00	0.73
	[0.13, 0.32]	[0.00, 0.11]	[0.68, 0.79]
Extraversion	**0.45**	0.00	0.55
	[0.38, 0.49]	[0.00, 0.05]	[0.51, 0.60]
Cognitive Ability	**0.67**	0.12	0.21
	[0.51, 0.81]	[0.00, 0.26]	[0.18, 0.25]

Table 1.4: Relationship Between Political Traits and Psychological Traits. Correlation coefficients and standard errors in brackets are shown. Note: cognitive ability measure is only available for males.

	Personal Control	Extraversion	Cognitive Ability
Vote (Parliament)	0.20	0.18	0.19
	[0.04]	[0.03]	[0.06]
Contact Politician	0.18	0.22	0.25
	[0.03]	[0.03]	[0.04]
Contact Official	0.18	0.22	0.18
	[0.03]	[0.03]	[0.04]
Participation Index (0-8)	0.17	0.19	0.24
	[0.02]	[0.02]	[0.02]
Interest in Politics	0.26	0.22	0.20
	[0.02]	[0.01]	[0.02]
Internal Efficacy	0.30	0.27	0.28
	[0.02]	[0.01]	[0.02]
External Efficacy	0.28	0.22	0.32
	[0.02]	[0.02]	[0.02]
Voting is a Civic Duty	0.14	0.14	0.09
	[0.02]	[0.02]	[0.02]
Informed about Econ. Policy	0.23	0.20	0.26
	[0.02]	[0.02]	[0.02]
Confidence in Politicians	0.29	0.17	0.24
	[0.02]	[0.02]	[0.03]

Table 1.5: Genetic and Environmental Correlation and 95% CIs from a Bivariate Cholesky ACE models ($r_c = 0$) of Personal Control, Extraversion, and Cognitive Ability with Political Traits. Significant genetic correlation estimates are bolded. Note: cognitive ability measure is only available for males.

	Personal Control		Extraversion		Cognitive Ability	
	r_g	r_e	r_g	r_e	r_g	r_e
Vote (Parl)	0.12	0.18	**0.28**	0.11		
	[-0.18, 1.00]	[0.02, 0.34]	**[0.08, 1.00]**	[-0.03, 0.26]		
Contact Pol	**0.39**	0.01	**0.40**	0.05	**0.39**	0.02
	[0.16, 0.84]	[-0.10, 0.12]	**[0.26, 0.57]**	[-0.07, 0.17]	**[0.24, 0.56]**	[-0.18, 0.23]
Contact Off	**0.39**	0.01	**0.42**	0.09	**0.27**	0.07
	[0.16, 0.84]	[-0.10, 0.12]	**[0.25, 1.00]**	[-0.02, 0.20]	**[0.10, 0.52]**	[-0.13, 0.27]
Index	**0.31**	0.03	**0.39**	0.07		
	[0.15, 0.57]	[-0.04, 0.09]	**[0.28, 0.57]**	[0.01, 0.13]		
Interest	**0.28**	0.12	**0.26**	0.19	**0.30**	0.00
	[0.15, 0.46]	[0.05, 0.19]	**[0.18, 0.36]**	[0.12, 0.26]	**[0.20, 0.41]**	[-0.12, 0.12]
Int Efficacy	**0.49**	0.10	**0.43**	0.16	**0.49**	-0.02
	[0.35, 0.74]	[0.04, 0.16]	**[0.34, 0.56]**	[0.11, 0.22]	**[0.46, 0.77]**	[-0.12, 0.09]
Ext Efficacy	**0.62**	0.08	**0.41**	0.11	**0.65**	-0.05
	[0.41, 1.00]	[0.02, 0.14]	**[0.30, 0.63]**	[0.05, 0.17]	**[0.52, 0.90]**	[-0.16, 0.05]
Duty	**0.21**	0.09	**0.31**	0.05	**0.19**	0.04
	[0.04, 0.45]	[0.02, 0.15]	**[0.19, 0.53]**	[-0.01, 0.11]	**[0.05, 1.00]**	[-0.06, 0.14]
Informed	**0.38**	0.04	**0.30**	0.12	**0.45**	-0.07
	[0.25, 0.58]	[-0.03, 0.11]	**[0.20, 0.39]**	[0.05, 0.18]	**[0.34, 0.58]**	[-0.18, 0.05]
Confidence	**0.41**	0.13	**0.32**	0.06	**0.44**	0.01
	[0.25, 0.69]	[0.06, 0.20]	**[0.21, 0.49]**	[0.00, 0.13]	**[0.31, 0.74]**	[-0.10, 0.13]

Table 1.6: Percentage of total correlation due to Genetic and Unique Environmental factors and 95% CIs. Note: cognitive ability measure is only available for males.

	Personal Control		Extraversion		Cognitive Ability	
	% Genetic	% Unique Environment	% Genetic	% Unique Environment	% Genetic	% Unique Environment
Vote (Parl)	0.26	0.74	0.65	0.35		
	[-0.43, 0.88]	[0.12, 1.43]	[0.21, 1.11]	[-0.11, 0.79]		
Contact Pol	0.93	0.07	0.88	0.12	0.97	0.03
	[0.30, 1.95]	[-0.95, 0.70]	[0.60, 1.19]	[-0.19, 0.40]	[0.73, 1.24]	[-0.24, 0.27]
Contact Off	0.95	0.05	0.76	0.24	0.87	0.13
	[0.41, 1.79]	[-0.79, 0.59]	[0.49, 1.05]	[-0.05, 0.51]	[0.45, 1.29]	[-0.29, 0.55]
Index	0.84	0.16	0.79	0.21		
	[0.47, 1.27]	[-0.27, 0.53]	[0.61, 0.97]	[0.03, 0.39]		
Interest	0.58	0.42	0.58	0.42	1.01	-0.01
	[0.34, 0.81]	[0.19, 0.66]	[0.42, 0.73]	[0.27, 0.58]	[0.82, 1.20]	[-0.20, 0.18]
Int Efficacy	0.69	0.31	0.64	0.36	1.03	-0.03
	[0.51, 0.88]	[0.12, 0.49]	[0.50, 0.76]	[0.24, 0.50]	[0.88, 1.18]	[-0.18, 0.12]
Ext Efficacy	0.73	0.27	0.70	0.30	1.06	-0.06
	[0.54, 0.93]	[0.07, 0.46]	[0.55, 0.86]	[0.14, 0.45]	[0.94, 1.19]	[-0.19, 0.06]
Duty	0.49	0.51	0.78	0.22	0.85	0.15
	[0.12, 0.85]	[0.15, 0.88]	[0.53, 1.04]	[0.00, 0.47]	[0.37, 1.29]	[-0.29, 0.63]
Informed	0.85	0.15	0.68	0.32	1.08	-0.08
	[0.58, 1.14]	[-0.14, 0.42]	[0.49, 0.85]	[0.15, 0.51]	[0.94, 1.23]	[-0.23, 0.06]
Confidence	0.60	0.40	0.79	0.21	0.98	0.02
	[0.39, 0.81]	[0.19, 0.61]	[0.57, 1.02]	[0.00, 0.43]	[0.82, 1.15]	[-0.15, 0.18]

Figures

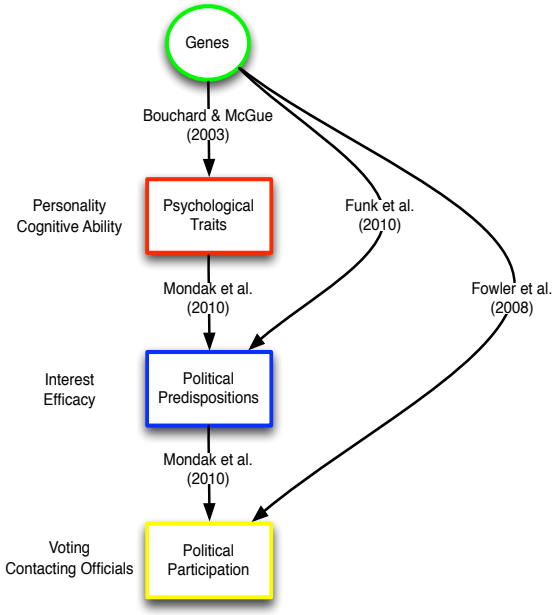


Figure 1.1: Demonstrated relationships (and representative citations) between genes, psychological traits, political traits, and acts of political participation. Lines in bold represent mediation relationship between psychological traits, political predispositions, and political participation.

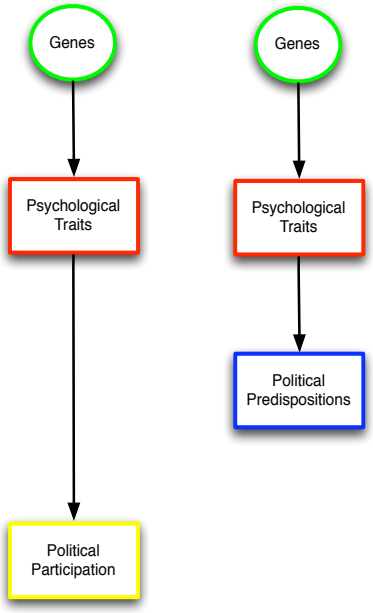


Figure 1.2: Two sub-components of the proposed causal pathway tested in this article.

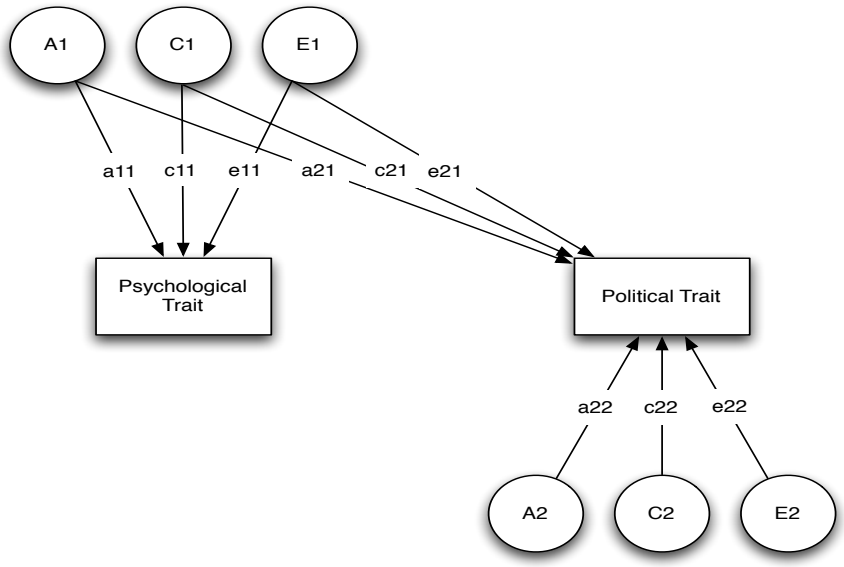


Figure 1.3: Bivariate Cholesky decomposition model. The circles represent latent traits and squares represent measured traits. The latent factors underlying psychological traits (A^1, C^1, and E^1) are assumed to also influence political traits, however the latent factors underlying political traits (A^2, C^2, and E^2) do not affect psychological traits.

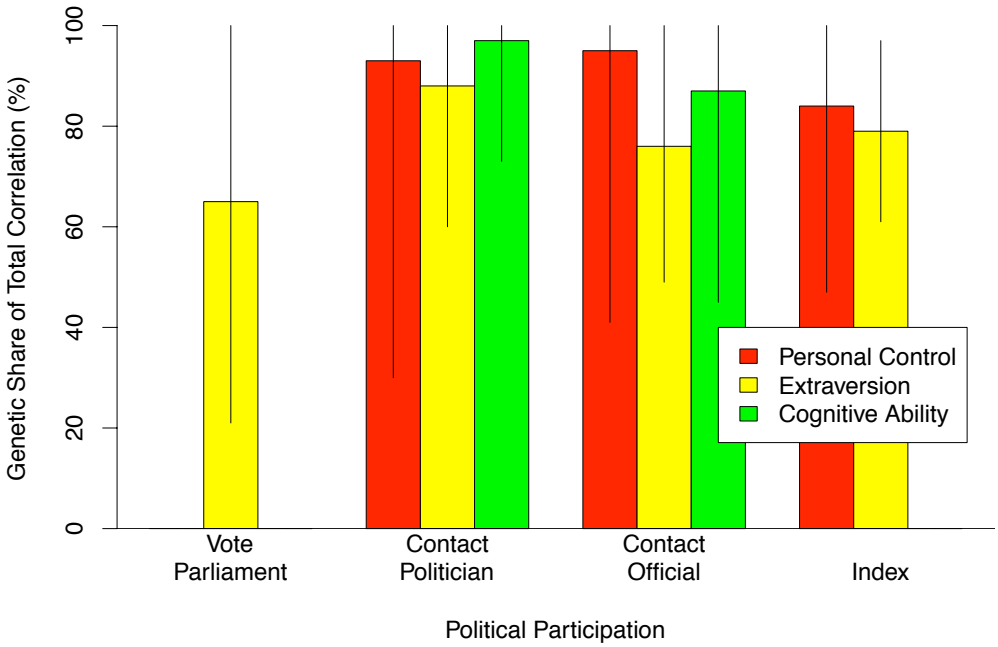


Figure 1.4: Percentage of the total correlation attributable to genetic factors from the model in which $r_c = 0$ are presented along with 95% confidence intervals. Note: the cognitive ability measure is only available for males.

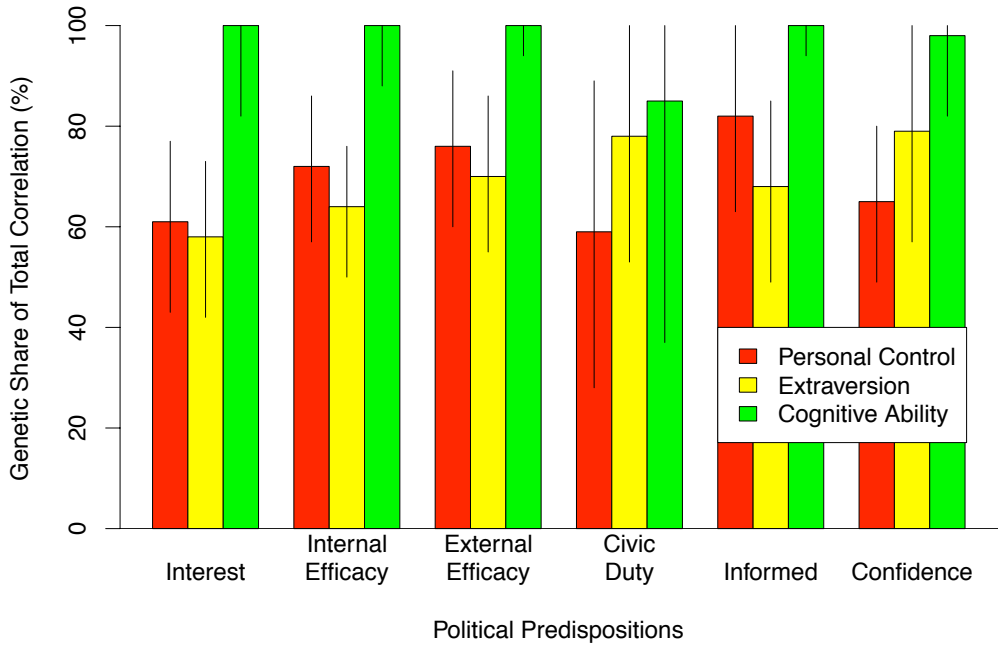


Figure 1.5: Percentage of the total correlation attributable to genetic factors from the model in which $r_c = 0$ are presented along with 95% confidence intervals. Note: the cognitive ability measure is only available for males.

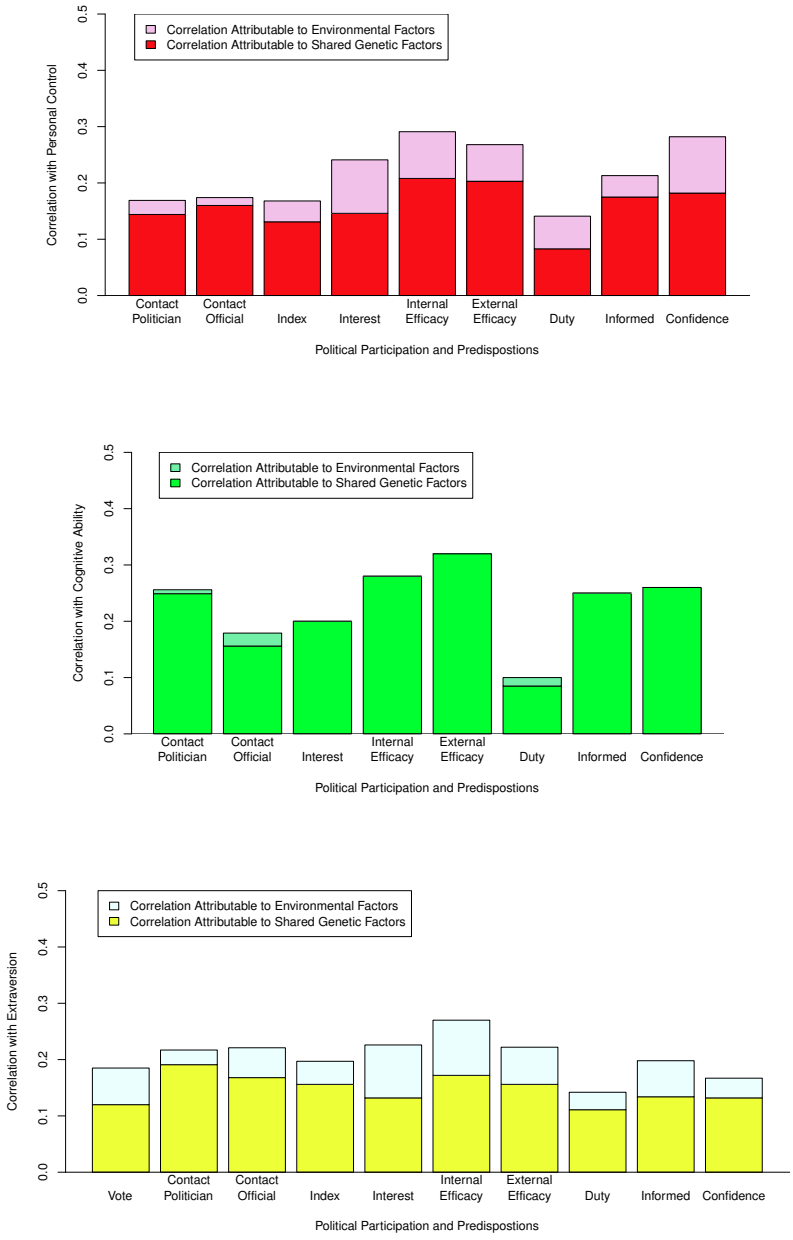


Figure 1.6: Correlations between psychological and political traits. The overall height of the bar is the total correlation, the dark portion is the genetic component, and the light portion is environmental component based on *Equation 24* (where $r_c = 0$).

Appendix

Appendix Table 1.1: Parameter estimates and 95% confidence intervals from a univariate ACE model for the combined sample as well as males and females estimated separately. The hypothesis that male and female estimates are different can be rejected for all models suggesting the combined model is appropriate.

	Combined			Males			Females		
	Heritability	Common Env	Unique Env	Heritability	Common Env	Unique Env	Heritability	Common Env	Unique Env
Vote (Parl)	0.41	0.00	0.59	0.28	0.00	0.72	0.46	0.00	0.54
	[0.03, 0.58]	[0.00, 0.29]	[0.42, 0.79]	[0.00, 0.60]	[0.00, 0.42]	[0.40, 1.00]	[0.00, 0.65]	[0.00, 0.39]	[0.35, 0.77]
Vote (EU)	0.17	0.19	0.64	0.07	0.33	0.60	0.25	0.09	0.66
	[0.00, 0.43]	[0.00, 0.36]	[0.55, 0.73]	[0.00, 0.46]	[0.02, 0.47]	[0.47, 0.72]	[0.00, 0.45]	[0.00, 0.33]	[0.55, 0.78]
Politician	0.52	0.00	0.48	0.56	0.00	0.44	0.46	0.00	0.54
	[0.31, 0.63]	[0.00, 0.15]	[0.37, 0.61]	[0.26, 0.70]	[0.00, 0.24]	[0.30, 0.61]	[0.07, 0.63]	[0.00, 0.29]	[0.37, 0.73]
Official	0.41	0.00	0.59	0.49	0.00	0.51	0.34	0.00	0.66
	[0.15, 0.53]	[0.00, 0.20]	[0.47, 0.72]	[0.13, 0.65]	[0.00, 0.27]	[0.36, 0.70]	[0.00, 0.51]	[0.00, 0.35]	[0.49, 0.85]
Protest	0.35	0.11	0.55	0.13	0.06	0.80	0.47	0.11	0.42
	[0.00, 0.61]	[0.00, 0.44]	[0.39, 0.73]	[0.00, 0.48]	[0.00, 0.38]	[0.52, 1.00]	[0.00, 0.74]	[0.00, 0.52]	[0.26, 0.63]
Boycott	0.29	0.08	0.63	0.09	0.19	0.73	0.41	0.02	0.57
	[0.00, 0.49]	[0.00, 0.33]	[0.51, 0.76]	[0.00, 0.48]	[0.00, 0.39]	[0.52, 0.90]	[0.00, 0.56]	[0.00, 0.33]	[0.44, 0.74]
Contribute	0.44	0.04	0.52	0.00	0.39	0.61	0.51	0.00	0.49
	[0.00, 0.65]	[0.00, 0.41]	[0.35, 0.74]	[0.00, 0.63]	[0.00, 0.56]	[0.36, 0.81]	[0.03, 0.72]	[0.00, 0.31]	[0.28, 0.76]
Petition	0.15	0.16	0.69	0.25	0.09	0.66	0.09	0.20	0.71
	[0.00, 0.39]	[0.00, 0.32]	[0.60, 0.78]	[0.00, 0.47]	[0.00, 0.35]	[0.53, 0.81]	[0.00, 0.40]	[0.00, 0.35]	[0.59, 0.81]
Index	0.36	0.05	0.59	0.21	0.17	0.61	0.42	0.00	0.58
	[0.20, 0.46]	[0.00, 0.18]	[0.54, 0.64]	[0.00, 0.45]	[0.00, 0.36]	[0.53, 0.70]	[0.25, 0.48]	[0.00, 0.14]	[0.52, 0.65]

Appendix Table 1.2: Parameter estimates and 95% confidence intervals from a univariate ACE model for the combined sample as well as males and females estimated separately. The hypothesis that male and female estimates are different can be rejected for all models suggesting the combined model is appropriate.

	Combined			Males			Females		
	Heritability	Common Env	Unique Env	Heritability	Common Env	Unique Env	Heritability	Common Env	Unique Env
Interest	0.50	0.06	0.44	0.59	0.00	0.41	0.42	0.12	0.46
	[0.35, 0.61]	[0.00, 0.19]	[0.39, 0.49]	[0.38, 0.66]	[0.00, 0.17]	[0.35, 0.49]	[0.22, 0.60]	[0.00, 0.28]	[0.40, 0.53]
Int Efficacy	0.36	0.00	0.64	0.34	0.00	0.66	0.37	0.00	0.63
	[0.23, 0.41]	[0.00, 0.09]	[0.59, 0.70]	[0.15, 0.42]	[0.00, 0.14]	[0.58, 0.74]	[0.18, 0.43]	[0.00, 0.15]	[0.57, 0.70]
Ext Efficacy	0.24	0.10	0.66	0.33	0.05	0.62	0.18	0.13	0.69
	[0.08, 0.39]	[0.00, 0.22]	[0.61, 0.72]	[0.09, 0.46]	[0.00, 0.23]	[0.54, 0.71]	[0.00, 0.37]	[0.00, 0.29]	[0.62, 0.76]
Duty	0.29	0.05	0.67	0.30	0.02	0.69	0.28	0.07	0.66
	[0.12, 0.39]	[0.00, 0.18]	[0.61, 0.73]	[0.04, 0.40]	[0.00, 0.22]	[0.60, 0.78]	[0.06, 0.42]	[0.00, 0.24]	[0.58, 0.73]
Informed	0.45	0.00	0.55	0.54	0.00	0.46	0.39	0.00	0.61
	[0.36, 0.51]	[0.00, 0.07]	[0.49, 0.60]	[0.30, 0.61]	[0.00, 0.20]	[0.39, 0.55]	[0.28, 0.46]	[0.00, 0.07]	[0.54, 0.69]
Confidence	0.37	0.06	0.57	0.45	0.02	0.53	0.32	0.09	0.60
	[0.19, 0.49]	[0.00, 0.20]	[0.51, 0.63]	[0.18, 0.55]	[0.00, 0.25]	[0.45, 0.62]	[0.09, 0.48]	[0.00, 0.27]	[0.52, 0.68]

Appendix Table 1.3: $-2\times$Log Likelihood for the full model and model in which c_{21} is dropped. The p-value is associated with a hypothesis test of the difference in likelihoods (χ^2 with one degree of freedom). Note: cognitive ability measure is only available for males.

	Personal Control			Extraversion			Cognitive Ability		
	ACE	$r_c = 0$	P-val	ACE	$r_c = 0$	P-val	ACE	$r_c = 0$	P-val
Vote (Parl)	14808.23	14808.23	0.96	16968.40	16968.40	1.00			
Contact Pol	15763.92	15763.91	0.92	17820.14	17820.14	1.00	6788.55	6787.36	0.27
Contact Off	15910.08	15910.08	1.00	18022.60	18022.60	1.00	6824.26	6822.98	0.26
Index	23814.21	23814.21	0.93	27322.98	27322.75	0.63			
Interest	20952.90	20952.83	0.81	24145.52	24145.50	0.89	9386.06	9383.77	0.13
Int Efficacy	26359.23	26359.23	1.00	30226.63	30226.63	1.00	11944.17	11941.01	0.08
Ext Efficacy	25946.88	25946.76	0.73	29983.60	29983.44	0.69	11751.45	11749.65	0.18
Duty	26922.29	26921.15	0.29	31294.32	31294.31	0.92	12346.00	12345.57	0.51
Informed	21528.56	21528.56	0.95	24903.89	24903.89	1.00	9639.90	9638.76	0.29
Confidence	20640.61	20640.49	0.73	23916.18	23916.04	0.70	9197.76	9197.46	0.58

Appendix Table 1.4: Genetic and Environmental Correlation and 95% CIs from a Bivariate Cholesky ACE models of Personal Control, Extraversion, and Cognitive Ability with Political Traits. Significant genetic correlation estimates are bolded. Note: cognitive ability measure is only available for males.

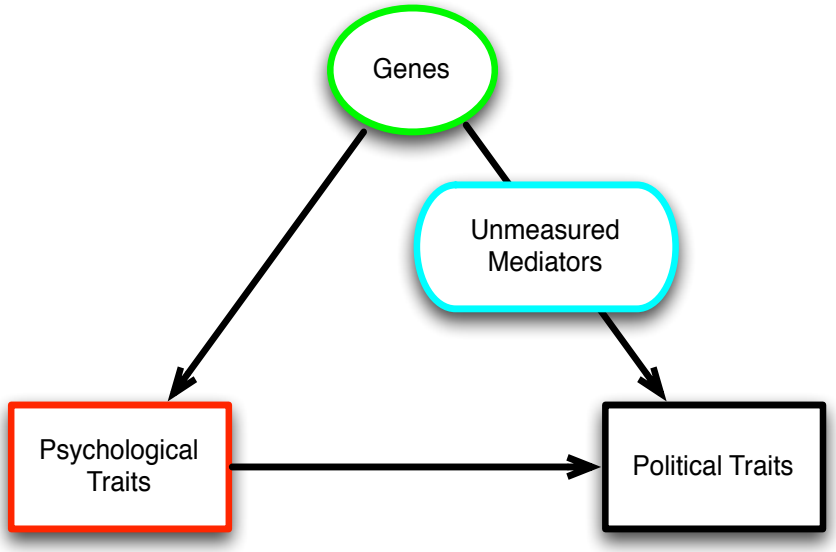
	Personal Control			Extraversion			Cognitive Ability		
	r_g	r_c	r_e	r_g	r_c	r_e	r_g	r_c	r_e
Vote	0.12	-0.72	0.18	**0.28**	-0.43	0.11			
	[-0.39, 1.00]	[-1.00, 1.00]	[0.02, 0.34]	**[0.04, 1.00]**	[-1.00, 1.00]	[-0.03, 0.26]			
Pol	**0.29**	-0.99	0.01	**0.40**	-0.53	0.05	0.28	1.00	0.05
	[-0.06, 1.00]	[-1.00, 0.99]	[-0.12, 0.14]	**[0.25, 0.61]**	[-1.00, 1.00]	[-0.07, 0.17]	[-0.02, 0.54]	[-1.00, 1.00]	[-0.16, 0.26]
Official	**0.39**	-0.21	0.01	**0.42**	0.87	0.09	0.12	1.00	0.10
	[0.01, 1.00]	[-1.00, 1.00]	[-0.10, 0.12]	**[0.23, 1.00]**	[-1.00, 1.00]	[-0.02, 0.20]	[-0.41, 0.45]	[-1.00, 1.00]	[-0.11, 0.30]
Index	0.30	0.46	0.03	**0.36**	1.00	0.08			
	[-0.11, 0.78]	[-0.32, 1.00]	[-0.04, 0.10]	**[0.17, 0.59]**	[-1.00, 1.00]	[0.01, 0.14]			
Interest	0.25	0.85	0.13	**0.26**	0.95	0.19	**0.19**	1.00	0.02
	[-0.07, 0.58]	[-1.00, 1.00]	[0.05, 0.20]	**[0.12, 0.40]**	[-1.00, 1.00]	[0.12, 0.26]	**[0.01, 0.37]**	[-1.00, 1.00]	[-0.10, 0.14]
Internal	**0.49**	-1.00	0.10	**0.43**	-0.14	0.16	**0.49**	1.00	0.00
	[0.29, 0.91]	[-1.00, 0.93]	[0.03, 0.16]	**[0.30, 0.58]**	[-1.00, 1.00]	[0.11, 0.22]	**[0.27, 0.68]**	[-1.00, 1.00]	[-0.11, 0.11]
External	**0.68**	-0.69	0.08	**0.39**	0.99	0.11	**0.56**	1.00	-0.04
	[0.30, 1.00]	[-1.00, 1.00]	[0.01, 0.14]	**[0.20, 0.64]**	[-1.00, 1.00]	[0.05, 0.17]	**[0.39, 0.81]**	[-1.00, 1.00]	[-0.14, 0.07]
Duty	**0.41**	-1.00	0.07	**0.31**	-0.26	0.05	0.29	-0.78	0.03
	[0.02, 1.00]	[-1.00, 1.00]	[0.01, 0.14]	**[0.11, 0.66]**	[-1.00, 1.00]	[-0.01, 0.11]	[-0.04, 1.00]	[-1.00, 1.00]	[-0.08, 0.14]
Inform	**0.38**	0.92	0.04	**0.30**	0.64	0.12	**0.37**	1.00	-0.05
	[0.11, 0.62]	[-0.85, 1.00]	[-0.03, 0.11]	**[0.15, 0.39]**	[-1.00, 1.00]	[0.05, 0.18]	**[0.20, 0.56]**	[-1.00, 1.00]	[-0.17, 0.07]
Conf	**0.45**	-1.00	0.13	**0.34**	-0.84	0.06	**0.37**	0.62	0.02
	[0.13, 0.96]	[-1.00, 1.00]	[0.05, 0.20]	**[0.18, 0.66]**	[-1.00, 1.00]	[-0.01, 0.13]	**[0.12, 0.73]**	[-1.00, 1.00]	[-0.10, 0.15]

Appendix Table 1.5: Percentage of total correlation due to Genetic, Common Environmental, and Unique Environmental factors and 95% CIs. Note: cognitive ability measure is only available for males.

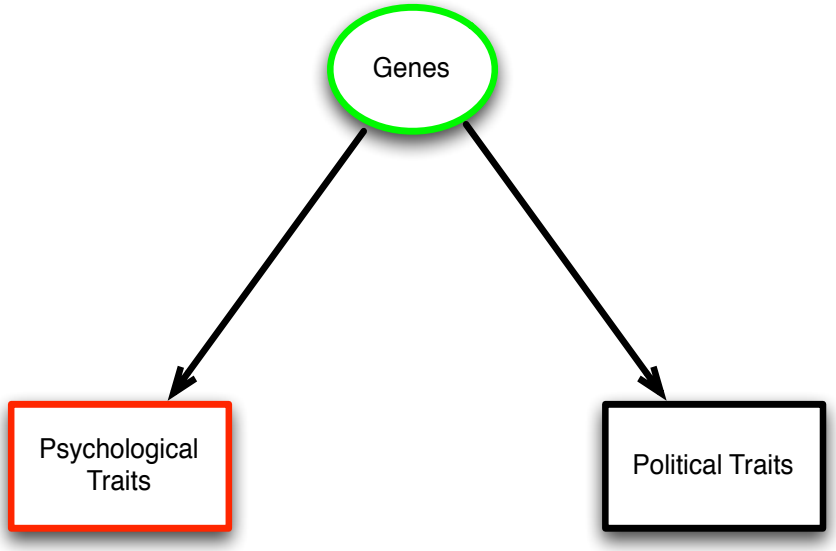
	Personal Control			*Extraversion*			*Cognitive Ability*		
	% Genetic	% Common Env	% Unique Env	% Genetic	% Common Env	% Unique Env	% Genetic	% Common Env	% Unique Env
Vote	0.26	0.00	0.74	0.65	0.00	0.35			
	[-0.64, 1.38]	[-0.81, 0.58]	[0.12, 1.43]	[0.09, 1.26]	[-0.39, 0.36]	[-0.11, 0.79]			
Pol	0.99	-0.05	0.06	0.88	0.00	0.12	0.65	0.29	0.06
	[-0.17, 2.93]	[-1.32, 0.87]	[-0.99, 0.70]	[0.52, 1.22]	[-0.17, 0.24]	[-0.19, 0.40]	[-0.03, 1.28]	[-0.22, 0.87]	[-0.21, 0.31]
Official	0.95	0.00	0.05	0.76	0.00	0.24	0.34	0.46	0.18
	[0.02, 2.03]	[-0.63, 0.72]	[-0.79, 0.59]	[0.35, 1.14]	[-0.24, 0.29]	[-0.05, 0.51]	[-0.85, 1.35]	[-0.34, 1.44]	[-0.23, 0.63]
Index	0.82	0.02	0.16	0.71	0.07	0.22			
	[-0.25, 1.86]	[-0.75, 0.88]	[-0.30, 0.57]	[0.29, 1.08]	[-0.20, 0.40]	[0.04, 0.41]			
Interest	0.51	0.07	0.43	0.58	0.01	0.42	0.58	0.40	0.03
	[-0.12, 1.05]	[-0.35, 0.56]	[0.19, 0.68]	[0.24, 0.81]	[-0.16, 0.27]	[0.27, 0.58]	[0.01, 1.17]	[-0.12, 0.89]	[-0.16, 0.21]
Internal	0.69	0.00	0.31	0.64	0.00	0.36	0.70	0.30	0.00
	[0.34, 1.05]	[-0.25, 0.27]	[0.11, 0.49]	[0.39, 0.79]	[-0.10, 0.19]	[0.24, 0.50]	[0.31, 1.09]	[-0.02, 0.63]	[-0.15, 0.15]
External	0.80	-0.06	0.26	0.65	0.05	0.30	0.83	0.22	-0.04
	[0.31, 1.23]	[-0.37, 0.33]	[0.04, 0.46]	[0.27, 0.95]	[-0.17, 0.35]	[0.14, 0.47]	[0.47, 1.19]	[-0.09, 0.52]	[-0.17, 0.08]
Duty	0.89	-0.33	0.45	0.80	-0.01	0.22	1.34	-0.44	0.11
	[0.04, 1.78]	[-1.01, 0.32]	[0.05, 0.84]	[0.27, 1.36]	[-0.43, 0.39]	[-0.05, 0.48]	[-0.22, 3.21]	[-2.08, 0.90]	[-0.41, 0.60]
Inform	0.85	0.00	0.15	0.68	0.00	0.32	0.85	0.21	-0.06
	[0.19, 1.23]	[-0.24, 0.52]	[-0.14, 0.43]	[0.30, 0.85]	[-0.07, 0.29]	[0.15, 0.51]	[0.42, 1.31]	[-0.17, 0.58]	[-0.21, 0.08]
Conf	0.66	-0.05	0.39	0.85	-0.06	0.20	0.81	0.16	0.03
	[0.16, 1.13]	[-0.40, 0.35]	[0.17, 0.60]	[0.42, 1.34]	[-0.42, 0.27]	[-0.03, 0.43]	[0.23, 1.43]	[-0.38, 0.66]	[-0.14, 0.20]

Appendix Table 1.6: Bootstrapped correlations and 95% confidence intervals for MZ and DZ twins, the difference between the two correlations, a P-value associated with a (one-sided) test of the hypothesis that the MZ correlation is higher than the DZ correlation, and the number of complete same-sex MZ and DZ pairs with non-missing data for each variable. Note: cognitive ability measure is only available for males.

	MZ	DZ	Diff	P-val	NMZ	NDZ
Vote (Parliament)	0.44	0.00	0.44	0.01	1087	1173
	[0.21, 0.62]	[-0.92, 0.29]				
Vote (EU)	0.37	0.28	0.09	0.07	1059	1143
	[0.29, 0.46]	[0.18, 0.37]				
Contact Politician	0.56	0.14	0.42	0.00	1083	1169
	[0.43, 0.68]	[-0.03, 0.29]				
Contact Official	0.44	0.13	0.30	0.00	1083	1169
	[0.30, 0.56]	[-0.04, 0.28]				
Attend Protest	0.43	0.28	0.15	0.11	1083	1169
	[0.24, 0.59]	[0.08, 0.43]				
Boycott	0.35	0.24	0.11	0.09	1083	1169
	[0.23, 0.46]	[0.12, 0.35]				
Contribute	0.43	0.26	0.16	0.12	1083	1169
	[0.22, 0.61]	[0.07, 0.42]				
Petition	0.30	0.24	0.07	0.14	1083	1169
	[0.21, 0.39]	[0.15, 0.32]				
Index	0.41	0.23	0.17	0.00	1018	1101
	[0.35, 0.46]	[0.17, 0.30]				
Interest	0.58	0.35	0.24	0.00	1112	1195
	[0.53, 0.63]	[0.28, 0.41]				
Int Efficacy	0.36	0.18	0.18	0.00	1075	1145
	[0.31, 0.42]	[0.12, 0.24]				
Ext Efficacy	0.35	0.24	0.11	0.02	1080	1159
	[0.29, 0.41]	[0.18, 0.30]				
Duty	0.33	0.20	0.13	0.00	1088	1169
	[0.27, 0.39]	[0.13, 0.27]				
Informed	0.51	0.28	0.23	0.00	1103	1187
	[0.46, 0.57]	[0.21, 0.34]				
Confidence	0.45	0.25	0.19	0.00	1112	1190
	[0.38, 0.51]	[0.19, 0.32]				
Personal Control	0.29	0.13	0.16	0.00	839	866
	[0.21, 0.36]	[0.06, 0.20]				
Extraversion	0.47	0.18	0.29	0.00	990	1051
	[0.41, 0.52]	[0.12, 0.24]				
Cognitive Ability	0.78	0.46	0.33	0.00	420	448
	[0.74, 0.82]	[0.37, 0.54]				



Appendix Figure 1.1: Mediation relationship between latent genetic factors, psychological traits, and political traits. The illustration also reflects the fact that our measures likely do not account for all of relationship between genes and political traits and therefore there are other unmeasured mediators.



Appendix Figure 1.2: Pleiotropic relationship between latent genetic factors, psychological traits, and political traits. In this case genetic factors are influencing psychological and political traits separately.

Participation

During the last five years, have you done any of the following to express your political opinions? It is fine to tick several response alternatives.
- Contacted a politician personally, in writing or through some other means?
- Contacted a public sector official?
- Participated in a protest action or demonstration?
- Participated in a boycott, for example a boycott of a certain good?
- Made financial contributions?
- Signed a petition?
- None of the above

When did you decide for which party to vote for in the last parliamentary election? Voting in the Swedish parliamentary election is based choosing a time versus the response "I did not vote in the election to the parliament".
Which party did you vote for in the last election to the EU parliament? Voting in the EU election is based on choosing a party versus the response "No party".

Predispositions and Attitudes

To what extent do you believe that people who are entitled to vote have a civic duty to cast their ballots in elections? Please indicate your response on the scale below. [1=No duty, 10=Very strong duty]

Are people like you more or less able than others to make politicians take your demands into consideration? Please indicate your response on the scale below. [1=Much less, 10=Much more]

Is your ability to understand what goes on in politics better or worse than that of other people? [1=Much worse, 10=Much better]

Generally speaking, how interested are you in politics? [Very interested / Not very interested / Not at all interested]

Generally speaking, how much confidence do you have in Swedish politicians? [Very large confidence / Fairly large confidence / Fairly little confidence / Very little confidence]

How informed would you say that you are about economic policy? [Very well informed / Well informed / Neither well informed or uninformed / Somewhat informed / Very informed]

Chapter 2

COMT, Extraversion, and Partisan Attachment

Introduction

A burgeoning literature focuses on the relationship between individual differences in personality traits and political behaviors (Gallego & Oberski 2011, Gerber, Huber, Doherty & Dowling 2010, Gerber, Huber, Doherty, Dowling & Ha 2010, Mondak et al. 2010, Gerber et al. 2008, Gerber et al. 2009, Mondak & Halperin 2008, Denny & Doyle 2008). A recent paper, Gerber, Huber, Doherty & Dowling (2010) demonstrated that the personality trait extraversion is a strong predictor of whether an individual affiliates with a major political party as well as the strength of that affiliation. The authors found that a two standard deviation increase in extraversion is associated with a 6 percentage point increase in the likelihood of affiliating with a major party.

Both personality traits and partisan attachment have a genetic basis. Behavior geneticists have long known that personality traits are heritable (Bouchard & McGue 2003) and an association has been established between variants of several individual genes and personality traits (Luo, Kranzler, Zuo, Zhang, Wang & Gelernter 2007, Luo, Kranzler, Zuo, Wang & Gelernter 2007, Luo, Zuo, Kranzler, Zhang, Wang & Gelernter 2008, Stein, Schork & Gelernter 2004, Stein, Fallin, Schork & Gelernter 2005, Reuter & Hennig 2005). Likewise, the strength of par-

tisan attachment has been shown to be heritable (Settle, Dawes & Fowler 2009, Hatemi et al. 2009) and a variant of the D2 dopamine receptor gene has been found to be associated with partisan attachment (Dawes & Fowler 2009).

While previous research in the nascent field of "genopolitics" has demonstrated that genes matter for political behaviors and attitudes (Alford, Funk & Hibbing 2005, Fowler, Baker & Dawes 2008), this article represents a step forward in that it attempts to answer *why* genes matter. The conjecture suggested by scholars studying the link between personality and political behavior is that genes have an indirect effect on political behaviors via their role in shaping personality traits (Mondak et al. 2010, Mondak & Halperin 2008). More specifically, personality traits *mediate* the relationship between genes and political behavior.[1] This conjecture seems plausible since the heritability of both personality traits and political behaviors are relatively large,[2] and the formation of personality traits are believed to predate the development of political attitudes and behaviors (Gerber et al. 2009). *However, this conjecture remains untested.* Two additional possibilities are that the same gene or set of genes influence personality traits and political behaviors independently rather than sequentially or that different genes altogether affect each respective trait. A test of these alternate hypotheses requires a genetically informative sample that also includes measures of personality traits and political behavior.

We examine a variant of the COMT gene, Val158Met, that has been found to be associated with extraversion (Stein et al. 2005, Reuter & Hennig 2005) and test the hypothesis that extraversion mediates the relationship between Val158Met and partisan attachment. To preview our results, we find a significant association between the COMT gene and partisan attachment and are able to replicate this result based on an independent data set. Further, this association is partially mediated by the personality trait extraversion. We find that approximately 8%

[1]This notion is represented by the quote from Mondak et al. (2010) "Because personality is substantially rooted in biology, any effects of personality on political behavior likely signal the mediated influence of biology" (p.90).

[2]Heritability estimates for personality traits range from 41% to 61% (Bouchard & McGue 2003) and estimate for the strength of partisan attachment (Settle, Dawes & Fowler 2009) is 53%.

of the observed association with partisan attachment is via extraversion. This represents the first formal test of a pathway between genes and political behavior and suggests an empirical framework for future work. Given the fact that a single gene likely affects a complex behavior like partisan attachment through a number of pathways (Munafo 2006), finding significant evidence of mediation is promising for future research. Also, since complex behaviors are influenced by many genes (Plomin 2008), this approach suggests variants related to Val158Met should also be investigated.

Genetic Polymorphisms

Genes are distinct regions of human DNA that form the blueprint for molecules that regulate the development and function of the human body. DNA is made up of subunits called nucleotides. There are four such nucleotides: adenine (A), cytosine (C), thymine (T), and guanine (G), named based on the nitrogenous base that they contain. Some genes come in different versions, known as *alleles*. A *polymorphism* exists when there is more than one type of allele (at a locus) that exists in the population. When individuals differ in terms of a single nucleotide pair, it is known as a single nucleotide polymorphism (SNP). For example, a portion of DNA from one individual may be C**C**TA and from another be C**T**TA. In this example, the SNP (in bold) has two alleles, C and T. An illustration of this is provided by *Figure 2.1*.

Most genes specify the composition of proteins (Hartl 2000) which are the basic building blocks for bodily structure and function. Polymorphisms are important to study because they may *change* the protein being produced or *regulate* protein production by turning genes on and off.[3] Complex behaviors, including political behaviors, are likely *polygenic*, meaning they are influenced by multiple genes (Mackay 2001, Plomin 2008). Moreover, phenotypes are typically shaped by a multitude of environmental forces. As a result, an association study examin-

[3]An example of the former is sickle-cell anemia. A SNP in the beta-globin gene comes in two versions, A and T. The T allele codes for the amino acid valine rather than glutamate which leads to protein chains that form crystals under low oxygen concentration. This causes red blood cells to collapse into sickle shapes (Hartl 2000).

ing a single gene is an oversimplification and may only capture a relatively small proportion of the observed variation in political behavior.

Personality, Neurotransmission, and COMT

The prevailing model of personality, the so-called Five Factor Model or Big Five, holds that generally speaking, personality can be characterized by five dimensions: extraversion, agreeableness, conscientiousness, neuroticism, and openness to experience. *Extraversion* is marked by sociability including friendliness towards others and a desire to be in their company (McCrae & Costa 1992). Based on their functional roles, specific neurotransmitter systems are theorized to play a prominent role in determining individual differences in personality traits (Revelle 2005, Depue 1995, Depue & Collins 1999).

Neurons are nerve cells that are responsible for sending, receiving, and processing information. In order for this information to be sent from one neuron to another, signals must cross a small gap called a synapse that exists between the axon of a sending neuron and dendrite of the target neuron. Neurotransmitters, released by the axon of the sending neuron cross the synaptic gap and bind with receptors on the dendrite of the postsynaptic (receiving) neuron, triggering changes in the postsynaptic neuron's metabolic activity. Neurotransmitters are reabsorbed into the presynaptic neuron via a transporter as part of a process called reuptake. Once the neurotransmitter is back inside the neuron it is degraded by enzymes so that its components can be reabsorbed into the cell. Reuptake is necessary to ensure proper neurotransmitter levels in the synapse.[4] Scholars seeking to understand behavior focus on genes involved in neurotransmission. These genes influence transcription, RNA processing, and translation as well as receptor density and binding, reuptake, and degradation of neurotransmitters.

Dopamine is responsible for the control of emotion and positive reinforcement and therefore the dopamine system is believed to influence approach-oriented

[4]Reuptake inhibitors are prescribed to inhibit the reuptake process. For example, selective serotonin reuptake inhibitors (SSRIs) like the antidepressant Prozac prevent the reuptake of serotonin and therefore increase the level of serotonin in the synapse. This allows for increased receptor binding and signaling resulting in improved mood.

behaviors like extraversion (Munafo, Clark, Moore, Payne & Flint 2003, Carver & Miller 2006, Depue 1995). The COMT gene codes for an enzyme called Catechol-O-Methyltransferase that degrades the neurotransmitter dopamine primarily in the prefrontal cortex (PFC), a region of the brain that is responsible for executive function like working memory. Val158Met is a functional SNP that results in a nucleotide substitution from G to A that changes the amino acid produced from valine to methionine. Like most SNPs Val158Met has two alleles: G (Val) and A (Met). The Val allele has been found to produce higher enzymatic activity: individuals with two Val alleles have been found to have 40% higher COMT enzyme activity in the brain of those with two Met alleles (Chen, Lipska, Hamlin, Ma, Matsumoto, Melhem, Kolachana, Hyde, Herman, Apud, Egan, Kleinman & Weinberger 2004). Therefore, the Met allele is associated with less effective breakdown of dopamine translating into higher levels of dopamine in the PFC relative to the Val allele.

A large body of empirical evidence has shown the Met allele to be associated with vulnerability to stress and anxiety (Goldman, Oroszi & Ducci 2005). The precise relationship between dopamine levels in the PFC and anxiety is not completely understood, however Bilder, Volavka, Lachman & Grace (2004) suggest that COMT's role in regulating the balance between the average level of dopamine maintained over long time horizons, tonic dopamine, and the amount of dopamine released in quick bursts, phasic dopamine, is crucial. The Met allele is known to increase tonic dopamine levels and reduces phasic levels in the PFC. Phasic release is in response to environmental stimuli and allows working memory to be updated with this new information. Therefore, muted phasic dopamine release results in suboptimal updating of working memory and thus the information stored in working memory may no longer reflect the present environment. If what is stored in working memory is based on stimuli originally perceived to be threatening, and thus prompting anxiety, this anxiety may persist even though a threat no longer does. The connect between anxiety and extraversion is that individuals suffering from anxiety had been shown to exhibit low extraversion (Bienvenu & Stein 2003).

Samples

This study employs three distinct samples. The first, referred to as the *primary sample*, consists of undergraduate psychology students at San Diego State University recruited between the fall of 1999 and the spring of 2007. As part of the study, subjects completed either the full NEO-Personality Inventory-Revised (NEO-PI-R) (McCrae & Costa 1992) or the shorter NEO Five Factor Inventory (NEO-FFI) (McCrae & Costa 2004). These are the two widely used measures of the Big Five personality traits. See Stein, Schork & Gelernter (2004) for a detailed description of this sample. The second sample, referred to as the *California replication*, is comprised of nuclear families including identical and non-identical twins, siblings, and their parents. A majority of these subjects were residing in the Southern California area at the time of their recruitment. See Zhang, Rao, Wessel, Kennedy, Rana, Taupenot, Lillie, Cockburn, Schork, Ziegler & O'Connor (2004) for a detailed description of this sample. The third sample, referred to as the *Sweden replication*, comes from the SALT study which is made up of Swedish twins born between 1936 and 1958. As part of the SALTY survey described in *Chapter 1*, subjects were asked whether or not they considered themselves as partisans.[5] See Lichtenstein, de Faire, Floderus, Svartengren, Svedberg & Pedersen (2002) for a description of the SALT sample.

In order to determine whether subjects in the primary and California replication samples were attached to a party, they were matched with public voting records available through the California Secretary of State's office.[6] California voting records identify whether registered voters wish to be affiliated with a political party. The matching process for the primary sample utilized available personal information including name, date of birth, phone number, and email address. The matching process for the California replication sample included home address in-

[5]The question asked "Many people perceive themselves as strong followers of a specific political party. But there are also a lot of people without such an attitude towards any of the parties. Do you usually consider yourself as for instance a folkpartist, socialdemokrat, moderat, centerpartist, vnsterpartist, miljpartist or kristdemokrat? Or do you have any such attitudes towards any of the parties?" The possible answers were "Yes, strongly convinced follower", "Yes, but not a very strongly convinced follower", or "No, not a follower of any party". An answer of yes was coded as 1 and no was coded as 0.

[6]Voter records from June 2009 were used.

formation. We were able to match 74% of the subjects from the primary sample and 75% from the replication sample, consistent with the fact that 75% of eligible Californians were registered to vote as of the November 2008 election.[7]

In the primary sample there are 510 subjects and for CA and Sweden replication samples respectively, 460 and 3783. The dependent variable for the US samples is whether a registered voter is affiliated with a major political party, defined as being registered as either a Democrat or a Republican. Summary statistics for the sample are presented in *Table 2.1*.

Population Stratification

A genetic association study indicates whether an allele is found more frequently than can be attributed to chance in a group exhibiting a particular trait than those without the trait. In our case, we seek to identify if the frequency of the Met allele is lower among those attached to a party than those not attached. A significant association can mean that the allele itself influences partisan attachment or that the observed association is a false positive signal due to what is known as *population stratification*. Population stratification occurs when groups have different allele frequencies due to their genetic ancestry. Political behavior in these groups may be affected by their environments, alleles other than the one of interest, or some unobserved factor. Once these two groups mix in a larger population, simply comparing the frequency of the Met allele to the the number of partisans would lead to a spurious association.

Unfortunately, subpopulations cannot be directly observed. However, there are several techniques regularly utilized in the literature to attempt to ameliorate the problem of population stratification. One approach is the structured association method (Pritchard, Stephens & Donnelly 2000). Structured association is a clustering technique that assumes a sample is made up of a mixture of individuals drawn from K subpopulations where K is likely unknown. Subpopulations are identified based on allele frequencies of polymorphisms spread across the genome,

[7]This information comes from the California Secretary of State (http://www.sos.ca.gov/elections/sov/historical-voter-reg/hist-voter-reg-and-part-general-elections-1910-2009.pdf).

grouping individuals who are genetically similar to one another. Individuals may be assigned to more than one subpopulation if their genetic information suggests that they are admixed. Subpopulation assignment may be based on a panel of several genetic markers that have been chosen based on their informativeness and then tests of association can then be conditional on subpopulation assignment.

Genome-wide association studies (GWAS), which tag a large number of genetic markers from across the genome, tend to take one of two main approaches to accounting for population stratification.[8] They either use the principal components (PCA) method developed by Price, Patterson, Plenge, Weinblatt, Shadick & Reich (2006) or a multidimensional scaling (MDS) method (Purcell, Neale, Todd-Brown, Thomas, Ferreira, Bender, Maller, Sklar, de Bakker, Daly & Sham 2007). The PCA method is based on the genetic correlation among individuals whereas the MDS method detects latent dimensions that best explain the observed genetic distance between individuals (Wang, Sun, Stang, Berlin, Wilcox & Li 2009). The top principal components or MDS dimensions are then included in the subsequent association analysis as controls.

Results

To test for an association between the Val158Met SNP and partisan attachment in our primary sample we employed the simple probit regression:

$$P(Partisan_i) = \phi(\alpha_1 + \beta_1 Met_i + \xi_i^{\mathrm{T}} X_i + \epsilon_{1i}) \tag{2.1}$$

where i indexes the subject, Met is the number of COMT Met alleles (0,1,or 2), and ξ is a vector of controls including age in 2009, an indicator variable for whether the subject is male. To guard against population stratification, we include subpopulation assignments based on the structured association method described in the previous section. Assignments to three population groups were derived from an analysis of 36 genetic markers using the software package STRUCTURE (Pritchard, Stephens & Donnelly 2000).[9] STRUCTURE adopts a Bayesian

[8] *Chapter 3* discusses GWAS in more detail.

[9] The marker panel includes D8S1179, D21S11, D7S820, CSF1PO, D3S1358, TH01, D13S317, D16S539, D2S1338, D19S443, vWA, TPOX, D18S51, D5S818, FGA, amelogenin, D1S196,

cluster approach to find the optimal number of clusters under the main modeling assumptions of Hardy-Weinberg equilibrium within populations and complete linkage equilibrium between loci within populations (Pritchard, Stephens & Donnelly 2000). The goal is to identify population groupings that are not in disequilibrium (Pritchard, Stephens & Donnelly 2000). A more detailed description of the ancestry-informative marker panel used here can be found in Yang, Zhao, Kranzler & Gelernter (2005).

Since the replication samples are both made up of nuclear families, and thus the observations are correlated, we estimate the probit model in (2.1) using general estimating equations (GEE) (Liang & Zeger 1986) with an exchangeable working correlation matrix. Population stratification is controlled for in the Sweden sample by including the first 10 PCAs and in the California sample by including the first MDS dimension as controls.

Table 2.1 presents the results for the test of association between the Val158Met SNP of the COMT gene and partisan attachment using our primary and replication samples. The *Met* allele is significantly associated with partisan attachment in the primary sample ($p = 0.002$). The Met allele remained significant ($p = 0.002$) in the primary model with the addition of SES measures.[10] The results based on the two replication samples are mixed. For the California replication sample, the number of Met alleles does not reach significance ($p = 0.25$), however the association between self-reported partisan affiliation and the number of Met alleles is significant at the 10% level ($p = 0.07$).

Figure 2.1 presents the simulated first differences for the primary and replication sample. Holding the control variables at their means and changing the number of Met alleles from zero to one decreases the likelihood of partisan attachment by approximately 10 percentage points and from zero to two by approximately 20 percentage points. Both simulated first differences are significantly different from zero. The effect sizes for the replication samples are much smaller than those for

D1S2628, D2S162, D2S319, D5S407, D5S410, D6S1610, D7S640, D7S657, D8S272, D8S1827, D9S175, D10S197, D10S1786, D11S935, D12S352, D14S68, D15S1002, D16S3017, D17S799, D22S274.

[10]The measures include family income and employment status at the time of participation in the original study. These results are not presented since they are nearly identical.

the primary sample. For the California replication sample, changing the number of Met alleles from zero to one decreases the likelihood of partisan attachment by approximately 3 percentage points and from zero to two by approximately 6 percentage points whereas for the Sweden sample they are 2 and 4 percentage points respectively. However, the simulated first differences are only suggestive since they are not significantly different from zero at the 5% level.

Mediation

We did find a strong significant association between the Val158Met SNP and partisan attachment based on the primary sample, however we only found relatively weak evidence of an association in one of our two replication samples. Therefore, our results should be interpreted with caution given the risk of a false-positive. Based on the findings of Gerber, Huber, Doherty & Dowling (2010) that extraversion is a strong predictor of partisan attachment, we formally test whether the effect of this SNP is mediated by the personality trait extraversion in the primary sample. In other words, is the Met allele associated with partisan attachment because carriers of this allele tend to be less extraverted and thus less likely to affiliate with social groups in general?

Based on Baron & Kenny (1986), a variable M mediates the relationship between an independent variable X, in our case the Val158Met SNP, and a dependent variable Y, in our case partisan attachment, if (a) X significantly predicts M and (b) M significantly predicts Y controlling for X (Baron & Kenny 1986). This relationship is examined *Table 2.3*. The results are consistent with both criteria laid out by Baron & Kenny (1986). The Met allele significantly predicts extraversion ($p = 0.01$) in the first regression and extraversion is significant ($p = 0.04$) in a model of partisan attachment even when the Met allele is included. The Met allele remains significant in the second regression suggesting partial mediation. However, these results are only suggestive.

To formally test for mediation, we follow Imai, Keele & Tingley (2010). Using a potential outcomes framework, the mediation model is defined as:

$$Extraversion_i = \alpha_2 + \beta_2 Met_i + \epsilon_{2i} \tag{2.2}$$

$$Partisan_i = \mathbf{1}\{Partisan_i^* > 0\} : Partisan_i^* = \alpha_3 + \beta_3 Met_i + \gamma Extraversion_i + \epsilon_{3i} \tag{2.3}$$

where the dependent variable is registering as a member of a party, the mediator is the personality trait extraversion, and the treatment is the number of COMT Met alleles. Both of the error terms are assumed to be i.i.d., $\epsilon_{2i} \sim N(0, \sigma_2^2)$, and $\epsilon_{3i} \sim N(0, 1)$ since we are estimating a probit model.

The causal mediation effect in the probit model is defined as:

$$\delta(Met) = \Phi\left(\frac{\alpha_3 + \beta_3 Met + \gamma(\alpha_2 + \beta_2)}{\sqrt{\sigma_2^2\gamma^2 + 1}}\right) - \Phi\left(\frac{\alpha_3 + \beta_3 Met + \gamma\alpha_2}{\sqrt{\sigma_2^2\gamma^2 + 1}}\right) \tag{2.4}$$

for $Met = 0, 1, 2$ due to the fact the treatment is either 0, 1, or 2 Met alleles. Since we have hypothesized that having the Met allele should be associated with reduced extraversion and thus a lower likelihood of partisan attachment, we consider the case of no Met alleles as the baseline condition and compute the causal mediation effects for 1 and 2 Met alleles compared to this baseline (Imai, Keele & Tingley 2010).

The proportion mediated for the case of either one or two Met alleles is:

$$\upsilon(Met) = \frac{\{\delta(0) + \delta(Met)\}/2}{\tau} \tag{2.5}$$

where $Met = 1, 2$ and the numerator is the average causal mediation affect. The quantity τ is the total effect defined as:

$$\tau(Met) = \Phi\left(\frac{\alpha_3 + \beta_3 Met + \gamma(\alpha_2 + \beta_2)}{\sqrt{\sigma_2^2\gamma^2 + 1}}\right) - \Phi\left(\frac{\alpha_3 + \gamma\alpha_2}{\sqrt{\sigma_2^2\gamma^2 + 1}}\right) \tag{2.6}$$

To estimate the average mediation effect as well as the proportion of the total effect that is mediated by extraversion, we used the R package *mediation* (Imai, Keele, Tingley & Yamamoto 2010). We employed the parametric approach with quasi-Bayesian bootstrap to produce confidence intervals based on 1000 simulations. We found a significant average mediation effect for both the case of 1

and 2 Met alleles compared to the baseline of no alleles. Also, 7.8% (95 % CIs 0.7%, 33%) of the total effect of 1 versus 0 Met allele and 7.4% (95 % CIs 0.1%, 29%) of the total effect of 2 versus 0 Met allele on partisan attachment is via the personality trait extraversion. It is important to note that the test of mediation was only performed on the primary sample because measures of personality traits are not available for the replication samples.

An important assumption of mediation analysis is that the error terms in (2.2) (ϵ_{2i}) and (2.3) (ϵ_{3i}) are uncorrelated (Imai, Keele & Tingley 2010). In other words, there are no unobserved confounding variables that may explain the relationship between the mediator and the outcome variable. However, unless both the treatment and mediator are randomly assigned, this assumption may not hold. In addition, since these confounders are unobserved the assumption cannot be empirically tested. The degree to which mediation estimates are sensitive to a violation of this assumption can be analyzed by estimating the average causal mediation assuming varying levels of correlation between ϵ_{2i} and ϵ_{3i} (Imai, Keele & Tingley 2010). Our mediation results are rather sensitive to potential confounders. For a treatment of 1 Met allele, the mediated effect is truly zero if the correlation between error terms is 0.11.

Discussion

We show in a sample of young adults that an extensively studied gene that is responsible for the degradation of the neurotransmitter dopamine is associated with partisan attachment. More specifically, the less active Met allele of the COMT Val158Met SNP is associated with a significantly lower probability among registered voters of being attached with a major party. The first differences for individuals who have one and two Met alleles compared to none are 10% and 20% respectively. The predicted probability of being a partisan when having 0, 1, and 2 Met alleles is 74%, 64%, and 54% respectively. However, we failed to replicate this association in a different sample of California voters. We did find weak evidence of a significant association between the Met allele and self-reported partisan attachment in a large sample of older Swedish citizens, however the effect sizes were

only about one-fifth of those found for the primary sample.

There are several differences in the primary and Sweden replication samples that may have contributed to the weak replication results and dramatically different effects sizes. The primary sample is much younger than the Swedish sample; the measures for the dependent variable is different in the Swedish sample; the political landscape and institutions of the United States and Sweden are very different; and the linkage disequilibrium patterns of both populations may be different. Future work comparing US and Swedish samples, possibly using data described in *Chapter 3*, is necessary to explore these possibilities. The failed replication using the California sample may simply be due to a lack of power due to a relatively small sample (Benjamin, Chabris, Glaeser, Gudnason, Harris, Laibson, Launer & Purcell 2007). However, until additional replications are attempted we cannot rule out the possibility of a false-positive.

Based on previous research, we hypothesize that having the Met allele leads to being less extraverted which in turn makes one less likely to be a partisan. We do find a significant indirect effect, however our results are suggestive of partial mediation. Approximately 8% of the observed association with partisan attachment is via extraversion. This result provides the first empirical support for a casual relationship between genes, personality, and political behavior. However, the fact that a vast majority of the association is not mediated by extraversion is also consistent with pleiotropy, meaning that Val158Met primarily influences both extraversion and partisan attachment independently rather than sequentially. In addition, a sensitivity analysis showed that our mediation results are highly sensitive to possible confounders.

While these results must be carefully interpreted, this paper makes at least three important contributions. Our main contribution is to conduct the first formal test of the conjecture that genes affect partisan attachment via their influence on the personality trait extraversion. This mediation hypothesis is plausible given that approximately half of the variation in both partisan attachment (Settle, Dawes & Fowler 2009) and extraversion (Bouchard & McGue 2003) can be attributed to genetic factors in general. More specifically, Stein et al. (2005) and Reuter & Hennig

(2005) suggest the link between Val158Met and extraversion and Gerber, Huber, Doherty & Dowling (2010) provide the link between extraversion and partisan attachment.

Another contribution is our presentation of the analytical framework necessary to explore the complex pathways that underlie an association between a candidate genetic polymorphism and a behavior (Munafo 2006). Since individual genes tend to influence many different traits at the same time and complex behaviors are influenced by many genes, it is necessary to investigate each of the proposed theoretical pathways. The next wave of work in the area of genopolitics must attempt to better understand the causal links between genes and political behaviors. Mediation analysis is invaluable for uncovering those links and will ultimately allow us to develop and improve theories of political behavior.

A related contribution is our demonstration that the proportion mediated by extraversion is relatively modest. This suggests, especially given the fact the Met allele remains significant in a model of partisan attachment even after extraversion is included, that the Val158Met SNP operates via additional pathways. Knowing that there are likely several mediators between specific genetic variants and political behaviors, each playing a relatively small role, is useful to set our expectations for future research. There is a consensus that approach behaviors in response to stimuli associated to reward are related to differential activity in dopamine neurotransmission (Revelle 2005, Munafo et al. 2003). Approach behaviors include extraversion, impulsivity, novelty seeking, and positive affectivity (Revelle 2005). Here we have focused on extraversion because of its clear link to partisanship, however it may be the case that these related behaviors are also mediators and should thus be studied further.

Our results, combined with the findings reported by Dawes & Fowler (2009), strongly suggests that the dopamine system is a good place to look for additional genetic variants associated with partisan attachment. The authors of that study found that a polymorphism of the D2 dopamine receptor gene linked to lower density of dopamine receptors, and thus weaker dopamine signaling in the brain, to be significantly associated with a lower likelihood of partisan attachment. Since

many genes influence a complex behavior like partisan attachment, it is likely that other systems play a role as well. A genome-wide association study, such as the study presented in *Chapter 3*, represents an unconstrained search testing hundreds of thousands of variants across the entire human genome. Since the Swedish sample has been genotyped for a genome-wide association study, the next step is to search for additional variants that may be associated with partisan attachment.

This study has at least two important limitations. First, it is possible that the estimate of the association between the Val158Met SNP and partisanship mediated by extraversion is biased downward because a self report measure of extraversion, such as the NEO may not be sensitive enough to capture genetic variation especially given our relatively small sample size (Ebstein 2006). Second, our dependent variable in the primary and California replication sample is technically party registration rather than party identification so we are limited to those who formally register with a party.

Finally, how does this work change the way we view partisan attachment? Our study underscores previous work demonstrating that individual differences in the propensity to form a partisan attachment can be traced back to differences in genetic endowments (Settle, Dawes & Fowler 2009, Dawes & Fowler 2009). The prevailing narrative of partisan attachment states that as political issues become more salient due to close associations with social groups and integration into the community, it results in the formation or strengthening of partisan ties (Campbell et al. 1960). This assertion, combined with empirical evidence, suggests that genes may have a downstream effect on partisan attachment via more basic predispositions towards social interactions. The Big Five trait extraversion is believed to represent this predisposition. While we find limited support for this proposed causal pathway, we believe this is the type of theorizing that will help discover the causal mechanisms between genes and political behaviors, ultimately leading to a better understanding of these behaviors.

Tables

Table 2.1: Sample frequencies, means, and standard deviations.

	Primary	CA Replication	Sweden Replication
Val / Val	0.32	0.26	0.20
Val / Met	0.50	0.49	0.50
Met / Met	0.18	0.25	0.30
Partisan	0.64	0.78	0.69
Age (mean)	26.21	47.73	61.66
Age (sd)	3.17	16.38	4.25
Male	0.26	0.27	0.43
N	510	460	3783
N with Extraversion	495	n/a	n/a

Table 2.2: Test of association between COMT Val158Met SNP and partisan attachment. The primary model is a simple probit and the replication models are a GEE probit with an exchangeable working correlation. The dichotomous dependent variable in the primary and California replication samples is whether a registered voter is affiliated with the Democratic or Republican party and in the Sweden replication sample is self-reported affiliation. *Met* is the number of Met alleles (0, 1, or 2). Controls for population stratification are included in each of the model but are not shown. Probit coefficients (Coef), standard errors (SE), and P-values are presented.

	Primary Sample			Replication Samples					
				California			Sweden		
	Coef	SE	P-val	Coef	SE	P-val	Coef	SE	P-val
Intercept	0.26	0.53	0.62	0.34	0.25	0.17	-0.62	0.32	0.06
Met	-0.26	0.09	0.00	-0.11	0.10	0.25	-0.06	0.03	0.07
Age	0.02	0.02	0.42	0.01	0.00	0.00	0.02	0.01	0.00
Male	-0.17	0.13	0.18	-0.23	0.15	0.13	0.14	0.04	0.00
N		510			460			3783	
Wald (χ^2)		16.2			33.7			44.4	
Wald D.F.		5			4			13	
Wald P-val		0.00			0.00			0.00	

Table 2.3: Test of association between COMT Val158Met SNP and both partisan attachment and extraversion. The partisan attachment model is a simple probit and the extraversion model is a linear regression. The dichotomous dependent variables are *Partisan*, which is whether a registered voter is affiliated with the Democratic or Republican party, *Met* is the number of Met alleles (0, 1, or 2), and *Extraversion*, which is the T-score of the NEO-FFI extraversion factor score. Probit and linear coefficients (Coef), standard errors (SE), and p-values are presented.

	Extraversion			Partisanship		
	Coef	SE	P-val	Coef	SE	P-val
Intercept	46.4	5.3	0.00	-0.31	0.61	0.50
Met	-1.96	0.74	0.01	-0.24	0.09	0.01
Extraversion				0.01	0.01	0.04
Age	0.10	0.28	0.71	0.02	0.02	0.37
Male	3.58	1.12	0.00	-0.22	0.13	0.10
N		496			496	
Wald Test of Model Fit (χ^2)		20.1			15.2	
Wald Test Degrees of Freedom		5			6	
Wald Test P value		0.00			0.02	

Figures

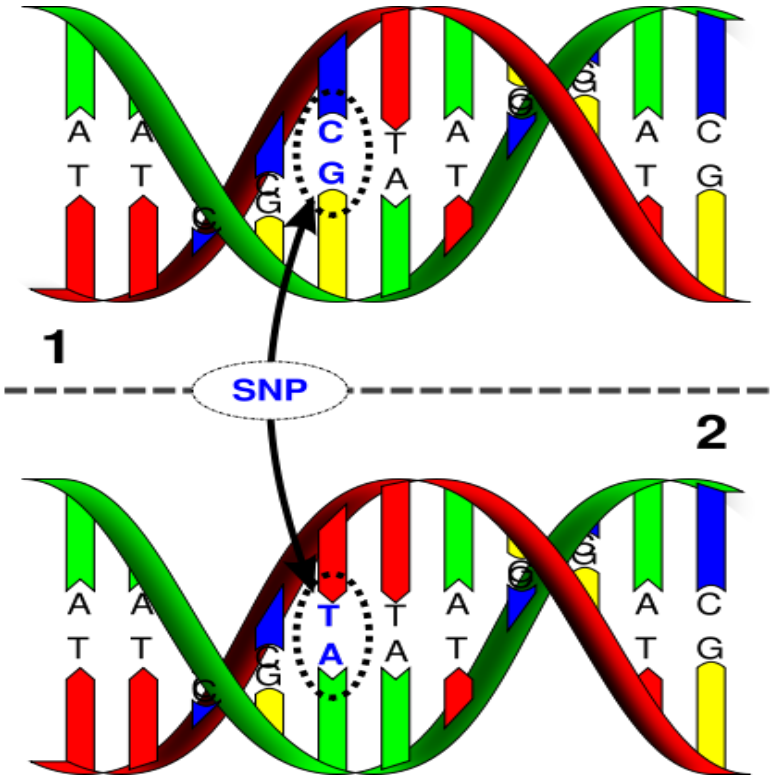


Figure 2.1: Single Nucleotide Polymorphism

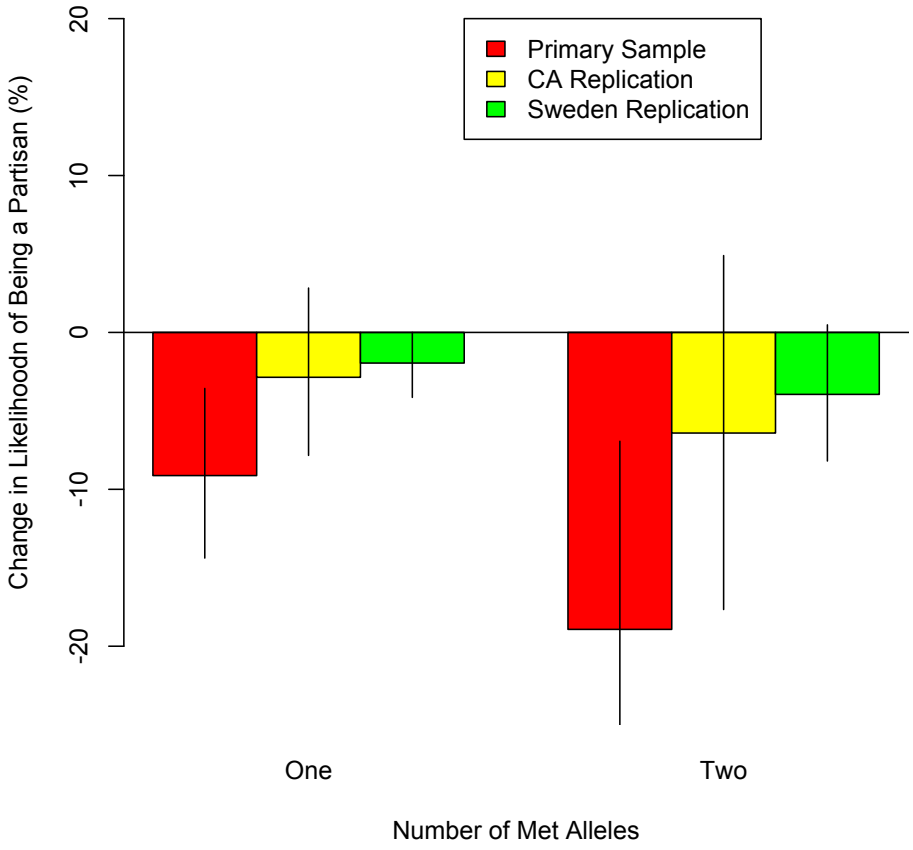


Figure 2.2: Changing the number of Met alleles yields significantly lower partisan attachment. First differences, based on simulations of *Table 2.2* parameters, are presented along with 95% confidence intervals. All other variables in the model are held at their means.

Chapter 3

A Genome-Wide Association Study of Voter Turnout

Introduction

Fowler, Baker & Dawes (2008) were the first to show that both validated and self-reported voter turnout have a significant genetic basis. In a follow-up study, Fowler & Dawes (2008) found significant associations between two genetic variants known to play an important role in the serotonin and dopamine neurotransmission systems and self-reported turnout. However, these two variants are a tiny subset of the likely many that are associated with turnout.

Fowler & Dawes (2008) represents what is known as a *candidate gene* association study in that only a small number of genetic variants are investigated and the choice of which variants to study is based on previous findings or hypothesized biological pathways. A major limitation of this approach is that researchers currently have an imperfect understanding of these pathways (Pearson & Manolio 2008). *Genome-wide* association studies (GWAS) represent an advancement beyond candidate gene studies because they investigate hundreds of thousands of variants across the entire human genome and are not constrained by prior hypotheses (Pearson & Manolio 2008). Due to advances in technology and constantly falling costs associated with genotyping, researchers are currently conducting genome-wide studies investigating over 1,000,000 SNPs (Witte 2010).

Political scientists are interested in studying genotypic data to better understand the biological mechanisms that underly behaviors of interest. The hope is that knowledge of such biological mechanisms will aid in developing or refining theories. For example, patience has been demonstrated theoretically and empirically (Fowler & Kam 2006) to be a determinant of whether or not individuals participate in politics. Economists simply take preferences as given, considering them as exogenous, however genetic differences have been shown to explain a portion of the heterogeneity in time preferences (Eisenberg, MacKillop, Modi, Beauchemin, Dan, Lisman, Lum & Wilson 2007). Therefore, incorporating genetic information may allow us to partition coarse concepts like patience into more fundamental biologically plausible components. A preference for immediate rewards is likely a holdover from smaller-scale societies when it often made evolutionary sense to consume immediately rather than wait since immediate survival is of the utmost importance. Then the bias towards impatience then may be due in part to the fact our genome is equipped to deal with problems we no longer face. This genetic predisposition may have consequences for modern behaviors like participating in politics.

In addition to informing theories of political behavior, understanding the biological underpinnings of political behaviors may help to inform policy decisions. Hypothetically, there may be some political behaviors that are strongly influenced by biological factors and thus may be difficult to alter with policy levers. For example, if it were found that all of the influence of patience on voter turnout were due to genetic factors then it suggests that spending considerable resources on a campaign to highlight the long-run benefits of political participation may have a limited impact.[1]

While thus far there have been no published GWAS of political behavior, political scientists are beginning to move towards this type of analysis by partnering with researchers in health and medicine who have collected this data for other purposes. The National Longitudinal Study of Adolescent Health (Add Health), a

[1]Of course just because a trait is influenced by genes does not mean environmental factors are not important. The point is that the interplay between genes and environment should be more carefully considered.

nationally representative study that has asked political questions in the last two of its waves, will soon release to researchers genome-wide marker data for a subset of its 12,200 participants. This will enable GWAS of political traits such as ideology, voting, partisanship, and overall participation.

This paper presents the first GWAS of voter turnout and utilizes three novel data sets. The paper is structured as follows: we first describe the sample used for the primary analysis, we then present the results from a twin study design of validated voter turnout, next we present the results from a GWAS analysis and attempted replication of using two different samples, and finally we discuss the results and suggest future steps for this project. To preview our findings, we demonstrate that voter turnout is moderately heritable with estimates comparable to previously reported results but we are unable to uncover specific genetic variants that are significantly associated with turnout after correcting for multiple hypothesis testing. Further, we failed to replicate "suggestive" associations in two additional data sets.

Sample

The primary data set used for this analysis is based on two studies actively being conducted by the Minnesota Center for Twin and Family Research (MCTFR). The first is the Minnesota Twin Family Study (MTFS) which is a population-based, longitudinal study of 1,197 monozygotic (MZ) and 684 dizygotic (DZ) like-sex twin pairs and their parents (Iacono, McGue & Krueger 2006). Data was collected on these twins and their parents at an initial assessment, as well as follow-ups for the twins occurring at roughly 3-year intervals. The MTFS is comprised of two age cohorts, one in which subjects were 11 years old at the time of their initial assessment and the other in which subjects were 17 years old. The younger cohort was born between 1977-1984 and the older cohort was born between 1972-1979. The second study, the Sibling Interaction and Behavior Study (SIBS), is a longitudinal study of adopted and non-adopted adolescents and their parents and includes 208 families with biological siblings (Iacono, McGue & Krueger 2006). Participants in both studies have been shown to be compara-

ble to the overall Minnesota population.[2] A sample of 8,405 individuals from the two studies were genotyped using Illumina human 660W quad chip which contains 592,652 SNPs. After quality control checks, 510,579 autosomal SNPs remained.

In order to construct a genetically informative sample of *validated* voting behavior, we matched the MTFS and SIBS data sets with public voting records available through the Minnesota Secretary of State's office using subject name, address, telephone number, and year of birth information.[3] The dependent variable for this analysis is turnout in the 2010 midterm general election held on November 2, 2010. Of the 8,405 genotyped subjects, 7,136 were both of legal voting age and current residents of Minnesota. The matching process yielded matches for 5,954 of the 7,136 genotyped subjects. The match rate of 83.4% is nearly identical to the percentage of the Minnesota voting eligible population registered for the 2010 election (82.3%). Since we have an up-to-date address and phone number for the GWAS participants we assume those Minnesota residents not matched to the voter records are not registered and thus are considered abstainers in this analysis. The overall rate of turnout for the sample is similar to statewide turnout (55.6% and 55.9% respectively).[4] Summary statistics for the sample are presented in *Table 3.1.*

Latent Model

Analysis

Twin studies compare the behavior (or *phenotypes*) of twins who share 100% of their genetic material (identical or *monozygotic* twins) to those who, on average, share 50% of their genetic material (fraternal or *dizygotic* twins).[5] The twin study design can be extended to include other family members of known genetic relatedness allowing the relaxation of certain model assumptions as well as the

[2]For a more detailed description of both the MTFS and SIBS studies see Iacono, McGue & Krueger (2006).

[3]The Minnesota voter file was created on December 25, 2010.

[4]Statewide statistics are taken from the Minnesota Secretary of State's web site (http://electionresults.sos.state.mn.us).

[5]For a more detailed discussion of twin models see *Chapter 1* and for a primer designed for political scientists see (Medland & Hatemi 2009).

estimation of additional parameters (Hatemi et al. 2010). Since the SIBS sample includes non-twin biological and unrelated siblings, we can estimate whether the environment twins share is significantly different than the environment non-twin siblings share. In addition, unrelated siblings give us a direct estimate in shared environment.

The so-called ACE model can be extended to include twin-only environment (ACTE):

$$y_{ij} = \mu + A_{ij} + C_j + T_j + E_{ij} \tag{3.1}$$

where j denotes the family, i denotes the individual twin in the family, $A_{ij} \sim N(0, \sigma_A^2)$ is the additive genetic component, $C_j \sim N(0, \sigma_C^2)$ is the shared environment component, $T_j \sim N(0, \sigma_T^2)$ is the twin-specific shared environmental component, and $E_{ij} \sim N(0, \sigma_E^2)$ is the unshared environment component.

For MZ twins, DZ twins, biological non-twin siblings, and unrelated siblings:

$$Var(y) = \sigma_A^2 + \sigma_C^2 + \sigma_T^2 + \sigma_E^2, \tag{3.2}$$

$$COV_{MZ}(y_{1j}, y_{2j}) = \sigma_A^2 + \sigma_C^2 + \sigma_T^2, \tag{3.3}$$

$$COV_{DZ}(y_{1j}, y_{2j}) = 0.5\sigma_A^2 + \sigma_C^2 + \sigma_T^2 \tag{3.4}$$

$$COV_{BIO}(y_{1j}, y_{2j}) = 0.5\sigma_A^2 + \sigma_C^2 \tag{3.5}$$

$$COV_{UR}(y_{1j}, y_{2j}) = \sigma_C^2 \tag{3.6}$$

The covariance equations reflect the genetic relatedness of sibling pairs and the assumed shared environmental conditions. Based on these equations, we can estimate the ACTE model via a random effects regression model where the 2×2 variance-covariance matrix is specified as:

$$\mathbf{\Omega_j} = \begin{bmatrix} \sigma_A^2 + \sigma_C^2 + \sigma_T^2 + \sigma_E^2 & R_j\sigma_A^2 + \sigma_C^2 + T_j\sigma_T^2 \\ R_j\sigma_A^2 + \sigma_C^2 + T_j\sigma_T^2 & \sigma_A^2 + \sigma_C^2 + \sigma_T^2 + \sigma_E^2 \end{bmatrix}$$

where R is the genetic relatedness of the twin pair equaling 1 for MZ twins, 0.5 for DZ twins and non-twin biological siblings, and 0 for unrelated siblings and T is an indicator for whether or not the sibling pair is made up of twins or non-twins. We follow the mixed-effects ACE model parameterization developed by Rabe-Hesketh,

Skrondal & A (2008). We extend their the ACE model to include a twin-effect such that

$$y_{ij} = \mu + A_{ij}(\phi_j + \sqrt{0.5}\bar{\gamma_j}) + A_j(\gamma_j + \sqrt{0.5}\bar{\gamma_j}) + C_j + \delta_j T_j + E_{ij} \tag{3.7}$$

where γ_j is an indicator for MZ twins and $\bar{\gamma_j}$ is an indicator for DZ twins and biologically related siblings, δ_j is a twin indicator, and ϕ_j is a biologically unrelated sibling indicator.

This yields $var(A_{ij}) = var(A_j) = \sigma_A^2, var(C_j) = \sigma_C^2, var(T_j) = \sigma_T^2$, and $var(E_{ij}) = \sigma_E^2$. Since our dependent variable is binary we model it as a probit. We use the variances of the random effects to generate estimates of heritability, common environment, and unshared environment.[6] Since the residual variance is fixed in a probit model this ACE model is unidentified. As a result, we must fix $\sigma_E = 1$. Therefore, the model we estimate is:[7]

$$P(Vote_{ij} = 1) = \Phi(\mu + A_{ij}(\phi_j + \sqrt{0.5}\bar{\gamma_j}) + A_j(\gamma_j + \sqrt{0.5}\bar{\gamma_j}) + C_j + \delta_j T_j) \tag{3.8}$$

To estimate the variance components in ACTE models we employ Bayesian methods, implemented using Markov Chain Monte Carlo (MCMC) algorithms.[8] We choose vague prior distributions to ensure they do not drive our results. For the thresholds we use a mean-zero normal distribution with variance $1,000,000$ and for the precision parameters associated with σ_A^2, σ_C^2, and σ_T^2 we use a Pareto distribution with shape parameter equal to 1 and scale parameter equal to 0.001 which is the equivalent of putting a uniform $(0, 1000)$ prior on the variances.[9] We began sampling from the joint posterior distribution after convergence was established using $\hat{R}$ (values of less than 1.1 on each parameter indicate convergence) (Gelman, Carlin, Stern & Rubin 2004). For all of the models the burn-in period was $500,000$ iterations and the chains were thinned by 500.

[6] They are defined as $\frac{\sigma_A^2}{\sigma_A^2+\sigma_C^2+\sigma_T^2+\sigma_E^2}$, $\frac{\sigma_C^2}{\sigma_A^2+\sigma_C^2+\sigma_T^2+\sigma_E^2}$, $\frac{\sigma_T^2}{\sigma_A^2+\sigma_C^2+\sigma_T^2+\sigma_E^2}$, and $\frac{\sigma_E^2}{\sigma_A^2+\sigma_C^2+\sigma_T^2+\sigma_E^2}$ respectively.

[7] Also included in the model are controls for age and gender.

[8] For a detailed discussion of Bayesian ACE models, we refer to van den Berg, Beem, and Boomsma (2006).

[9] A Pareto distribution has proven to work well for variance components in genetic models (Burton, Tiller, Gurrin, Cookson, Musk & Palmer 1999).

In addition to estimating an ACTE model we test whether the twin-effect can be dropped by comparing model fit based on the deviance information criterion (DIC), a Bayesian method for model comparison analogous to the Akaike Information Criterion (AIC) in maximum likelihood estimation. Models with smaller DIC are considered to be superior (Gelman *et al.* 2004). We also ran an ACE model excluding non-twin biological and unrelated siblings.[10]

Results

As a first step, we compare bootstrapped correlations for MZ and DZ twins (*Table 3.2*). If MZ twins have a significantly higher correlation than DZs this is suggestive of genetic influence.[11] The difference between MZ and DZ twins for turnout is significant ($P = 0.003$ one-sided). Also, the correlation for unrelated sibling pairs provide a direct estimate of shared environment which turns out to be quite similar to the estimate inferred by comparing MZ and DZ twins.[12] Finally, the correlation for biological sibling is expected to be similar to the correlation for DZ twins under the assumption that fraternal twins are no more similar, genetically or environmentally, than ordinary siblings. The difference between DZ twins and biologically related siblings is not significant in our sample ($P = 0.16$ two-sided), however the correlation for biologically related siblings is noisy due to small sample size. The difference would also be captured by the modeled twin effect which compares twins to biologically related and unrelated non-twin siblings.

Parameter estimates from the structural equations models are presented in *Table 3.3*. The ACTE and nested ACE model yield estimates of heritability of 0.42 [0.24, 0.60] and 0.49 [0.28, 0.67] respectively. The common environment is small but significant in both models, 0.13 [0.01, 0.27] and 0.18 [0.03, 0.37], and the unshared environment is the second largest component, 0.38 [0.31, 0.48] and 0.33 [0.26, 0.41]. The twin-effect is not significantly different from zero in the ACTE model suggesting that twins and non-twins do not appear to behave

[10] This model is not nested in the other two so the DICs cannot be directly compared.

[11] This is a one-tail test since our hypothesis is that the MZ correlation is higher than the DZ correlation.

[12] $2 * CORR_{DZ} - CORR_{MZ}$

differently. An interpretation of this finding is that twins are not environmentally different than non-twins. The ACTE model fits worse than the ACE based on the DIC statistic and the estimates along with their credible intervals are illustrated graphically using *Figure 3.1.* However, while dropping insignificant parameters from the model is common practice in behavior genetics, it is not without its critics (Goldberger 2002) because the reported p-values do not reflect the model selection procedure.

There are a few key points to take away from the biometric modeling results. First, all three heritability estimates are between 0.41 and 0.49 which accords with the validated turnout results reported by Fowler, Baker & Dawes (2008).[13] However, the authors of that study found a larger shared environmental component. This may be due to differences in the two samples or it may be that we are better able to estimate shared environment in this study since we include unrelated siblings in the analysis. Second, this establishes the upper bound of additive genetic influence that can be accounted for by the GWAS analysis. As will be discussed later, GWAS studies have consistently failed to account for the amount of heritable variation identified by twin studies. This is known as the "missing heritability" problem.

Genome-wide Association

Two issues all genome-wide association studies face are population stratification and multiple testing. A significant association can mean that the allele itself influences partisanship or that the observed association is a false positive signal due to what is known as *population stratification.* Population stratification occurs when groups have different allele frequencies due to their genetic ancestry. Political behavior in these groups may be affected by their environments, alleles other than the one of interest, or some unobserved factor. Once these two groups mix in a larger population, simply comparing the frequency of an allele to the number of voters in the mixed population would lead to a spurious association.

[13]It is important to point out that Fowler, Baker & Dawes (2008) is the only published study demonstrating voter turnout to be heritable. Therefore, this is the first study validating their result.

As discussed in *Chapter 2*, one approach to accounting for population stratification in a genome-wide association study is to use the principal components approach developed by Price et al. (2006). This approach computes principal components based the SNPs across the genome and the first 10 principal components associated with the 10 largest eigenvalues capture nearly all of the information about the underlying population structure of the sample. This is the approach we take for this analysis.

Due to the fact that we will be testing a large number of hypotheses we should expect to find some significant associations that are purely the result of sampling variation. Therefore, we must correct for this fact. The conventional way to do this is to use a Bonferroni correction which divides the p-value by number of tests performed. Since we are testing 510,579 autosomal SNPs, to achieve genome-wide significance we need a p-value of $9.79 * 10^{-8}$ or smaller.

Analysis

A case-control genetic association study indicates whether a particular allele is found more frequently than can be attributed to chance in a group exhibiting a particular trait than those without the trait. In our case, is the frequency of a particular allele higher among voters than non-voters? To test for an association between each of the SNPs and turnout we can repeatedly run the the probit regression model:

$$P(Vote_i = 1) = \Phi(\tau + \beta_{SNP} SNP_i + \beta_{Age} Age_i + \beta_{Male} Male + \beta_{PC_k} PC_{ik}) \quad (3.9)$$

where i indexes the subject, SNP is the number of a particular allele (0,1,2) for the SNP being tested, Age is age at the time of election, $Male$ is an indicator variable for whether the subject is male (1) or female (0), and PC is a vector of the 10 principal components ($k = 1...10$). Since the sample is made up of nuclear families, and thus the observations are correlated, we estimate the model using general estimating equations (GEE) (Liang & Zeger 1986) with an exchangeable working correlation matrix.

Results

The p-values corresponding to regressions of voter turnout on each SNP are represented in *Figure 3.2*. The blue line represents the Bonferroni threshold for genome-wide significance. None of the SNPs reach genome-wide significance and the closest achieves a p-value of 10^{-6} which can at best be interpreted as suggestive. The top 40 SNPs in terms of significance are listed in *Table 3.4*.

The Bonferroni correction is overly conservative since it does not account for the fact that SNPs may be correlated with one another due to linkage disequilibrium. Therefore, some of these SNPs may be true associations rather than false positives. This is especially true given the fact that since GWAS focus on common variants, and these common variants individually have very small effects, many true associations are ignored due to the correction. Finally, while our sample size is not small, GWAS often require very large samples Benjamin et al. (2007). In an effort to see if any of our top hits may be real, we attempt to replicate them.

Replication

We attempt to replicate a subset of the 40 most significant associations available in two distinct samples. The first replication sample comes from the SALT study which is made up of Swedish twins born between 1936 and 1958 (see Lichtenstein et al. (2002) for a description of the SALT sample). A subset of SALT respondents (N=10,946) were genotyped by the SNP&SEQ Technology Platform, Uppsala, using the Illumina HumanOmniExpress BeadChip genotyping platform. Of those genotyped, 3,947 participants also completed the SALTY survey described in *Chapter 1*. These subjects have been genotyped for 26 of the top 40 MN SNPs. As part of they SALTY survey, respondents reported whether they voted in the 2006 Swedish parliamentary as well as the 2009 European Union election

The second replication sample is comprised of nuclear families including identical and non-identical twins, siblings, and their parents collected by researchers at the University of California, San Diego School of Medicine. This sample was also used in the analysis presented in *Chapter 2* (for a detailed description of the sample, see Zhang et al. (2004)). 455 subjects were genotyped on 592,312 SNPs

using the Illumina 610 Quad genotyping array. In order to determine whether or not subjects voted in the 2010 general election they were matched with public voting records available through the California Secretary of State's office.[14] The matching process for the replication sample was nearly identical to that of the Minnesota sample utilizing available personal information including name, date of birth, phone number, and home address information. We were able to match 75% of the subjects which was close to the 73.4% of eligible Californians were registered to vote as of the November 2010 election.[15]

Since both replication samples also includes multiple members from the sample family, we employ the same modeling strategy as with the Minnesota sample. Population stratification is in the Sweden sample controlled for by including the first 10 PCAs and in the California sample, by including the first MDS dimension as controls.

The results for the replications are presented in *Table 3.5*. After Bonferroni correction, none of the Minnesota SNPs in either of the replication samples are significant. rs6539695 is associated with self-reported turnout in the 2009 EU parliamentary election at the uncorrected 5% level and rs13289732 as well as rs10979414 are associated with validated turnout in the 2010 California midterm election at the 10% level. rs9871575, rs3792785, rs8030871, rs17133433, and rs1637995 are also significant at the uncorrected 5% level but the associations are the opposite direction as the Minnesota GWAS.

Discussion

The estimates from the latent modeling based on twin, biological non-twin, and unrelated siblings suggests that over 40% of the variation in voter turnout could be attributed to additive genetic factors. However, we were not able to identify any genome-wide significant associations when analyzing over 500,000 genetic markers typed across the genome. We were also not able to replicate any of the suggestive associations in two replication samples after correcting for multiple testing. As

[14]Voter records are from December 2010.

[15]This information come from the California Secretary of State (http://www.sos.ca.gov/elections/sov/2010-general/04-historical-voter-reg-participation.pdf).

was discussed in *Chapter 2*, the lack of replication may be due to false-positive signals of association in the Minnesota sample or alternatively due to differences in the dependent variable, sample sizes, and political environment across the three samples.

While the Minnesota GWAS results may be viewed as disappointing, they are not surprising given the fact that voting in an election is a complex behavior that must be influenced by many genes with very small individual effects. This is compounded by the fact that the relationship between genes and turnout is likely mediated by many different phenomenon. Therefore, it will be very difficult to identify these effects after correcting for multiple testing without very large sample sizes (Benjamin et al. 2007).

This research project can be taken in a few different direction. One possibility is to pool these results, as well as results from future GWAS of voter turnout, into a meta-analysis in order to increase our power to detect small effects. As was mentioned in the introduction, Add Health will release a large GWAS sample which contains self-reported measures of turnout. Social scientists are also beginning to follow the model currently practiced in medical genetics of creating large-scale consortia made up of many different studies in order to achieve the power necessary to detect small effects. For example, the Social Science Genetic Association Consortium (SSGAC) was recently launched in collaboration with a team of social scientists and geneticists. The SSGAC is currently made up of thirty studies whose combined sample size exceeds 100,000 individuals with genome-wide association data. A long-term goal of SSGAC is to ultimately study several additional traits of interest to political scientists.

Another alternatively is to aggregate the effect of many SNPs into an index and in order to determine if there is a polygenic effect on turnout that is not being detected due to the small effect of each individual SNP failing to survive multiple testing correction (Wray, Goddard & Visscher 2007). While this strategy lessens the severity of correcting for multiple testing, information about specific SNPs is potentially lost. This is important if SNPs, based on their function, provide information about potential causal pathways.

While the goal of conducting the research presented in this chapter is to better understand the biological mechanisms that underly behaviors of interest, our results illustrate that this will be a difficult task. Even though the biometric modeling done on the sample suggests that nearly half of the variation in turnout is due to genetic factors, the GWAS failed to uncover any genome-wide significant associations. This suggests, as Benjamin et al. (2007) argues, that much larger sample sizes may be necessary to study political traits such as voter turnout.

Tables

Table 3.1: Sample summary statistics.

Model	Vote	Age	Male
MZ Twins	0.38	27.76	0.48
	[0.48]	[6.16]	[0.50]
DZ Twins	0.36	27.49	0.47
	[0.48]	[6.09]	[0.50]
BIO Sibs	0.32	23.30	0.49
	[0.47]	[2.82]	[0.50]
UR Sibs	0.29	23.76	0.43
	[0.46]	[2.88]	[0.50]
Full Sample	0.56	40.81	0.47
	[0.50]	[16.12]	[0.50]

Table 3.2: Bootstrapped correlations and 95% confidence intervals for MZ and DZ twins, biologically related and unrelated siblings, and the number of complete same-sex pairs with non-missing data for turnout.

Model	Correlation	N Pairs
MZ Twins	0.69	799
	[0.62, 0.75]	
DZ Twins	0.49	400
	[0.35, 0.62]	
BIO Sibs	0.18	81
	[-0.16, 0.53]	
UR Sibs	0.24	138
	[-0.05, 0.50]	

Table 3.3: Summary of Model Results. Note: The ACTE model consists of additive genetic factors (A), shared or common environmental factors (C), shared twin environment (T), and unshared environmental factors (E). The shared twin environment is dropped in the ACE model. It is estimated with controls for age and gender.

Model	Heritability (%)	Shared Environ	Twin Effect	Unshared Environ	DIC
ACTE	0.42	0.13	0.07	0.38	3153.0
	[0.24, 0.60]	[0.01, 0.27]	[0.00, 0.21]	[0.31, 0.48]	
ACE (All)	0.49	0.18		0.33	2982.0
	[0.28, 0.67]	[0.03, 0.37]		[0.26, 0.41]	
ACE (Twins)	0.41	0.26		0.33	2482.0
	[0.12, 0.66]	[0.04, 0.52]		[0.26, 0.41]	

Table 3.4: 40 most significant SNPs in the MN GWAS sample.

SNP	Chr	Minor Allele	Estimate	P-val	N
rs8030871	15	G	0.17	$2.69*10^{-6}$	6607
rs1157449	8	A	-0.16	$3.33*10^{-6}$	6607
rs333122	17	G	-0.12	$3.37*10^{-6}$	6607
rs7738388	6	G	-0.11	$5.13*10^{-6}$	6607
rs968048	4	G	-0.11	$5.72*10^{-6}$	6607
rs206777	6	G	-0.11	$6.30*10^{-6}$	6607
rs4390542	15	A	0.15	$6.92*10^{-6}$	6607
rs6062340	20	G	0.17	$8.34*10^{-6}$	6596
rs312357	4	A	-0.11	$9.23*10^{-6}$	6554
rs2346115	12	G	0.14	$9.37*10^{-6}$	6607
rs2236426	7	A	0.16	$9.85*10^{-6}$	6607
rs10866945	8	G	0.13	$1.36*10^{-5}$	6607
rs312368	4	A	-0.11	$1.37*10^{-5}$	6606
rs1858367	8	G	-0.18	$1.57*10^{-5}$	6596
rs312394	4	A	-0.11	$1.66*10^{-5}$	6607
rs1155088	8	G	-0.11	$1.80*10^{-5}$	6607
rs3792785	5	G	0.18	$2.10*10^{-5}$	6607
rs9871575	3	G	0.11	$2.45*10^{-5}$	6606
rs1858369	8	G	-0.17	$2.84*10^{-5}$	6607
rs2899492	15	G	0.14	$2.85*10^{-5}$	6604
rs13289732	9	G	-0.11	$2.86*10^{-5}$	6607
rs1490882	4	A	-0.11	$3.05*10^{-5}$	6607
rs580962	6	G	-0.10	$3.09*10^{-5}$	6607
rs12744696	1	A	-0.16	$3.10*10^{-5}$	6607
rs11900053	2	A	-0.18	$3.11*10^{-5}$	6607
rs1779186	1	C	-0.16	$3.32*10^{-5}$	6605
rs7524360	1	A	-0.16	$3.35*10^{-5}$	6607
rs858447	1	G	-0.16	$3.78*10^{-5}$	6607
rs6539695	12	C	0.20	$3.94*10^{-5}$	6607
rs2040840	7	G	0.10	$4.06*10^{-5}$	6607
rs1832446	6	A	-0.10	$4.07*10^{-5}$	6607
rs10979414	9	A	-0.10	$4.12*10^{-5}$	6607
rs6835841	4	A	-0.10	$4.13*10^{-5}$	6607
rs12672266	7	G	0.13	$4.26*10^{-5}$	6607
rs11847577	14	G	-0.12	$4.48*10^{-5}$	6607
rs206767	6	C	-0.10	$4.60*10^{-5}$	6607
rs897275	8	A	0.10	$4.66*10^{-5}$	6605
rs1637995	19	A	0.34	$4.89*10^{-5}$	6607
rs17133433	11	A	0.13	$4.91*10^{-5}$	6607
rs2414167	15	G	0.13	$4.96*10^{-5}$	6607

Table 3.5: Attempted replication of 26 out of the 40 most significant SNPs in the MN sample based on samples from Sweden and CA. Significant associations at uncorrected levels of significance are in bold.

	Sweden Replication						California Replication		
	2006 Swedish			2009 EU			2010 Midterm		
SNP	Est	P-val	N	Est	P-val	N	Est	P-val	N
rs6539695	0.15	0.21	3782	**0.13**	**0.04**	3735	0.13	0.53	415
rs13289732	-0.02	0.68	3782	0.03	0.37	3735	**-0.20**	**0.07**	415
rs10979414	-0.02	0.70	3785	0.03	0.40	3738	**-0.20**	**0.09**	415
rs9871575	-0.07	0.18	3782	-0.07	0.03	3735	0.09	0.46	415
rs12744696	-0.07	0.35	3777	-0.02	0.64	3730	-0.17	0.29	415
rs3792785	-0.18	0.02	3785	-0.04	0.45	3738	0.04	0.79	415
rs17133433	0.05	0.43	3714	-0.03	0.40	3670	-0.47	0.00	415
rs1637995	0.24	0.27	3785	0.04	0.74	3738	-0.57	0.03	415
rs8030871	-0.13	0.06	3782	-0.04	0.35	3735	0.11	0.49	415
rs858447	-0.10	0.20	3782	-0.03	0.55	3735	-0.15	0.33	415
rs312368	-0.06	0.28	3756	-0.00	0.90	3709	0.03	0.77	415
rs580962	-0.05	0.34	3785	0.00	0.90	3738	-0.05	0.65	415
rs206777	-0.05	0.32	3783	0.00	0.97	3736	-0.06	0.60	414
rs206767	-0.06	0.29	3784	-0.00	0.96	3737	-0.15	0.14	415
rs1832446	0.05	0.38	3785	0.01	0.86	3738	0.05	0.66	414
rs7738388	0.05	0.41	3780	0.01	0.73	3732	-0.02	0.87	415
rs2040840	-0.06	0.27	3783	-0.02	0.56	3736	0.06	0.57	414
rs12672266	-0.02	0.80	3784	-0.04	0.28	3737	0.08	0.59	415
rs2236426	0.00	0.96	3779	0.01	0.91	3732	0.08	0.60	415
rs897275	-0.05	0.35	3785	-0.01	0.69	3738	-0.13	0.19	415
rs1858367	0.13	0.26	3761	0.09	0.14	3714	0.03	0.86	415
rs2346115	-0.09	0.19	3785	-0.04	0.34	3738	-0.14	0.35	415
rs11847577	0.02	0.76	3785	-0.02	0.65	3738	0.10	0.43	415
rs2414167	-0.09	0.16	3783	-0.05	0.16	3736	0.05	0.73	415
rs333122	-0.01	0.81	3785	-0.00	0.95	3738	-0.15	0.19	415
rs6062340	-0.07	0.37	3780	-0.01	0.84	3733	-0.03	0.88	410

Figures

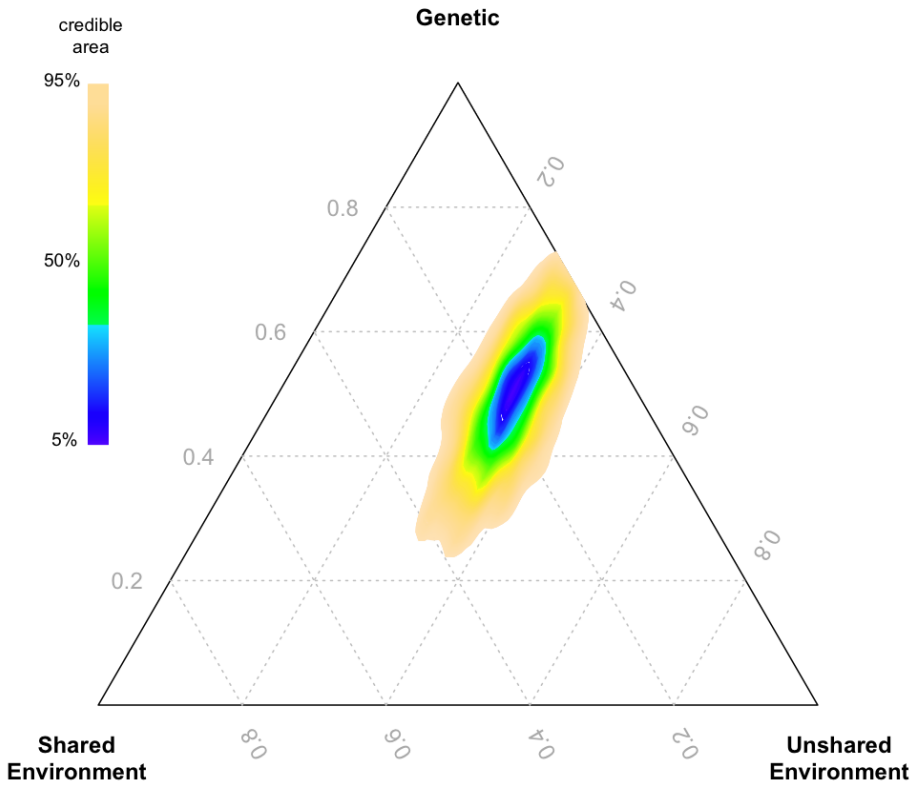


Figure 3.1: The heritability of validated turnout. Ternary plot shows the posterior distribution of estimated components of total variance in an ACE model of voting among MN citizens. The plot represents the proportions of three variance components (genetic, shared environment, and unshared environment) that sum up to one. A point in the center indicates all three components contribute equally, whereas a point at a vertex indicates that a single component fully explains the variance. Colors indicate credible areas calculated by using 2,000 posterior draws to estimate a three-dimensional kernel density. The blue areas indicate the highest density regions with the most credible estimates, while the beige areas contain 95% of the draws (i.e., the probability that the true estimates lie outside the colored region is 0.05. Mean heritability is estimated to be 49%.

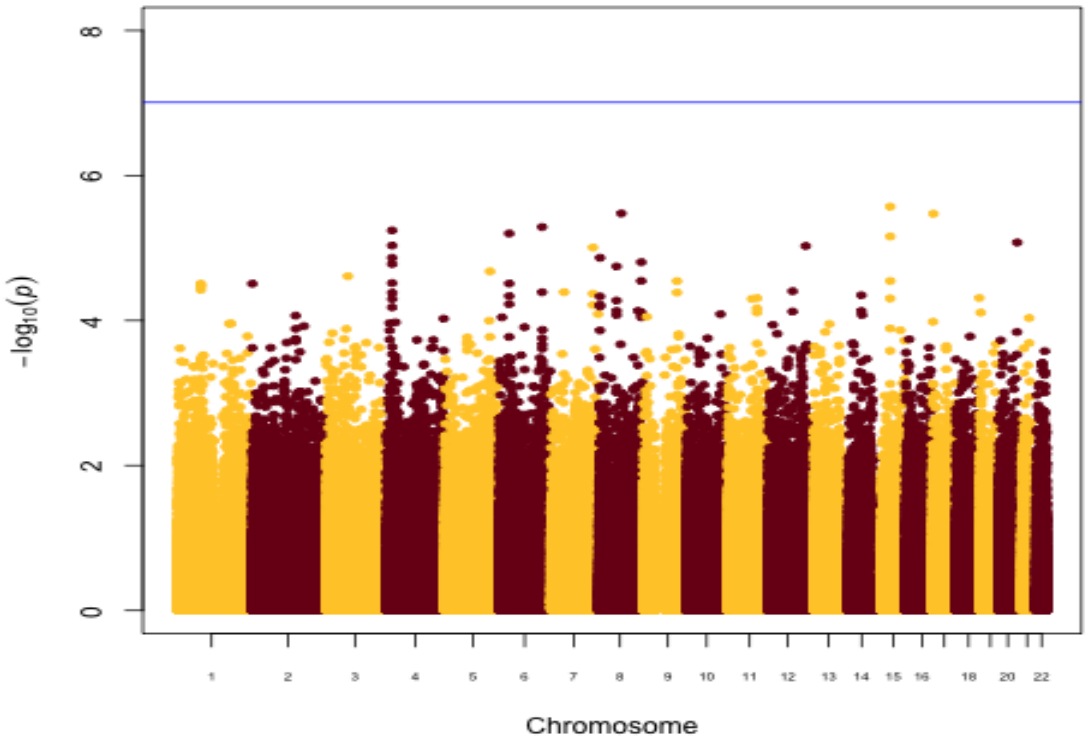


Figure 3.2: Manhattan plot of p-values associated with SNPs on autosomal chromosomes. P-values are represented as $-Log_{10}$. The blue line is the threshold for genome-wide significance based on the Bonferroni correction.

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