

COMT, Extraversion, and Partisan Attachment

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Abstract

A burgeoning literature focuses on the relationship between individual differences in personality traits and political behaviors (Gerber, Huber, Raso & Ha 2008, Gerber, Huber, Doherty & Dowling 2009, Mondak & Halperin 2008, Denny & Doyle 2008). Two recent papers found that extraversion is a strong predictor of whether an individual affiliates with a major political party as well as the strength of the affiliation (Gerber et al. 2008, Gerber et al. 2009). Extraversion (Bouchard & Loehlin 2001) and partisan attachment (Settle, Dawes & Fowler 2009, Hatemi, Hibbing, Alford, Martin & Eaves 2009) have been shown to be heritable and both have been linked to the neurotransmitter dopamine (Dawes & Fowler 2009, Munafo, Clark, Moore, Payne & Flint 2003, Carver & Miller 2006, Depue 1995). This paper tests the hypothesis that extraversion *mediates* a relationship between COMT and partisan attachment. COMT is a gene that regulates dopamine levels in the brain and that has previously been found to be associated with extraversion (Stein, Fallin, Schork & Gelernter 2005, Reuter & Hennig 2005). We find that individuals with a less efficient version of a well-studied variant of the COMT gene are significantly less likely to identify themselves as partisans than those with the more efficient allele. Further, extraversion partially mediates this association. This is the first such formal test of a pathway between genes, personality, and political behavior and suggests an empirical framework for future research.

Introduction

A burgeoning literature focuses on the relationship between individual differences in personality traits and political behaviors (Gerber et al. 2008, Gerber et al. 2009, Mondak & Halperin 2008, Denny & Doyle 2008). Two recent papers, Gerber et al. (2008) and Gerber et al. (2009) find that extraversion is a strong predictor of whether an individual affiliates with a major political party as well as the strength of that affiliation. In fact, Gerber et al. (2009) show, using a sample of more than 10,000 individuals, that the magnitude of the effect of extraversion is larger than education, age, income, and religious attendance.

Both personality traits and partisan attachment have a genetic basis. Behavior geneticists have long known that personality traits are highly 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 et al. 2005, Reuter & Hennig 2005). Likewise, the strength of partisan 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).

The next wave of research must test whether these distinct lines of research actually form two links in a causal chain. One possibility is that personality traits *mediate* the relationship between genes and political behavior. Research focussing on the link between personality traits and political behaviors suggest that since heritability of both personality traits and political behaviors are large, and the formation of personality traits predates political participation, it is likely to be the case

that genes influence personality which then affects political behavior (Gerber et al. 2009).¹ Another possibility is that the same gene or set of genes influences personality traits and political behaviors independently rather than sequentially. Verhulst, Hatemi & Eaves (2009) found this to be the case for personality and political attitudes. A test of these hypotheses requires a sample that includes individual-level genetic, personality, and political behavior information.

We examine a variant of 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% 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.

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

¹Heritability estimates for personality traits range from 41% to 61% (Bouchard & McGue 2003) and estimates for turnout (Fowler, Baker & Dawes 2008) and strength of partisan attachment (Settle, Dawes & Fowler 2009) are 53% and 46% respectively.

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 CCTA and from another be CTTA. In this example, the SNP (in bold) has two alleles, C and T. An illustration of this is provided in the appendix (*Figure 3*).

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.² 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 examining 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

²An 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).

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.³ 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 behaviors like extraversion (Munafo et al. 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

³Reuptake 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.

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 two 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. The second sample, referred to as the *replication sample*, 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. Subjects in both studies volunteered, were paid for their participation, and gave written consent. Both studies were approved by the University of California, San Diego Institutional Review Board. In addition, both studies asked participants to report race/ethnicity information and genotyped a majority sample for the COMT Val158Met polymorphism.⁴

In order to determine whether or not subjects are attached to a party, they were matched with public voting records available through the California Secretary of State's office.⁵ 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 replication sample included home address information. 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

⁴See Stein, Schork & Gelernter (2004) and Zhang, Rao, Wessel, Kennedy, Rana, Taupenot, Lillie, Cockburn, Schork, Ziegler & O'Connor (2004) for a detailed description of the respective samples.

⁵Voter records are as of June 2009.

Californians were registered to vote as of the November 2008 election.⁶

In the primary and replication samples respectively, 511 and 622 subjects have genotypic information for Val158Met. The dependent variable in this study is whether a registered voter is affiliated with a major political party, defined as being registered as either a Democrat or a Republican.⁷ Summary statistics for the sample are presented in the appendix *Table 3*. The replication sample is older, on average, and therefore has a higher percentage of partisans. Both samples are also made up primarily of females. The distribution of Val158Met are comparable to one another as well as to other published studies.

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

⁶This information come 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>).

⁷We refer to this throughout the paper as partisan attachment.

techniques regularly utilized in the literature to attempt to ameliorate the problem of population stratification. A typical way to control for this problem is to include controls for the self-reported race of the subject. Another 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, 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. Tests of association can then be conditional on subpopulation assignment.

Results

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

$$\text{logit}(P[\textit{Partisanattachment}_i = 1]) = \tau + \beta_{\textit{Met}}\textit{Met}_i + \beta_{\textit{Age}}\textit{Age}_i + \beta_{\textit{Male}}\textit{Male} + \beta_Z Z_i \quad (1)$$

where i indexes the subject, \textit{Met} is the number of COMT Met alleles (0,1,or 2), \textit{Age} is age in 2009, and \textit{Male} is an indicator variable for whether the subject is male (1) or female (0). To guard against population stratification, we used 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).⁸ STRUCTURE adopts a Bayesian cluster approach to find the optimal

⁸The marker panel includes D8S1179, D21S11, D7S820, CSF1PO, D3S1358, TH01, D13S317, D16S539, D2S1338, D19S443, vWA, TPOX, D18S51, D5S818, FGA, amelogenin, D1S196, D1S2628, D2S162, D2S319, D5S407, D5S410,

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). Z is a vector of the subpopulation assignments.

Since the replication sample is made up of nuclear families, and thus the observations are correlated, we estimate the logit model in *Equation 1* using general estimating equations (GEE) (Liang & Zeger 1986). GEE models require the specification of the within-family correlation structure which in our case is known based on genetic theory. We can explicitly model the correlation for each pair of individuals (i and i') within a family as (see Medland & Hatemi (2009) for a primer on biometric theory and modeling geared for political scientists):

$$\rho_{i,i'} = R_{i,i'}a^2 + \gamma_{i,i'}c^2 \tag{2}$$

where R is the degree of genetic relatedness, equalling 1 for monozygotic twin pairs and 0.5 for both dizygotic twin and parent-child pairs, and γ is the degree to which pairs share a common environment, assumed to be equal to 1 for siblings and 0 for parent-child pairs. The three parameters a^2 , c^2 , e^2 are the standardized variances of the latent genetic, common environment, and unique environment components respectively. The most common approach to estimating a^2 , c^2 , and e^2 is to employ structural equations modeling within a maximum likelihood framework based a sample of

D6S1610, D7S640, D7S657, D8S272, D8S1827, D9S175, D10S197, D10S1786, D11S935, D12S352, D14S68, D15S1002, D16S3017, D17S799, D22S274.

monozygotic and same-sex dizygotic twins (Medland & Hatemi 2009). Since the replication sample includes 115 monozygotic and 44 dizygotic complete same-sex twin pairs, this is the approach we take.⁹

The estimates are $a^2 = 0.52$ (0.26, 0.78), $c^2 = 0.00$ (0.00, 0.02), and $e^2 = 0.48$ (0.22, 0.74).¹⁰ Plugging the estimates into *Equation 2* yields a correlations between monozygotic twins of 0.52 and a correlation between non-monozygotic siblings, as well as between parents and their children, of 0.26. The GEE model is estimated using a “fixed” working correlation structure where the correlation between individuals is either 0.56 or 0.26 depending on the genetic relatedness between pairs.¹¹

Subpopulation assignments based on the structured association method are not available for the replication sample, therefore we control for the effects of the underlying population structure by including a vector of indicator variables (Z) for whether a subject self-reported as Black, Asian, Hispanic, Native American, Filipino, or Other (omitted category is White).

Table 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. Both samples show the *Met* allele is significantly associated with partisan attachment ($p = 0.002$ and $p = 0.05$).¹² The *Met* allele remained significant ($p = 0.002$) in the primary model with the addition of SES measures.¹³ *Figure 1* presents the simulated first differences for the primary model. Holding the

⁹The components of the ACE model are estimated using Mplus (Muthen & Muthen 2007) and controls are included for sex and age.

¹⁰These estimates are very similar to those reported for strength of partisan attachment by Settle, Dawes & Fowler (2009)

¹¹Using only the parameter estimate for a^2 does not account for the uncertainty associated with the estimate. Therefore, we drew 1000 estimates of a^2 from a $N \sim (0.521, 0.133)$, constructed the correlation matrix given each estimate, and ran the GEE regression. The mean p-value from this process was 0.05.

¹²The results are similar for the primary model when self-reported race is used instead of the subpopulation assignments. The parameter estimate on *Met* is -0.42 ($p = 0.004$).

¹³The measures include family income and employment status at the time of participation in the original study.

	Primary Sample			Replication Sample		
	Coef	SE	P Value	Coef	SE	P Value
Intercept	0.39	0.88	0.66	0.41	0.35	0.23
Met	-0.43	0.14	0.00	-0.29	0.15	0.05
Age	0.03	0.03	0.42	0.03	0.01	0.00
Male	-0.28	0.21	0.18	-0.39	0.20	0.05
SP 1	0.25	0.29	0.40			
SP 2	-0.38	0.39	0.32			
Black				-0.09	0.43	0.83
Asian				-0.40	0.77	0.60
Hispanic				0.97	0.43	0.02
Native American				-1.14	1.88	0.58
Filipino				0.01	0.20	0.98
Other				-1.14	1.11	0.30
<i>N</i>		510			622	
Wald Test of Model Fit (χ^2)		12.8			36.8	
Wald Test Degrees of Freedom		5			9	
Wald Test P value		0.00			0.03	

Table 1: Test of association between COMT Val158Met SNP and partisan attachment. The primary model is a simple logit and the replication model is a GEE logit with a “fixed” working correlation based on *Equation 2*. The dichotomous dependent variable is *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), *SP* is the subpopulation assignment, and *Black*, *Asian*, *Hispanic*, *Native American*, *Filipino*, and *Other* are self-reported race categories. Logit coefficients (Coef), standard errors (SE), and p-Values are presented.

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.

Mediation

We find a highly significant association between the Val158Met SNP and partisan attachment based on the primary sample and are able to replicate this finding in another distinct sample. The next

These results are not presented since they are nearly identical.

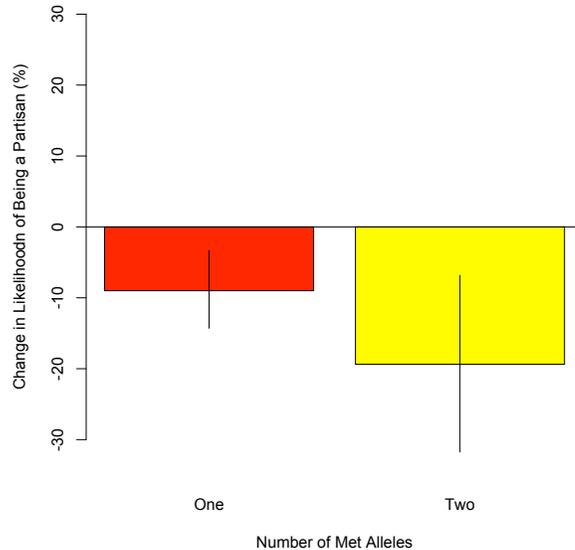


Figure 1: Changing the number of Met alleles yields significantly lower partisan attachment. First differences, based on simulations of *Table 1* (primary) parameters, are presented along with 95% confidence intervals. All other variables in the model are held at their means.

step, suggested by the findings of Gerber et al. (2008) and Gerber et al. (2009) that extraversion is a strong predictor of partisan attachment, is to formally test whether effect of this SNP is mediated by the personality trait extraversion. 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?

To formally test for mediation, we followed the steps laid out by 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 (1) X significantly predicts M and (2) M significantly predicts Y controlling for X (Baron & Kenny 1986). This relationship is illustrated in *Figure 2*.

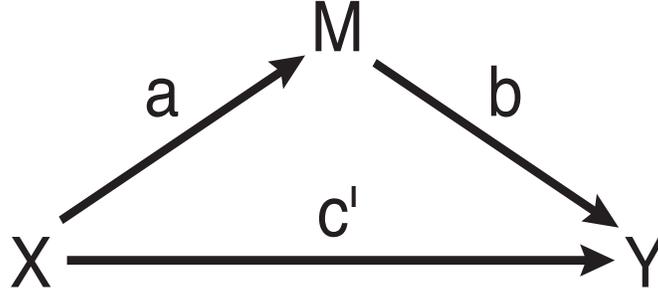


Figure 2: The mediation relationship described by Baron and Kenny (1986). Path c' is called the direct effect and the product of paths a and b is the indirect effect.

A test of whether extraversion mediates the association found between Val158Met and partisan attachment can be represented in the context of this analysis by the following two equations:¹⁴

$$Extraversion_i = \beta_0 + \beta_{Met}Met_i + \beta_{Age}Age_i + \beta_{Male}Male_i \quad (3)$$

$$\text{logit}(P[Partisan_i = 1]) = \delta_0 + \delta_{Met}Met_i + \delta_{Extraversion}Extraversion_i + \beta_{Age}Age_i + \beta_{Male}Male_i \quad (4)$$

The indirect effect $ab = \beta_{Met}\delta_{Extraversion}$, the direct effect $c' = \delta_{Met}$, and the total effect is equal to $ab + c'$. The size of the mediated effect in relation to the total effect can be calculated by $\frac{|ab|}{|ab|+|c'|}$.

The parameter estimates from equations (3) and (4) are presented in *Table 2*.

The results in *Table 2* are consistent with both criteria laid out by Baron & Kenny (1986). The Met allele significantly predicts extraversion ($p = 0.01$) in *Model 3* and extraversion is significant ($p = 0.04$) in a model of partisan attachment (*Model 4*) even when the Met allele is included. The Met allele remains significant in *Model 4* suggesting partial mediation. However, these results are only suggestive. To formally test for mediation, ab must be estimated. Since ab is not necessarily

¹⁴The vector of the subpopulation assignments (Z) has been suppressed for presentation purposes. Subpopulation assignments are included as controls in both models.

	<i>Extraversion</i> (3)			<i>Partisanship</i> (4)		
	Coef	SE	P Value	Coef	SE	P Value
Intercept	46.4	5.3	0.00	-0.56	1.02	0.58
Met	-1.96	0.74	0.01	-0.39	0.14	0.01
Extraversion				0.02	0.01	0.04
Age	0.10	0.28	0.71	0.03	0.03	0.42
Male	3.58	1.12	0.00	-0.36	0.22	0.10
SP 1	1.98	1.51	0.19	0.21	0.30	0.49
SP 2	1.39	2.04	0.50	-0.37	0.39	0.35
<i>N</i>	496			496		
Wald Test of Model Fit (χ^2)	20.1			15.4		
Wald Test Degrees of Freedom	5			6		
Wald Test P value	0.00			0.02		

Table 2: Test of association between COMT Val158Met SNP and both partisan attachment and extraversion. The partisan attachment model is a simple logit 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. *SP* is the subpopulation assignment. Logit and linear coefficients (Coef), standard errors (SE), and p-values are presented.

normally distributed (Bollen & Stine 1990, Lockwood & MacKinnon 1998, Mackinnon, Lockwood, Hoffman & West 2002, Shrout & Bolger 2002), the approach most researchers take is to bootstrap the estimated indirect effect and construct confidence intervals based on the bootstrapped values (Bollen & Stine 1990, Shrout & Bolger 2002). If ab is bounded away from zero the indirect effect can be considered significant.

To estimate the indirect 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 2009). We employed the Baron & Kenny (1986) parametric approach with quasi-Bayesian bootstrap to produce confidence intervals based on 1000 simulations.¹⁵ We found a significant mediated effect ($ab = -0.0077$, 95 % CI $-0.0185, -0.0002$) and 8% (95 % CI 5%, 22%) of the effect of the Met

¹⁵A nonparametric bootstrap produces similar results.

allele on partisan attachment is via the personality trait extraversion.¹⁶ The test of mediation was only performed on the primary sample because the replication sample lacks measures of personality traits.

Discussion

We show 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. Further, this association was replicated in a different sample of California voters. 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.

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 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.

We feel this paper makes three important contributions. Our main contribution is to con-

¹⁶The results are nearly identical when family income and employment status, both asked at the time of participation in the original study, are included in the model of partisan attachment. The proportion mediated is 8% (95% CI 5%, 21%).

duct the first formal test of a specific pathway from genes to partisan attachment. Another contribution is our presentation of the analytical framework necessary to explore the complex pathways that underly 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. Finally, a related contribution is our demonstration that proportion mediated by extraversion is relatively modest. This suggests, especially given the fact the Met allele remains significant in *Model4*, 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.

This study has a few 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 is technically party registration rather than party identification. However, our party registration variable is identical to the one used by (Gerber et al. 2008) in their analysis of Connecticut voters that produced results very similar to a standard measure of strength of party identification (Gerber et al. 2009). Finally, genetic analyses are vulnerable to producing a spurious association due to population stratification. We were able to replicate our association result but

did not have the necessary data to test for mediation in our replication sample. Therefore, even though we controlled for population stratification by including subpopulation assignments based on 36 ancestry-informative genetic markers, we urge caution in interpreting our mediation results until they can be replicated in another sample.

Appendix

Sample Statistics

	Primary	Replication
Val / Val	0.32	0.25
Val / Met	0.50	0.49
Met / Met	0.18	0.26
Partisan	0.64	0.79
Age (mean)	24.21	49.50
Age (sd)	3.17	17.50
Male	0.26	0.31
Black	0.03	0.07
Asian	0.05	0.03
Hispanic	0.19	0.11
Native American	0.00	0.00
Filipino	0.08	0.01
White	0.57	0.77
Other	0.07	0.00
<i>N</i>	510	622
<i>N</i> with Extraversion	496	n/a

Table 3: Sample frequencies, means, and standard deviations.

Single Nucleotide Polymorphism

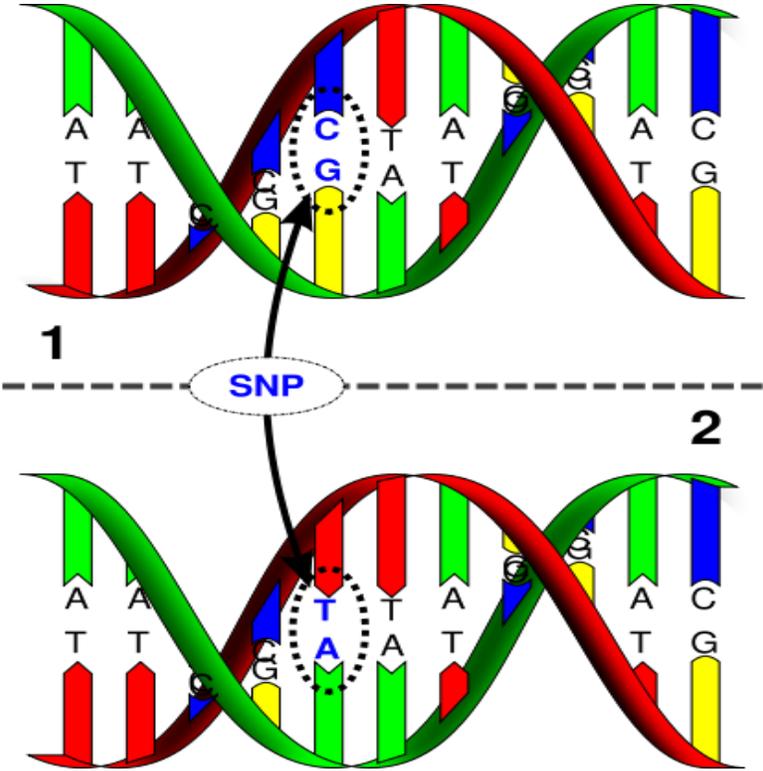


Figure 3: Single Nucleotide Polymorphism

The ACE Model

In order to estimate the degree to which partisan attachment is heritable, we study the partisan attachment of (identical) monozygotic (MZ) twins who were conceived from a single egg and (non-identical) dizygotic (DZ) twins who were conceived from two separate eggs. MZ twins share 100% of their genes, while DZ twins share only 50% on average. Thus, if partisan attachment has a genetic basis, MZ twins should exhibit more concordance (both twins vote or both twins abstain) than DZ twins. Moreover, if we assume that MZ twins and DZ twins share comparable environments, then we can use these concordances to estimate explicitly the relative influence of genetic, shared environmental, and unshared environmental factors.

The basic 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 unshared environmental (E). This is the so-called ACE model. Common environment includes the family environment in which both twins were raised, as well as any other factor to which both twins were equally exposed. In contrast, the unshared environment includes idiosyncratic influences that are experienced individually.

The purpose of the ACE model is to measure the total variance in a given phenotype (in our case, partisan attachment) and then estimate the extent to which genetic and environmental influences separately contribute to the total observed variance. The role of genotype and environment are not measured directly but their influence is inferred through their effects on the covariances of twin siblings (?). More formally, these components are derived from known relationships between three observed statistics (?):

$$\sigma_P^2 = \sigma_A^2 + \sigma_C^2 + \sigma_E^2 \tag{5}$$

$$COV_{MZ} = \sigma_A^2 + \sigma_C^2 \quad (6)$$

$$COV_{DZ} = \frac{1}{2}\sigma_A^2 + \sigma_C^2 \quad (7)$$

where σ_P^2 is the observed phenotypic variance (the same for MZ and DZ twins), COV_{MZ} and COV_{DZ} are the observed covariances between MZ and DZ twins, and σ_A^2 , σ_C^2 , and σ_E^2 are the variance components for genes, common environment, and unshared environment, respectively.

These relationships yield three equations and three unknowns thus :

$$\begin{pmatrix} a^2 \\ c^2 \\ e^2 \end{pmatrix} = \begin{pmatrix} 1 & 1 & 0 \\ \frac{1}{2} & 1 & 0 \\ 1 & 1 & 1 \end{pmatrix}^{-1} \begin{pmatrix} COV_{MZ} \\ COV_{DZ} \\ \sigma_P^2 \end{pmatrix}$$

where the variance components are standardized by dividing by the total phenotypic variance ($a = \frac{\sigma_A^2}{\sigma_P^2}$, $c = \frac{\sigma_C^2}{\sigma_P^2}$, $e = \frac{\sigma_E^2}{\sigma_P^2}$).

The structural equation specification of our model is:

$$\sigma_{Partisan}^2 = a^2 + c^2 + e^2 \quad (8)$$

$$COV_{MZ} = a^2 + c^2 \quad (9)$$

$$COV_{DZ} = \frac{1}{2}a^2 + c^2 \quad (10)$$

Since partisan attachment is a dichotomous variable, the model assumes that it has an underlying normal latent distribution and we only observe two distinct values (0,1). The observed

binary value is transformed into the continuous latent variable using a probit function based some threshold below which we observe no partisan attachment and above which we observe attachment, and the variance. A probit model does not allow both the threshold and variance to be estimated, therefore the variance is fixed to 1. This means that rather than estimating e^2 , it is solved for by $1 - a^2 - c^2$ based on *Equation 8*.

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