The CHRNA6 Gene, Patience, and Voter Turnout

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Abstract

Political scientists have recently explored the genetic basis of political participation. Fowler, Baker & Dawes (2008) recently showed in two independent samples of twins that voter turnout is heritable and Fowler & Dawes (2008) identified two specific genes associated with turnout. Earlier work (Fowler & Kam 2006) has demonstrated a link between patience and turnout. We combine these insights by investigating two genes that are associated with patience and may explain variation in voter turnout. Using data from the National Longitudinal Study of Adolescent Health, we show that individuals with a version of the CHRNA6 gene are significantly less likely to have voted in the 2000 presidential election. We hypothesize that the association between the CHRNA6 gene and voting is mediated by patience.

Introduction

Voting requires citizens to incur costs in the short term in exchange for an expected benefit in the future. Citizens need to devote at least minimal attention to politics, make a decision between the parties or candidates on offer, and incur the costs of going to the polls. These costs are all paid before an election. By contrast, the non-expressive benefits of voting in an election, conferred to oneself or others, are only realized in the period after an election. Given this tradeoff, voting may be an act for the patient (Fowler & Kam 2006). This article draws on the intertermporal aspect of voting to demonstrate a direct link between genes related to impulsivity and the decision to vote. In doing so, it furthers our understanding of the etiology of political participation.

Recent work has demonstrated that variation in political participation can be attributed to genes. Using twin studies, Fowler, Baker & Dawes (2008) demonstrated that voter participation in two different samples suggested heritability of approximately 50%. Following up on this finding, Fowler & Dawes (2008) identified two genes, MAOA and 5HTT, that are associated with voter turnout. Additional research has also shown that political attitudes (Alford, Funk & Hibbing 2005) and partisan attachment (Settle, Dawes & Fowler 2009, Hatemi, Hibbing, Alford, Martin & Eaves 2009, Dawes & Fowler 2009), both closely related to turnout, also have a genetic basis.

One potential pathway linking genes to voter turnout is via the trait impulsivity. Like turnout, impulsivity is heritable (Jang, McCrae, Angleitner, Riemann & Livesley 1998,

Bouchard & McGue 2003) and is also a significant predictor of turnout behavior (Fowler & Kam 2006). This article tests for an association between two variants of the CHRNA6 gene and two of the CHRNB3 gene, both previously found to be associated with impulsive behaviors, and voter turnout. Based on the relationship between these genes and a strong preference for immediate over delayed gratification, we hypothesize they will be associated with a decreased likelihood of voting.

Our paper proceeds as follows. We review the theoretical case for the relationship between patience or impulsivity and the decision to vote. We then review previous findings related to CHRNA6 and related genes, paying particular attention to the behavioral and psychological correlates of these genes. Following this, we review our data, the basics of genetic association studies, and our research design. We then present our results, followed by a discussion of these findings.

Impulsivity, Patience and Turnout

We define impulsivity as an unwillingness or inability to forgo immediate gratification in exchange for greater reward at a later time. Our conception of impulsivity is interchangeable with that of patience where patience is defined as a "time preference" gauging the trade-off between present and future consumption (Becker & Mulligan 1997). Studies in psychology have demonstrated that impulsivity exists very early in life (Mischel, Shoda & Rodriguez 1989), is stable (Mischel, Shoda & Peake 1988, Funder, Block & Block 1983), and is related to other traits such as intelligence (Mischel & Metzner 1962) and social responsi-

bility (Mischel & Gilligan 1964, Stumphauzer 1972). Moreover, this trait has implications throughout the life-cycle. For example, individual-level impulsivity has been correlated to lifetime earnings, investment and saving, smoking, alcohol and other substance abuse, gambling, and educational attainment (Funder, Block & Block 1983, Mischel 1974).

How is impulsivity or patience related to voting? The classic calculus of voting suggests that the decision to vote depends on the expected benefits to be received from a vote less the cost of voting, or $PB - C$, where P is the probability of being decisive, B are the benefits to be received by the desired election outcome, and C is the cost of voting. While the costs of voting, such as learning the issues and going to the polls, are paid on or before election day, the benefits of having a policy implemented usually take much longer to realize. Therefore, the rational choice calculation $PB - C$ is largely a comparison of present costs and future benefits. More patient individuals are likely more willing to pay the present costs even though they do not gain the benefits immediately. Impulsive individuals, on the other hand, do not tend to delay gratification and thus are more likely to heavily weight the present costs over future benefits. Citizens voting to maintain the democratic form of governance must also take a longer view. Likewise, citizens with social preferences (Fowler 2006, Loewen 2010, Dawes, Loewen & Fowler 2009, Edlin, Gelman & Kaplan 2007) trade off present costs for future benefits to other citizens. In short, the importance of patience applies with equal felicity to a variety of theoretical explanations of the decision to vote.

In addition to the theoretical plausibility of this relationship, empirical evidence also

exists. Fowler & Kam (2006) present results supporting the argument that more patient individuals are more likely to vote. The authors elicited patience in a laboratory setting by administering what is often referred to as a delay discounting task where subjects are asked questions of the form "Would you prefer to have $10 today or $15 in one month?" The amounts of the immediate rewards and duration of the delay are successively modified and subject choices can be used to derive their discounting function. Fowler and Kam found that those more willing to wait for a higher payoff are also more likely to report having voted. Less direct evidence is provided by Gerber, Huber, Raso & Ha (2008), who explore the association of "Big Five" personality traits with voter participation. Following John & Srivastava (1999), they note that conscientiousness is related to impulse control and task and goal-oriented behavior. They find that this trait is weakly related to self-reported turnout. Moreover, in keeping with Whiteside & Lynam (2001)'s conception of impulsivity as positively related to emotional stability, they also find a relationship between this stability and voter participation.[1]

In this article, we examine two genes that have been previously associated with impulsive behavior, CHRNA6 and CHRNB3. We provide a brief general overview of genetic concepts followed by a review of previous research on these genes, our data, and our research design.

[1]By contrast, Denny & Doyle (2008) find a negative but insignificant relationship between impulsivity, measured at age 16, and turnout among British citizens 23 years later.

CHRNA6 and CHRNB3 Genes

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. *Receptors* on the sending and target neurons play a critical role in this process. 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. The release of these neurotransmitters is modulated by receptors located on the sending neuron. In both cases, the binding of neurotransmitters to receptors cause changes in the receptor's metabolic activity that facilitates neurotransmission.

Nicotine acetylcholine receptors (nAChRs) are located throughout the central nervous system and are presynaptic and postsynaptic (Dani & Bertrand 2006). Nicotine acetylcholine receptors are triggered by the neurotransmitter acetylcholine (ACh) but are also triggered by nicotine. When either ACh or nicotine bind to the receptor it causes a pore in the receptor's membrane to open allowing ions to flow into and out of the cell. After a few milliseconds the ion channel closes again and remains closed while at rest. Prolonged exposure to ACh or nicotine results in the nAChR becoming desensitized and as a result remains closed and unresponsive to ACh or nicotine binding (Dani & Bertrand 2006, Meyer, Yoshikami & McIntosh 2008). Nicotine acetylcholine receptors are made up of a combination of two different protein subunits, α and β. There are 8 different α (α2 - α7, α9 - α10) and

four different β subunits (β2 - β4) (Dani & Bertrand 2006). The triggering, closure, and desensitization of nAChRs are each influenced by the α and β subunits of which they are comprised (Dani & Bertrand 2006).

Researchers studying impulsive behavior have focussed on presynaptic nAChR receptors, and the genes that code for them, because they have been shown to modulate dopamine release in the midbrain (Wonnacott 1997). Dopamine in the substantia nigra compacta (SNc) plays a critical role in positive reinforcement associated with learned behavior as well as maintaining established stimulus response habits (Meyer, Yoshikami & McIntosh 2008). The SNc also supplies dopamine to the striatum, an area of the brain that has been demonstrated to be highly active during laboratory tests of delay discounting (Hariri, Brown, Williamson, Flory, de Wit & Manuck 2006).

Two particular subunits that have been shown to influence dopamine release are α6 and β3 (Cui, Booker, Allen, Grady, Whiteaker, Marks, Salminen, Tritto, Butt, Allen, Stitzel, McIntosh, Boulter, Collins & Heinemann 2003, Meyer, Yoshikami & McIntosh 2008). The CHRNA6 gene codes for α6 receptor subunits and the CHRNB3 gene codes for β3 subunits. CHRNA6 and CHRNB3 are associated with impulsive behaviors like nicotine initiation and dependence (Li et al 2005, Feng et al. 2004, Hoft et al 2009b, Schlaepfer et al 2008) and alcohol abuse (Hoft et al 2009a). These behaviors are considered to be impulsive because they privilege immediate small rewards over long-term health benefits. These behaviors are in turn highly correlated with impulsivity as measured by the delay discount task (Madden, Petry, Badger & Bickel 1997, Bickel, Odum & Madden 1999).

Taken together, these results suggest that such genes play an important role in the regulation of impulsive behavior precisely because of the central role of the dopamine system in the mediation of pleasurable rewards (Schlaepfer, Hoft & Ehringer 2008). Related evidence from other genes in the dopamine system provides similar evidence. Given the plausibility of this relationship between nAChRs, impulsivity, and turnout, we set out to explore the relationship between four variants on two nAChRs and voter turnout. We present these results after reviewing our sample and research design.

Add Health Sample

The National Longitudinal Study of Adolescent Health (Add Health) is a nationally representative study that explores the causes of health-related behavior of adolescents in grades 7 through 12 and their outcomes in young adulthood.[2] The first wave of the Add Health study was collected in 1994-1995 when subjects were between 11 and 19 years old, the second wave in 1996, and the third wave in 2001-2002 when subjects were between 18 and 26 years old. The third wave was made up of 15,170 of the original Wave I participants.

In Wave I of the Add Health study, researchers created a genetically informative sample of sibling pairs. These pairs include all adolescents that were identified as twin pairs, siblings, half siblings, or unrelated siblings raised together. The Wave I sibling-pairs sample has been found to be similar in demographic composition to the full Add Health

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

sample (Jacobson & Rowe 1998). Genetic markers were typed for 2,574 individuals from the genetically informative sample as part of Wave III including four nAChR single nucleotide polymorphisms (SNPs).[3] These SNPs are rs2304297 and rs892413 on CHRNA6 as well as rs4950 and rs13280604 on CHRNB3. Subjects also answered "Did you vote in the most recent [2000] presidential election?"[4] Subjects also answered additional questions about their political preferences and participation as well as socioeconomic status.

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 an allele is lower among voters than abstainers. A significant association can mean that the allele itself influences turnout 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 the number of voters would lead to a spurious association.

There are two main research designs employed in association studies: *case-control*

[3] See the appendix for a brief discussion of genetic concepts, including single nucleotide polymorphisms.

[4] A limitation of self-reported turnout is that it is often inflated. However, Fowler, Baker & Dawes (2008) show similar heritability estimates for both validated and self-reported turnout.

designs and *family-based* designs. Case-control designs compare the frequency of alleles or genotypes among subjects that exhibit a trait of interest to subjects who do not. As a result, they are vulnerable to population stratification. A typical way to control for this problem is to include controls for the race / ethnicity of the subject or to limit the analysis to a specific racial or ethnic group. Family-based designs eliminate the problem of population stratification by using family members, such as parents or siblings, as controls. Tests using family data compare whether offspring exhibiting the trait receive a risk allele from their parents more often than would be expected by chance.[5] A major limitation of family-based studies is that they tend to be underpowered, thus prone to Type I error (Xu & Shete 2006). In this study we employ both case-control and family-based designs.

Case-Control Results

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

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

where i and j index subject and family respectively, X is the number of SNP alleles (0,1,or 2), Z is a matrix of variables to control for underlying population structure of the Add Health samples as well as other variables that may influence voter turnout, U is a family

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

random effect that takes into account the fact the observations are not independent because siblings come from the same family. The random effect controls for genetic and environmental correlation among family members. For rs2304297 (SNP1), rs4950 (SNP3), and rs13280604 (SNP4) X is the number of G alleles and for rs892413 (SNP2) it is the number of C alleles. The allele frequencies for each SNP are presented in the appendix.

To control the effects of the underlying population structure, we use indicator variables for whether a subject self-reported as Black, Hispanic, Asian, or Native American (omitted category is White). Following the policy of the United States Census, Add Health allows respondents to mark more than one race. In our sample, 108 subjects chose 2 races, 9 subjects chose 3 races, and 37 subjects chose no race. Those 117 subjects choosing more than one race were omitted from the analysis to limit the risk of population stratification. We also re-run the analysis restricting the sample to only white non-hispanic respondents (58% of the sample).

Table 1 presents the results for the test of association between the four SNPs and turnout. This baseline model includes controls for age, gender, and race. The odds ratio of the allele parameter estimate is an individual's odds of voting if he or she has one allele compared to having no alleles (or having two alleles compared to one allele). A significant odds ratio implies that the CHRNA6 or CHRNB3 genes are associated with turnout. *Table 1* shows that the two CHRNA6 SNPs are significant at conventional levels ($p = 0.01$ and $p = 0.04$), however only the CHRNA6 SNP rs2304297 is significant after correcting for multiple testing. A Bonferroni correction for multiple testing requires the corrected significance level

	SNP1		*SNP2*		*SNP3*		*SNP4*	
	OR	P value	OR	P value	OR	P value	OR	P value
SNP1: G	**0.79**	**0.01**						
SNP2: C			0.84	0.04				
SNP3: G					0.88	0.11		
SNP4: G							0.89	0.15
Age	1.13	0.00	1.12	0.00	1.11	0.00	1.13	0.00
Male	1.07	0.51	1.03	0.78	1.06	0.55	1.08	0.46
Black	2.06	0.00	1.86	0.00	1.66	0.00	1.77	0.00
Asian	0.73	0.16	0.82	0.37	0.86	0.48	0.78	0.27
Native American	0.87	0.71	0.84	0.64	0.78	0.49	0.90	0.77
Hispanic	0.83	0.30	0.81	0.22	0.78	0.15	0.80	0.19
Intercept	0.06	0.00	0.07	0.00	0.08	0.00	0.06	0.00
N	2117		2193		2175		2068	
Deviance	2850		2956		2943		2799	
Deviance(*constant*)	2886		2987		2969		2830	

Table 1: Models of association between CHRNA6 and CHRNB3 SNPs and voter turnout. All results are expressed in odds ratios (OR). The model is a mixed-effects logit which estimates a random intercept for each family (not shown). Detailed variable descriptions are provided in the appendix.

to be equal to the original significance threshold divided by the number of tests. In our case $0.05/4 = 0.0125$.[6]

In *Table 2* we further test the association between rs2304297 and turnout. First, to try and minimize the risk of population stratification we restrict the analysis to self-reported whites. The association remains significant ($p < 0.01$). Second, we include a number of factors previous studies have found to influence turnout in the baseline model.[7] These include partisanship (Bartels 2000), cognitive ability[8] (Deary, Batty & Gale 2008, Denny

[6]Uncorrected p-values are presented in the *Table 1*.

[7]See Blais (2000) for a review

[8]We use the Picture Vocabulary Test (PVT) administered by Add Health which is thought to be a good measure of verbal IQ (Rowe, Jacobson & Van den Oord 1999)

	White Only		*with Controls*	
	OR	P value	OR	P value
SNP1: G	**0.73**	**0.01**	**0.75**	**0.01**
Age	1.10	0.01	1.10	0.01
Male	1.20	0.16	1.14	0.26
Partisan			4.33	0.00
Attendance			1.22	0.00
Cognitive			1.01	0.00
Income			1.00	0.99
Black			2.28	0.00
Asian			1.00	1.00
Native Am			0.97	0.90
Hispanic			1.09	0.67
Intercept	0.10	0.01	0.02	0.00
N	1340		1972	
$Deviance$	1807		2372	
$Deviance(constant)$	1822		2690	

Table 2: Models of association between CHRNA6 SNP and voter turnout. All results are expressed in odds ratios (OR). The model is a mixed-effects logit which estimates a random intercept for each family (not shown). Detailed variable descriptions are provided in the appendix.

& Doyle 2008), church attendance (Verba, Schlozman & Brady 1995), and income (Verba, Schlozman & Brady 1995). Consistent with previous findings nearly all of these controls are significant, however the rs2304297 SNP remains significant even after their inclusion in the model.

Finally, *Figure 1* presents the simulated first differences for the rs2304297 model in *Table 1*. Holding the control variables at their means and changing the number of G alleles from zero to one decreases average turnout by about 5 percentage points and from zero to two by about 10 percentage points. Both simulated first differences are significantly different from zero.

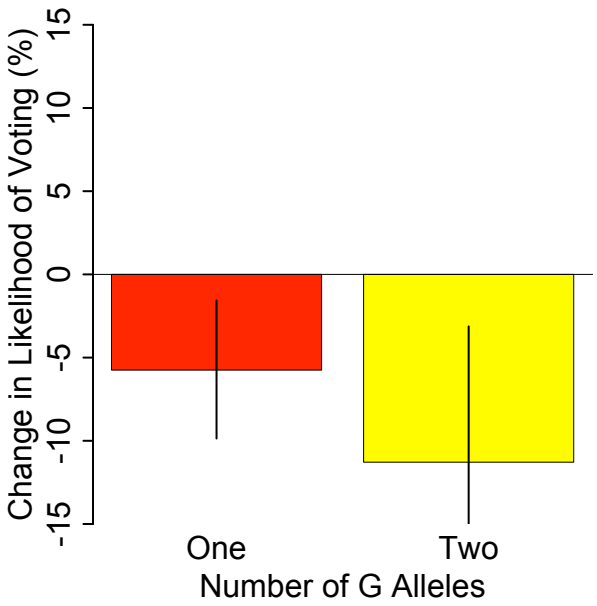

Figure 1: Changing the number of G alleles yields significantly lower turnout. First differences, based on simulations of *Table 1* (baseline) parameters, are presented along with 95% confidence intervals. All other variables in the model are held at their means.

Family-Based Results

In order to ensure population stratification is not driving our results, we also use a family-based test of the association between the G allele of rs2304297 and turnout. Spielman & Ewens (1998) constructed a sibling-based test of association, known as the sib TDT, for binary traits. In this test, sibling members of a nuclear family are compared with one another to determine whether the allele frequency among those "affected" siblings is significantly different from "unaffected siblings" (voters and abstainers in our case). For the sib TDT to be a valid test of association, sibships must be made up of exactly two siblings, one

affected and one unaffected, with different genotypes.[9] If there is no association, which is the null hypothesis, then each genotype is equally likely for affected and unaffected sibs in these sibships (Spielman & Ewens 1998). Based on this criteria, we have 82 sibships (164 individuals) in our sample.

The sib TDT supports the finding of an association between the G allele and turnout ($\chi^2 = 4.26$, $p = 0.04$) that was found using the case-control approach.[10] However, the simple sib TDT does not allow the inclusion of potentially relevant covariates like age and gender. An alternative approach is to model the sib TDT using a retrospective logistic regression model (Waldman, Robinson & Rowe 1999, Zou 2006). *Table 3* shows that the logistic sib TDT also yields a significant association with the inclusion of age and gender as covariates ($p = 0.03$).

	Retrospective Logit	
	OR	P value
Vote	0.62	0.03
Age	1.07	0.34
Male	0.85	0.48
Intercept	0.23	0.33
N		328
LL		441
$LL(constant)$		446

Table 3: Family-based test of association between CHRNA6 and turnout. The model is a mixed-effects logit which estimates a random intercept for each family (not shown). The dichotomous dependent variable is whether or not the kth allele ($k = 1, 2$) is an G allele [G allele = 1, G allele = 0]. Odds ratios (OR) are presented. Detailed variable descriptions are provided in the appendix.

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

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

In summary, the case-control test shows a significant association between the rs2304297 SNP and turnout even after accounting for the fact we conducted four tests. This finding is confirmed by a family-based test that is designed to protect against population stratification.

Discussion

This study found that a gene variant that codes for the $\alpha 6$ subunit of the nicotine acetylcholine receptor is significantly associated with self-reported turnout. This gene is also associated with impulsive behaviors like nicotine addiction (Li et al 2005, Feng et al. 2004, Hoft et al 2009b, Schlaepfer et al 2008) and alcohol abuse (Hoft et al 2009a). Further, this gene is known to regulate dopamine release in areas of the brain that are activated during participation in laboratory experiments designed to measure impulsivity (Hariri et al. 2006). Finally, impulsivity has been shown to be a predictor of whether or not an individual votes (Fowler & Kam 2006). Taken together, we theorize that the CHRNA6 SNP rs2304297 is associated with turnout via its effect on impulsivity.

The study of voter turnout is central to political science, not least because of the centrality of elections to political life and because of the place of voter turnout at the head of a broader array of participatory actions. To date, this study has focussed almost exclusively on institutional, historical, and sociodemographic factors. Recently, however, scholars have turned their attention to basic differences between individuals as explanations for different propensities to vote in elections. For example, scholars have identified personality traits which are correlated with voter turnout (Gerber et al. 2008, Blais & Labbé-

St-Vincent 2010, Whiteside & Lynam 2001, Denny & Doyle 2008). More closely related to this study, scholars have demonstrated that voter turnout is heritable (Fowler, Baker & Dawes 2008) and is associated with genetic variants related to the serotonin system (Fowler & Dawes 2008) and the dopamine system (Dawes & Fowler 2009). Also, more general prosocial orientations closely tied to turnout (Fowler 2006, Edlin, Gelman & Kaplan 2007) have been shown to be heritable (Cesarini, Dawes, Johannesson, Lichtenstein & Wallace 2008, Cesarini, Dawes, Johannesson, Lichtenstein & Wallace 2009). Next steps in this research suggest considering more links in the chain from genes to behavior. In addition to personality measures, researchers could search for differences in neurological and cognitive processes (Schreiber, Simmons, Dawes, Flagan, Fowler & Pauls 2009) or in physiological processes (Oxley, Smith, Alford, Hibbing, Miller, Scalora, Hatemi & Hibbing 2008). In doing so, we can search for both direct and indirect relationships between each of these factors and political participation. Likewise, we can broaden our investigation to other forms of political engagement.

This research has three possible limitations. First, we have demonstrated a direct relationship between this SNP and voter turnout and have argued that this is due to patience. However, we do not have an instrument to directly measure the role of patience in turnout. This limits our conclusions until we can conduct a formal test of mediation.

Second, our sample is representative of young adults in America, but not the broader population. Our finding may not generalize to the adult population, though we have no reason to believe that the relationship between patience and turnout differs for younger and older adults. Nonetheless, these results would best be replicated in a larger population.

Third, our work may not account for environmental variation between our subjects. For example, we may be missing important differences in the information environments of our subjects (DiMaggio, Hargittai, and Neuman 2001). We likewise may be overlooking important differences in the competitiveness of down-ballot races (Shachar and Nalebuff 1999). Finally, we do not have explicit controls for the expected benefits of the election for different voters. All of these factors can be expected to affect the decision to vote, and some may in fact interact with patience. We have two responses to this limitation. First, we have good reason to believe that genes are causally prior to environmental factors. After all, they are set at conception. Second, even if the effects of the gene in question are mediated or conditional on environment, we would still expect the gene to matter for at least some individuals, and likely more strongly than the average estimate presented here.

The great majority of the work of identifying the genetic basis of political behavior remains to be done. We cannot, for example, expect to find a single voting gene. Rather, we should look to candidate genes which regulate or influence behavioral analogues to political behavior and then measure their effects on this behavior. We should also seek out and measure environmental variation that will allow us to know the conditional effects of genes. Finally, we should seek to replicate our work in different populations and different contexts. It is challenging work, but it is the way forward if we wish to realize a more comprehensive account of political participation.

Appendix

Variable Definitions

Partisan is the answer to the question "Do you identify with a specific political party?" *Attendance* is constructed from the response to the question "How often have you attended [church/synagouge/temple/mosque/religious] services in the past 12 months?" The categories of attendance are never, at least a few times but no more than once a month (baseline), and more than once a month. Other *race/ethnicity* indicator variables based on the questions "Are you of Hispanic or Latino origin?" and "What is your race? [white/black or African American/American Indian or Native American/Asian or Pacific Islander]". *Age* is self-reported age and *Male* is an indicator taking the value of 1 if the respondent is a male and 0 for a female. "Including all the income sources...what was your total personal income before taxes in [2000/2001]?" Those who failed to respond were asked the follow-up question "What is your best guess of your total personal income before taxes? [less than $10,000 , $10,000 to $14,999, $15,000 to $19,999, $20,000 to $29,999, $30,000 to $39,999, $40,000 to $49,999, $50,000 to $59,999, $60,000 to $74,999, $75,000 or more]. *Cognitive Ability* is the score on the Picture Vocabulary Test, which measures verbal intelligence.

Summary Statistics

	Mean	Standard Deviation
SNP1 G alleles	0.69	0.72
SNP2 C alleles	0.63	0.71
SNP3 G alleles	0.66	0.71
SNP4 G alleles	0.67	0.74
Vote	0.46	0.50
White	0.72	0.45
Black	0.19	0.39
Native American	0.02	0.15
Hispanic	0.12	0.33
Male	0.48	0.50
Partisan	0.37	0.48
Age	21.9	1.7
Income	2.8	1.5

Table 4: Sample means, standard errors, and 95% confidence intervals. Those subjects identifying themselves as more than one race are excluded from the analysis.

	0	1	2
SNP1	0.46	0.39	0.15
SNP2	0.51	0.36	0.13
SNP3	0.48	0.38	0.14
SNP4	0.49	0.35	0.16

Table 5: Percentage of the sample with 0,1,or 2 alleles. For rs2304297 (SNP1), rs4950 (SNP3), and rs13280604 (SNP4) the number of G alleles is presented and for rs892413 (SNP2) it is the number of C alleles.

Single Nucleotide 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. This is illustrated in *Figure 2*.

Single Nucleotide Polymorphism

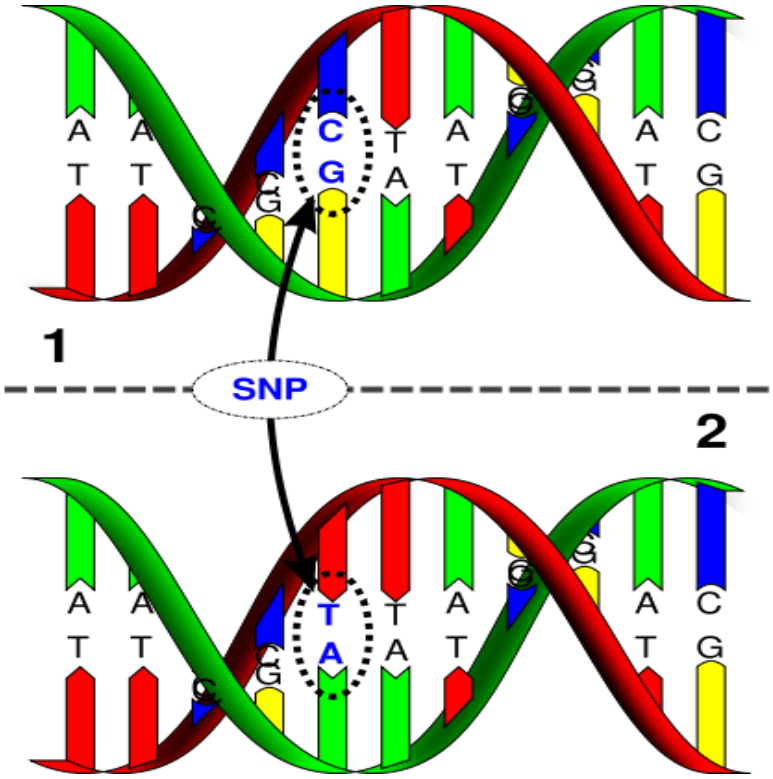

Figure 2: Single Nucleotide Polymorphism

References

Alford, J., C. Funk & J. Hibbing. 2005. "Are Political Orientations Genetically Transmitted?" *American Political Science Review* 99(2):153–167.

Bartels, L. 2000. "Partisanship and Voting Behavior, 1952-1996." *American Journal of Political Science* 44(1):35–50.

Becker, G.S. & C.B. Mulligan. 1997. "The endogenous determination of time preference." *Quarterly Journal of Economics* 112:729–758.

Bickel, W., A. Odum & G. Madden. 1999. "Impulsivity and Cigarette Smoking: Delay Discounting in Current, Never, and Ex-Smokers." *Psychopharmacology* 146(4):447–454.

Blais, André. 2000. *To Vote or Not to Vote*. University of Pittsburgh Press.

Blais, André & Simon Labbé-St-Vincent. 2010. "Personality Traits and the Propensity to Vote." Université de Montréal working paper.

Bouchard, T. & M. McGue. 2003. "Genetic and Environmental Influences on Human Psychological Differences." *Journal of Neurobiology* 54(1):4–45.

Cesarini, D., C. Dawes, M. Johannesson, P. Lichtenstein & B. Wallace. 2008. "Heritability of Cooperative Behavior in the Trust Game." *Proceedings of the National Academy of Sciences* 105:3721–3726.

Cesarini, D., C. Dawes, M. Johannesson, P. Lichtenstein & B. Wallace. 2009. "Genetic Variation in Preferences for Giving and Risk Taking." *Quarterly Journal of Economics* 124(2).

Cui, C., T. Booker, R. Allen, S. Grady, P. Whiteaker, M. Marks, O. Salminen, T. Tritto, C. Butt, W. Allen, J. Stitzel, M. McIntosh, J. Boulter, A. Collins & S. Heinemann. 2003. "The Beta3 Nicotine Receptor Subunit: A Component of Alpha-Conotoxin MII-Binding Nicotinic Acetylcholine Receptors that Modulate Dopamine Release and Related Behaviors." *The Journal of Neuroscience* 23(35):11045–11053.

Dani, J. & D. Bertrand. 2006. "Nicotine Acetylcholine Receptors and Nicotinic Cholinergic Mechanisms of the Central Nervous System." *Annual Review of Pharmacology and Toxicology* 47:699–729.

Dawes, C. & J. Fowler. 2009. "Partisanship, Voting, and the Dopamine D2 Receptor Gene." *Journal of Politics* TBD.

Dawes, C., Peter John Loewen & J. Fowler. 2009. "Social Preferences and Political Participation." Univeristy of California, San Diego Working Paper.

Deary, I., D. Batty & C. Gale. 2008. "Childhood Intelligence Predicts Voter Turnout, Voting Preferences, and Political Involvement in Adulthood: The 1970 British Cohort Study." *Intelligence* 36:548–555.

Denny, K. & O. Doyle. 2008. "Political Interest, Cognitive Ability and Personality: Determinants of Voter Turnout in Britain." *British Journal of Political Science* 38:291–310.

Edlin, A., A. Gelman & N. Kaplan. 2007. "Voting as a Rational Choice: Why and How People Vote to Improve the Well-Being of Others." *Rationality and Society* 19(3):293–314.

Fowler, J. 2006. "Altruism and Turnout." *Journal of Politics* 68(3):674–683.

Fowler, J. & C. Kam. 2006. "Patience as a Political Virtue: Delayed Gratification and Turnout." *Political Behavior* 28:113–128.

Fowler, James H. & Christopher T. Dawes. 2008. "Two Genes Predict Voter Turnout." *Journal of Politics* 70(3):579–594.

Fowler, James H., Laura A. Baker & Christopher T. Dawes. 2008. "Genetic Variation in Political Behavior." *American Political Science Review* 102(2):233–248.

Funder, D.C., J.H. Block & J. Block. 1983. "Delay of gratification: Some longitudinal personality correlates." *Journal of Personality and Social Psychology* 44:1198–1213.

Gerber, A., G. Huber, C. Raso & Shang Ha. 2008. "Personality and Political Behavior." Unpublished Manuscript.

Guo, G. & H. Zhao. 2000. "Multilevel Modeling for Binary Data." *Annual Review of Sociology* 26:441–462.

Hariri, A., S. Brown, D. Williamson, J. Flory, H. de Wit & S. Manuck. 2006. "Preference for Immediate over Delayed Reward Is Associated with Magnitude of Ventral Striatal Activity." *The Journal of Neuroscience* 26(51):13213–13217.

Hatemi, Peter K., John Hibbing, John Alford, Nicholas Martin & Lindon Eaves. 2009. "Is There a Party in Your Genes." *Political Research Quarterly* 62(3):584–600.

Jacobson, K. & D. Rowe. 1998. "Genetic and Shared Environmental Influences on Adolescent BMI: Interactions with Race and Sex." *Behavior Genetics* 28(4):265–278.

Jang, K., R. McCrae, A. Angleitner, R. Riemann & W. Livesley. 1998. "Heritability of Facet-Level Traits in a Cross-Cultural Twin Sample: Support for a Hierarchical Model of Personality." *Journal of Personality and Social Psychology* 74(6):1556–1565.

John, O.P. & S. Srivastava. 1999. The Big-Five Trait Taxonomy: Theory, Measurement, and Theoretical Perspectives. In *Handbook of Personality Theory and Research*, ed. L.A. Pervin & O.P. John. 2nd ed. New York: Guilford.

Loewen, Peter John. 2010. "Antipathy, Affinity, and Political Participation: How Our Concern for Others Makes Us Vote." *Canadian Journal of Political Science* .

Madden, G., N. Petry, G. Badger & W. Bickel. 1997. "Impulsive and Self-Control in Opioid-Dependent Patients and Non-Drug-Using Control Participants: Drug and Monetary Rewards." *Experimental and Clinical Psychopharmacology* 5(3):256–262.

Meyer, E., D. Yoshikami & M. McIntosh. 2008. "The Neuronal Acetylcholine Receptors Alpha4* and Alpha6* Differentially Modulate Dopamine Release in Mouse Striatal Slices." *Journal of Neurochemistry* 105:1761–1769.

Mischel, W. 1974. "Processes in delay of gratification." *Advances in Experimental Social Psychology* 7:249–292.

Mischel, W. & C. Gilligan. 1964. "Delay of gratification, motivation for the prohibited gratification, and responses to temptation." *Journal of Abnormal and Social Psychology* 69:411–417.

Mischel, W. & R. Metzner. 1962. "Preference for delayed reward as a function of age, intelligence, and length of delay interval." *Journal of Abnormal and Social Psychology* 64:425–431.

Mischel, W., Y. Shoda & M. Rodriguez. 1989. "Delay of gratification in children." *Science* 244(4907):933–968.

Mischel, W., Y. Shoda & P.K. Peake. 1988. "The nature of adolescent competencies predicted by preschool delay of gratification." *Journal of Personality and Social Psychology* 54:687–696.

Oxley, Douglas R., Kevin B. Smith, John Alford, Matthew V. Hibbing, Jennifer L. Miller, Mario Scalora, Peter K. Hatemi & John R. Hibbing. 2008. "Political Attitudes Vary with Physiological Traits." *Science* 321(5896):1667–1670.

Rowe, D., K. Jacobson & E. Van den Oord. 1999. "Genetic and Environmental Influences on Vocabulary IQ: Parental Education Level as a Moderator." *Child Development* 70(5):1151–1162.

Schlaepfer, Isabel R., Nicole R. Hoft & Marissa A. Ehringer. 2008. "The genetic components of alcohol and nicotine co-addiction: From genes to behavior." *Current Drug Abuse Review* 1(2):124–134.

Schreiber, Darren, Alan N. Simmons, Christopher T. Dawes, Taru Flagan, James H. Fowler & Martin P. Pauls. 2009. "Red Brain, Blue Brain: Evaluative Processes Differ in Democrats and Republicans." Working Paper.

Settle, Jaime E., Christopher T. Dawes & James H. Fowler. 2009. "The Heritability of Partisan Attachment." *Political Research Quarterly* 62(3):601–613.

Spielman, R. & W. Ewens. 1998. "A Sibship Test for Linkage in the Presence of Association: The Sib Transmission/Disequilibrium Test." *American Journal of Human Genetics* 62:450–458.

Stumphauzer, J.s. 1972. "Increased delay of gratification in young prison inmates through imitation of high-delay peer models." *Journal of Personality and Social Psychology* 21:10–17.

Verba, S., K. Schlozman & H. Brady. 1995. *Voice and Equality: Civic Volunteerism in American Politics*. Harvard University Press.

Waldman, I., B. Robinson & D. Rowe. 1999. "A Logistic Regression Based Extension of the TDT for Continuous and Categorical Traits." *Annals of Human Genetics* 63:329–340.

Whiteside, S.P. & D.R. Lynam. 2001. "The Five Factor Model and impulsivity: Using a structural model of personality to understand impulsivity." *Personality and Individual Differences* 30:669–689.

Wonnacott, S. 1997. "Presynaptic Nicotinic ACh receptors." *Trends in Neuroscience* 20:92–98.

Xu, H. & S. Shete. 2006. "Mixed-effects Logistic Approach for Association Following Linkage Scan for Complex Disorders." *Annals of Human Genetics* 71:230–237.

Zou, G. 2006. "Statistical Methods for the Analysis of Genetic Association Studies." *Annals of Human Genetics* 70:262–276.