Two Genes Predict Voter Turnout

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Abstract

Fowler, Baker & Dawes (2007) recently showed in two independent studies of twins that voter turnout has very high heritability. Here we investigate two specific genes that may contribute to variation in voting behavior. Using data from the National Longitudinal Study of Adolescent Health, we show that individuals with a polymorphism of the MAOA gene are significantly more likely to have voted in the 2004 presidential election. We also find evidence that an association between a polymorphism of the 5HTT gene and voter turnout is moderated by religious attendance. These are the first results ever to link specific genes to political behavior.

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Introduction

Social scientists have shown that basic political attitudes like liberalism and conservatism are likely to be heritable (Alford, Funk & Hibbing 2005, Hatemi, Medland, Morley, Heath & Martin 2007). While the choice of a particular candidate or party does not appear to be heritable, a significant proportion of the variation in the decision to participate in politics can be attributed to genetic factors. Fowler, Baker & Dawes (2007) recently studied the voting behavior of two populations of twins and showed that heritability accounted for 53% of the variation in validated turnout of those living in Los Angeles county and 72% of the self-reported turnout in a nationally-representative sample of young adults. They also showed that heritability accounted for 60% of the variation in a general index of political participation, including contributing to campaigns, running for office, volunteering for political organizations, and attending protests. These results were the first to suggest that humans exhibit inherent variability in their willingness to participate in politics.

However, these initial results based on twin studies beg the question "which genes?" The natural place to start the search for such genes is among those that have already been shown to account for variation in social behavior. And among these, MAOA and 5HTT are prime candidates. These two genes transcribe neurochemicals that exert a strong influence on the serotonin system in parts of the brain that regulate fear, trust, and social interaction (Bertolino, Arciero, Rubino, Latorre, Candia & Mazzola 2005, Hariri, Mattay, Tessitore, Kolachana, Fera & Goldman 2002, Hariri, Drabant, Munoz, Kolachana, Mattay, Egan & Weinberger 2005, Heinz, Braus, Smolka, Wrase, Puls, Hermann & et al. 2005, Meyer-Lindenberg, Buckholtz, Kolachana, Hariri, Pezawas, Blasi, Wabnitz, Honea, Verchinski, Callicott, Egan, Mattay & Weinberger 2006, Eisenberger, Way, Taylor, Welch & Lieberman 2007). MAOA and 5HTT have been studied for more than twenty years and much is known about the way different versions of their genes regulate transcription, metabolism, and signal transfers between neurons, all of which have an effect on social interactions (Craig 2007). In particular, the less transcriptionally efficient alleles of these genes have been associated with a variety of antisocial behaviors (Rhee & Waldman 2002).

In this article, we hypothesize that people with more transcriptionally efficient alleles of the MAOA and 5HTT genes are more likely to vote. An association between a gene and political behavior may also be moderated by environmental factors. This phenomenon is known as a gene-

environment (GxE) interaction (Shanahan & Hofer 2005). We therefore also hypothesize that an association between each of these genes and voting may be moderated by social activity. Using data from the National Longitudinal Study of Adolescent Health, we conduct gene and gene-environment association tests on the relationship between turnout and MAOA and 5HTT. The results show that both genes are significantly associated with the decision to vote. Moreover, the association between 5HTT and turnout is moderated by exposure to religious social activity. These findings have important implications for how we both model and measure political interactions.

Past Work on the Genetic Basis of Political Participation

Although we are not the first to suggest a link between genes and political participation, this study is the first to investigate an association between specific genes and political behavior. Early work studied the importance of personality in political participation, but this literature focused exclusively on environmental factors, asserting that people who are reared in similar ways will have similar personalities (Lane 1959, Levinson 1958) or that the role of personality is to mediate social influences on participation (Krause, Houlihan, Oberlander & Carson 1970). Additional earlier studies focused on the importance of adolescent socialization in the development of political behaviors, but these scholars never considered the role of genes in the link between parent and child. Merelman (1971) explicitly addressed this shortcoming, arguing that both genes and environment are probably important. He lamented the fact that genetic explanations had been ignored by social scientists:

> "[T]his natural tendency to examine one environmental factor after another ad infinitum does a genetic explanation something of an injustice. The problem is that while we can examine environmental variables directly, we can usually only infer genetic effects, and so our natural tendency is to slight the latter perspective. In short, our procedures, following the line of least methodological resistance, impinge heavily upon our theoretical perspectives." (1044)

In spite of Merelman's early call for attention, genetic studies of participation were not forthcoming. Scholars continued to focus on personality factors underlying participation like efficacy (Finkel 1985) and self-esteem (Sears 1987) without considering the fact that these factors may be

heritable. A few political scientists have argued on general principle that genes must play a role in political behaviors like participation (Carmen 2004, Masters 1990, Somit & Peterson 1998) but they have left the work of testing their hypotheses to others.

A wide range of studies have already shown that variation in prosocial personality and behavior can be attributed to genes (McGue, Bacon & Lykken 1993, Rushton, Fulker, Neale, Nias & Eysenck 1986, Scourfield, John, Martin & McGuffin 2004). This literature suggests that innate dispositions play an impotant role in an individual's willingness to participate in social activities or to engage in acts that primarily benefit others. Meanwhile, a growing number of observational studies, theoretical models, and laboratory experiments suggest that prosocial attitudes and behavior are important factors for explaining voter turnout and political participation. For example, Knack (1991) creates an index of "social altruism" from questions about charity, volunteer work, and community involvement on the 1991 NES Pilot Study and finds a positive relationship between the index and turnout. Similarly, Jankowski (2007) finds a relationship between turnout and humanitarian norms from questions on the 1995 NES Pilot Study. Edlin, Gelman & Kaplan (2007) show that a variety of aggregate features of turnout can be easily explained by incorporating prosocial preferences into the decision-theoretic calculus of voting, and Jankowski (2002) shows that this reasoning extends to a game-theoretic model. Finally, experimental studies utilizing dictator games to measure revealed social preferences show that individuals who are more willing to engage in costly giving to others are also more likely to vote (Fowler 2006*a*) and participate in politics (Fowler & Kam 2007, Dawes & Fowler 2007).

Thus, we hypothesize that genes may influence voting and political participation because they influence a generalized tendency to engage in prosocial behavior via their functional role in nuerochemical processes. Although Fowler, Baker & Dawes (2007) have already shown that a large fraction of the variation in voter turnout and political participation can be attributed to genetic factors, to date no specific genes have been identified in this process. It is crucial to point out at the outset that we cannot test, given our data, the potential causal pathways we suggest. Therefore, the goal of this study is to show association rather than causality.

Some Basic Genetics Concepts

Genes are distinct regions of human DNA that form the blueprint for molecules that regulate the development and function of the human body. There are an estimated 25,000 genes (most of which exist in multiple copies) in the 46 chains, or chromosomes, that make up all human DNA. Almost all human cells contain the same inherited DNA chains that are fixed from the moment of conception. This is an important point for social scientists. Since genes are fixed, they represent the purest measure of biological inheritance, virtually unaffected by environment and able to be collected at any point throughout a person's life.

At conception individuals inherit one half of their DNA from each parent, with one copy of each gene coming from the mother and one copy from the father. Some genes come in different versions, known as "alleles"—for example, sickle cell disease results from a particular allele coding for abnormal rather than normal hemoglobin. Each parent has two separate copies of an allele at each "locus", or location, on the chromosome, but each sperm or egg cell contains only one of these alleles. Thus a child has a 50% chance of receiving a particular allele from a particular parent. For example, suppose that at a given locus there are two possible alleles, A and B. If both parents are "heterozygous" at that locus, meaning they each have an A and a B allele (AB), then a given offspring has a 25% chance of being "homozygous" for A (AA), a 25% chance of being homozygous for B (BB) and a 50% chance of being heterozygous (AB or BA—order is irrelevant).

Genes transcribe proteins and following this process, these proteins begin a cascade of interactions that regulate bodily structure and function. Many of the observable traits and behaviors of interest, referred to as "phenotypes", are far downstream from the original "genotypes" present in the DNA. While in some cases one allele can single-handedly lead to a disease (such as Sickle Cell Anemia, Huntingtons disease, and cystic fibrosis), the vast majority of phenotypes are "polygenic", meaning they are influenced by multiple genes (Mackay 2001, Plomin 2008), and are shaped by a multitude of environmental forces. As a result, simple association models between genotype and phenotype are an important first step to establish candidate genes, but they are not the end of the story. It is also important to investigate the extent to which genetic associations are moderated by environmental factors ("environmental modifiers") and other genes ("genetic modifiers").

Serotonin, Genes, and Social Behavior

Twin studies have already established that genetic factors account for a significant proportion of the variation in antisocial behaviors (Rhee & Waldman 2002), including substance abuse, impulsivity, criminality, precocious sexuality, and a combination of these behaviors called antisocial personality disorder (ASPD). However, twin studies cannot establish which genes are implicated. It is likely that dozens, if not hundreds of genes influence sociability (Mackay 2001, Plomin 2008). As a result, scientists typically start with "candidate" genes that are known to influence related behaviors or processes in the body. For social behavior, this means focusing on genes that affect brain development, neurotransmitter synthesis and reception, hormone regulation, and transcription factors (Damberg, Garpenstrand, Hallman & Oreland 2001).

To determine whether genes affect voting behavior we chose two candidate genes that have already received a great deal of attention for their association with antisocial behavior. These genes, MAOA and 5HTT[1], are critical to the metabolism of serotonin in the brain. As shown in Figure 1, serotonin is a chemical that is released when a neuron "fires" and sensed by a receptor on the receiving neuron, passing an electric potential across a gap called a nerve synapse (the nerve that fires is on the "pre-synaptic" side of the gap). Signals are carried throughout the body by the sequential firing of one neuron after another across these synapses. When an individual experiences stress, it causes increased neuron activity, stimulating the release of excess serotonin into the gaps between the synapses (Chaouloff, Berton & Mormede 1999). If serotonin remains outside the cells, it can oxidize into a toxin that kills both the pre-synaptic and post-synaptic neurons. The body's homeostatic response to this excess serotonin is to reabsorb it into the pre-synaptic neuron via a transporter in the cell wall, called 5HTT. Once the "reuptake" of serotonin is complete and it is back inside the neuron, an enzyme called monoamine oxidase A (MAOA) degrades the serotonin so that its components can be reabsorbed in the cell. The genes responsible for transcribing 5HTT and MAOA are eponymous—the 5HTT gene produces 5HTT and the MAOA gene produces MAOA.

Animal studies indicate that the serotonin system has an important effect on social behavior. Rhesus macaque monkeys with impaired serotonin metabolisms are impulsive and aggressive in response to social stressors (Kraemer, Ebert, Schmidt & McKinney 1989) and studies of rodents

[1]The 5HTT gene has several other names, including HTT, SLC6A4, and SERT.

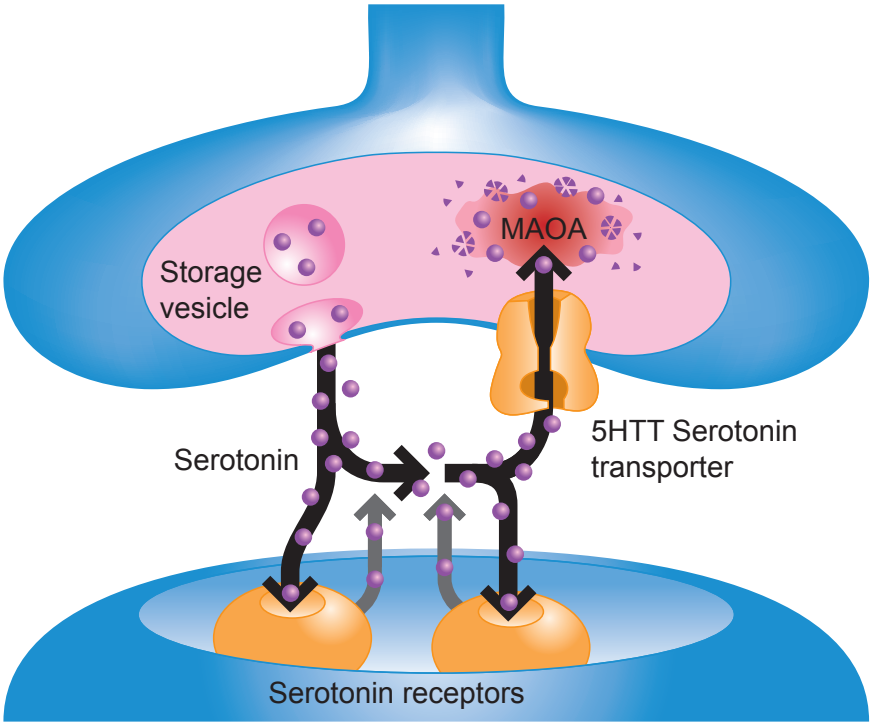

Figure 1: Simple representation of the release, reception, and recycling of serotonin in neurons.

show that acute emotional stress affects the way MAOA breaks down serotonin in several areas of the brain (Popova, Voitenko & Maslova 1989, Virkkunen, Goldman, Nielsen & Linnoila 1995). In mice, social stress increases transcription of both MAOA and 5HTT (Filipenko, Beilina, Alekseyenko, Dolgov & Kudryavtseva 2002) and knock-out studies that eliminate the MAOA gene in subjects cause enzymatic activity to come to a complete halt (Cases, Seif, Grimsby, Gaspar & Chen 1995). In monkeys, 5HTT is densely concentrated in the output regions of the amygdala, which affects fear recognition (O'Rourke & Fudge 2006) and MAOA has been shown to alter the structure of the brain in mice (Cases, Vitalis, Seif, De Maeyer, Sotelo & Gaspar 1996). This evidence suggests that any deficiency in the genes that regulate serotonin metabolism will have a direct effect on the brain that tends to reduce the ability to process and respond to social stress. These effects have been linked specifically to a genetic polymorphism in monkeys that is closely related to that observed in humans (Suomi 2003, Newman, Syagailo, Barr, Wendland, Champoux & et al. 2005). There is also strong evidence that the serotonin system affects complex social traits in humans (Balciuniene & Jazin 2001) and 5HTT and MAOA frequently serve as targets for antidepressants and illegal recreational drugs (Craig 2007, Livingston & Livingston 1996).

The 5HTT gene contains a 44 base-pair variable-number tandem repeat (VNTR) polymorphism[2] in the promoter region[3] that is believed to be responsible for variation in transcriptional activity. The transcriptional efficiency of the "long" version of this allele is associated with a much higher basal activity than the shorter allele (Lesch, Bengel, Heils, Sabol, Greenberg, Petri & et al. 1996, Little, McLaughlin, Zhang, Livermore, Dalack & McFinton 1998). MAOA has a 30 base-pair VNTR polymorphism located in the promoter region. The "high" version of this polymorphism significantly increases the transcriptional efficiency of MAOA (Sabol, Hu & Hamer 1998, Denney, Sharma, Dave & Waguespack 1994, Denney, Koch & Craig 1999). The less transcriptionally efficient alleles of both 5HTT and MAOA have been linked to antisocial behavior (Vanukov, Moss, Yu & Deka 1995, Hsu, Loh, Chen, Chen, Yu & et al 1996, Lawson, Turic, Langley, Pay, Govan & et al. 2003, Domsche, Sheehan, Lowe, Kirley, Mullins & et al 2005, Saito, Lachman, Diaz, Hallikaien, Kauhanen & et al 2002, Schmidt, Sander, Kuhn, Smolka, Rommelspacher & et al 2000, Samo-

[2]A VNTR polymorphism is a repeated segment of DNA that varies among individuals in a population.

[3]A promoter region is the regulatory region of DNA that tells transcription enzymes where to begin. These promoter regions typically lie upstream from the genes they control.

chowiec, Lesch, Rottmann, Smolka, Syagailo & et al 1999, Contini, Marques, Garcia, Hutz & Bau 2006) which appears to be mediated by certain parts of the brain. For example, the development of the amygdala and orbitofrontal cortex has been linked to a small genetic locus which contains the gene for MAOA (Good, Lawrence & Thomas 2003). Furthermore, a number of studies show that the amygdala becomes "hypersensitive" during the presentation of aversive or threatening social stimuli in individuals with either the short 5HTT allele (Bertolino et al. 2005, Hariri et al. 2002, Hariri et al. 2005, Heinz et al. 2005) or the low MAOA allele (Meyer-Lindenberg et al. 2006). Eisenberger et al. (2007) report similar results, noting that the link to antisocial behavior results from an increased sensitivity to negative socioemotional experiences (though in their study they claim the effect is mediated via the dorsal anterior cingulate cortex).

Not all studies show a direct relationship between these polymorphisms and behavior. Instead, developmental or concurrent environments may moderate an association between genes and observed social behavior. A gene-environment (GxE) interaction has been identified in many cases for antisocial behavior (Caspi, McClay, Moffitt, Mill, Martin, Craig, Taylor & Poulton 2002, Foley, Eaves, Wormley, Silberg, Maes & et al. 2004, Haberstick, Lessem, Hopfer, Smolen, Ehringer, Timberlake & Hewitt 2005, Nilsson, Sjoberg, Damberg, Leppert, Ohrvik & et al 2006, Kim-Cohen, Caspi, Taylor, Williams, Newcombe & et al 2006), the most famous of which is the Caspi *et al.* (2002) paper. This work shows that exposure to stressors like child abuse at early developmental stages may interact with the low MAOA polymorphism resulting in antisocial behavior later in life. This is an important point—in these studies the gene itself was not associated with the behavior. Rather, it was the combination of both gene and environment that yielded a significant association.

Two Hypotheses for Genes and Turnout

A growing literature suggests that voter turnout is a *prosocial* behavior that is strongly influenced by other-regarding preferences (Edlin, Gelman & Kaplan 2007, Fowler 2006*a*, Jankowski 2007, Jankowski 2002, Fowler & Kam 2007). Given that polymorphisms of MAOA and 5HTT appear to influence antisocial behavior, we therefore hypothesize that they will also be associated with voting behavior. One difficulty of the voting experience is that one's preferred candidates sometimes lose. This loss has been theorized to reduce future motivations to vote (Bendor, Diermeier & Ting 2003,

Fowler 2006*b*) and Kanazawa (1998) has even shown empirically that turnout declines among those whose favorite candidates lost the previous election. In addition, people may prospectively consider how they will feel about a loss before deciding whether or not to vote, or whether they will even pay attention to the election. Those who are overly sensitive to social conflict may choose to stay home and ignore politics, while less sensitive individuals will not take the potential emotional stress caused by the loss of their favorite candidates into consideration. Thus, we expect individuals with the "high" MAOA polymorphism and "long" 5HTT polymorphism will be more likely to turn out to vote.

However, an association between either MAOA and 5HTT and voting may not be direct. Instead, an association between a gene and turnout may be moderated by environmental factors. A vast literature on turnout suggests the importance of voter mobilization efforts (Wielhouwer & Lockerbie 1994), religious group activity (Cassel 1999), and other kinds of social contacts that have an influence on political participation (Verba, Schlozman & Brady 1995, Huckfeldt 1979). Religious group activity in particular has been singled out as one of the strongest predictors of voter turnout, even more so than socioeconomic status (Olsen 1972, Sallach, Babchuk & Booth 1972). However, scholars have had difficulty interpreting this association. Religious groups might stimulate political activity directly or as byproducts of their tendency to increase civic skills, political interest, feelings of efficacy, access to political information, and a sense of civic duty. Testing all of these possible explanations, Cassel (1999) suggests that the main reason for the association is that religious groups build a sense of belonging to a larger community. However, it may not be possible to build such a sense in people who are too averse to social conflict, since they will resist appeals to become involved. We therefore hypothesize that MAOA and 5HTT, when interacted with religious group activity, may be significantly associated with turnout. Specifically, individuals who are actively involved in their religious organizations and who have the "high" MAOA allele or the "long" 5HTT allele will be more likely to vote than others.

Data

All of our analysis is based on individual-level genetic and survey data collected as part of the The National Longitudinal Study of Adolescent Health (Add Health). Add Health is a study

that explores the causes of health-related behavior of adolescents in grades 7 through 12 and their outcomes in young adulthood. The first wave of the Add Health study (1994-1995) selected 80 high schools from a sampling frame of 26,666. The schools were selected based on their size, school type, census region, level of urbanization, and percent of the population that was white. Participating high schools were asked to identify junior high or middle schools that served as feeder schools to their school. This resulted in the participation of 145 middle, junior high, and high schools. From those schools, 90,118 students completed a 45-minute questionnaire and each school was asked to complete at least one School Administrator questionnaire. This process generated descriptive information about each student, the educational setting, and the environment of the school. From these respondents, a core random sample of 12,105 adolescents in grades 7-12 were drawn plus several over-samples, totaling more than 27,000 adolescents. These students and their parents were administered in-home surveys in the first wave. Wave II (1996) was comprised of another set of in-home interviews of more than 15,000 students from the Wave I sample and a follow-up telephone survey of the school administrators. Finally, Wave III (2001-2002) consisted of an in-home interview, six years later, of 15,170 Wave I participants. The result of this sampling design is that Add Health is a nationally representative study. Women make up 49% of the study's participants, Hispanics 12.2%, Blacks 16.0%, Asians 3.3%, and Native Americans 2.2%.[4] Participants in Add Health also represent all regions of the country: the Northeast makes up 17% of the sample, the South 27%, the Midwest 19%, and the West 17%.

In Wave I of the Add Health study, researchers created a genetically informative sample of sibling pairs based on a screening of the in-school sample of 90,114 adolescents. These pairs include all adolescents that were identified as twin pairs, half siblings, or unrelated siblings raised together. Twins and half biological siblings were sampled with certainty. The Wave I sibling-pairs sample has been found to be similar in demographic composition to the full Add Health sample (Jacobson & Rowe 1998). Allelic information for six genetic markers are available for 2,574 individuals as part of Wave III,[5] including markers that identify alleles of MAOA and 5HTT. Details of the DNA

[4] A breakdown for those providing DNA samples is presented in the appendix.

[5] We do not use the Add Health sampling weights because more than a third of subjects in the genetic sample had a co-sibling that was interviewed as part of Wave III but not as part of the original Wave I sampling frame (Lessem, Hopfer, Haberstick, Timberlake, Ehringer, Smolen & Hewitt 2006). Therefore, sampling weights could not

collection and genotyping process are available at the Add Health website (Add Health Biomarker Team 2007).

MAOA alleles consist of 2 repeats, 3 repeats, 3.5 repeats, 4 repeats, and 5 repeats with 291, 321, 336, 351, and 381 base-pair fragment sizes respectively. The 291 and 321 base-pair alleles are believed to have lower transcriptional efficiency than the 336, 351, and 381 base-pair alleles (Sabol, Hu & Hamer 1998, Denney et al. 1994). Following Haberstick et al. (2005), we group the 291 and 321 base-pair allele to form a "low" transcription group and the 336, 351, and 381 base-pair alleles to form a "high" transcription group. Allele frequency for the low grouping is 41% and high grouping is 59% in our sample. 5HTT alleles similarly vary in their transcriptional activity, with a "long" 528 base-pair allele associated with a much higher basal activity than the shorter 484 base-pair allele. Allele frequency for the short allele is 42% and long allele is 58%.

Nearly 80% of the sibling-pairs sample participants in Wave I also participated in Wave III and provided information about their recent religious and political activity and attitudes as well as DNA samples. In particular, subjects reported how often they attended church, synagouge, temple, mosque, or other religious services in the past 12 months. The categories for response were 'never', 'a few times', 'several times', 'once a month', '2 or 3 times a month', 'once a week', and 'more than once a week'. We simplified these responses by grouping them into three categories of attendance: never, at least a few times but no more than once a month, and more than once a month.

Subjects also answered "Did you vote in the most recent [2000] presidential election?" While this question gives us a valuable opportunity to explore the genetic basis of political behavior, we want to make clear two limitations of the data. First, it would be preferable to have information about validated turnout because of the well-known problem of over-reporting—many people who say they voted actually did not (Karp & Brockington 2005). However, Fowler, Baker & Dawes (2007) show that a substantial genetic component exists for both validated and self-reported turnout, and they do not find a statistically meaningful difference in the size of the component for the different measures. Second, it would also be preferable to have information about the voting behavior of older adults. The Add Health sample is restricted to individuals who are 18-26 years old during

be constructed for these subjects. Limiting our analysis to only individuals in the genetic sample for which weights could be determined would greatly reduce statistical power.

Wave III, so it is possible that our results apply only to the *initiation* of turnout behavior in young adults and not to its subsequent development as people age (Plutzer 2002).

Genetic Association

Genetic association studies test whether an allele or genotype occurs more frequently within a group exhibiting a particular trait than those without the trait. For example, is the frequency of a particular allele or genotype higher among voters than non-voters? However, a significant association can mean one of three things: (1) The allele itself influences voting behavior; (2) the allele is in "linkage disequilibrium" with an allele at another locus that influences voting; or (3) the observed association is a false positive signal due to population stratification.

Population stratification occurs because groups may have different allele frequencies due to their genetic ancestry. Turnout in these groups may be the product of their environments, alleles other than the one of interest, or some unobserved reason.[6] For example, two groups may not have mixed in the past for cultural reasons. Through the process of natural selection or genetic drift these groups may develop different frequencies of a particular allele. At the same time, the two groups may also develop divergent behaviors that are not influenced by the allele but completely by the environment in which they live. Once these two groups mix in a larger population, simply comparing the frequency of the allele to the observed behavior would lead to a spurious association.

There are two main research designs employed in association studies, case-control designs and family-based designs. Case-control designs compare the frequency of alleles or genotypes among subjects that exhibit a trait of interest to subjects who do not.[7] As a result, case-control designs are vulnerable to population stratification if either group is especially prone to selection effects. 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

[6] Given our data, we cannot differentiate between 1 and 2. In order to do so we would need additional genetic information about loci in close proximity to the locus of interest. Thus, a significant association means that either a particular allele, or one likely near it on the same gene, significantly influences voting behavior.

[7] Controls may be randomly selected from the population or those known not to exhibit the trait.

parents more often than would be expected by chance. This design is very powerful in minimizing type I error but also suffers from much lower power in detecting a true association. Xu & Shete (2006) show, based on extensive simulation work, that a case-control association study using a mixed-effects logistic regression outperforms family-based designs in detecting an association while at the same time effectively limiting type I error.

Model

To test for genetic association we employ a mixed-effects logistic regression model (Guo & Zhao 2000, Xu & Shete 2006):

$$P[Y_{ij} = 1|Z_{kij}, U_j] = logit\left(\beta_0 + \beta_G G_{ij} + \beta_E E_{ij} + \beta_{GxE}(G_{ij} * E_{ij}) + \beta_k Z_{kij} + U_j\right)$$

where i and j index subject and family respectively. For the MAOA gene, $G = 1$ if the subject's genotype is HH, and $G = 0$ for genotypes Hl or ll (where H represents having a copy of a 336, 351, or 381 base-pair "high" allele, and l represents having a copy of a 291 or 321 base-pair "low" allele). For the 5HTT gene, $G = 1$ if the subject's genotype is LL or Ls and $G = 0$ if the subject's genotype is ss (where L represents having a copy of the 528 base-pair "long" allele and s represents having a copy of the 484 base-pair "short" allele).[8] The variable E is an environmental variable we believe moderates the influence of the genotype on voting behavior. We test one such variable, regular attendance of religious services. Z is a matrix of variables to control for underlying population structure of the Add Health samples as well as potentially mediating factors like age, gender, income, and education that have been found to significantly influence turnout. Finally, the variable

[8]We classified genotypes based on transcriptional efficiency. The long allele of the 5HTT gene and the high allele of the MAOA gene has been associated with higher transcriptional efficiency (Lesch et al. 1996, Little et al. 1998, Sabol, Hu & Hamer 1998, Denney et al. 1994, Denney, Koch & Craig 1999). Males are homozygous for the MAOA gene (HH or ll), however females may be heterozygous (Hl). Following previous studies, we classify heterozygous females as the Low genotype and all homozygous individuals as the High genotype (Frazzetto, Di Lorenzo, Carola, Proietti, Sokolowska, Siracusano, Gross & Troisi 2007, Fan, Fossella, Sommer, Wu & Posner 2003). Previous research has shown that being homozygous for the short allele (ss) makes one more vulnerable to negative environmental stimuli compared to being heterozygous for the short allele or homozygous for the long allele (Ls or LL) (Caspi, Sugden, Moffitt, Taylor, Craig, Harrington, McClay, Mill, Martin, Braithwaite & Poulton 2003, **?**, **?**). Therefore, we combined Ls and LL into the Long genotype and ss into the Short genotype.

U is a family random effect that controls for potential genetic and environmental correlation among family members.

To control the effects of the underlying population structure, we include indicator variables for whether a subject self-reported as Black, Hispanic, Asian, or Native American (base category is White). Following the policy of the United States Census, Add Health allows respondents to mark more than one race. Since this complicates the ability to control for stratification, we exclude these individuals ($N = 117$), but supplementary analysis including them yields substantively identical results. We also exclude from the data analysis non-citizens and people less than 18 years of age on Election Day since they are not legally eligible to vote. This leaves us with a sample size of 2,329 individuals.[9]

The odds ratio of β_G is an individual's odds of voting if he or she is HH genotype for the MAOA gene compared to an individual with an Hl or ll genotype. A significant odds ratio means that lacking a "short" allele is associated with higher turnout when compared to having at least one "short" allele. For the 5HTT gene, the odds ratio of β_G is an individual's odds of voting given he or she has at least one "long" allele (LL or Ls) compared to having no long alleles (ss). Therefore, a significant odds ratio implies that having a "long" 5HTT allele is associated with the decision to vote. Finally, the odds ratio of β_{GxE} is whether having a specific genotype combined with being exposed to an environmental effect influences turnout behavior, even after controling for both main effects.

Results

Table 1 shows the results of several specifications of the models to test the hypothesis that genes are associated with voter turnout and whether the association is moderated by religious service attendance. Each of these specifications includes variables for age, gender, and race to control for population stratification. *Model 1* shows that the "high" allele of MAOA is significantly associated with increased voter turnout ($p = 0.03$). This model suggests that the odds of a person with the "high" version of the MAOA gene voting are 1.26 times greater than that of a person with

[9] Our sample contains 451 single-person families, 884 families with two siblings, 34 families with 3 siblings, and 2 families with 4 siblings.

the "low" version.[10] *Model 2* rejects the hypothesized relationship between 5HTT and voting ($p = 0.99$). Thus, only MAOA appears to be directly associated with turnout.

The next two models test the possibility that attendance of religious services moderates an association between each of the genes and turnout. *Model 3* suggests that no moderation relationship exists for MAOA ($p = 0.93$) but it also shows the robustness of the direct association with turnout since including attendance and an interaction in the model does not alter the significance of the main effect ($p = 0.02$). In contrast, *Model 4* indicates that an association between 5HTT and voting is in fact moderated by attendance ($p = 0.01$). The odds of voting for those with the "long" version of the 5HTT gene and who frequently attend religious services are 1.58 greater than people with the "short" version.[11] To test the robustness of the direct and moderated associations, we model both of them simultaneously in *Model 5*. The results show that both odds ratios remain significant at $p < 0.04$.[12]

In Figure 2 we summarize our results for MAOA and the interaction between 5HTT and attendance by simulating first differences from the coefficient covariance matrix of *Model 1* and *Model 4*. Holding all else constant and changing the MAOA gene of all subjects from "low" to "high" would increase average turnout in this hypothetical population by about 5 percentage points. Changing the 5HTT gene of all religious attendees from "short" to"long" would increase average turnout in that group by about 10 percentage points.

Model 6 includes a number of factors previous studies have found to influence turnout. These variables may in fact *mediate* the relationship between the genes we have identified and turnout.[13] For example, MAOA and 5HTT may be associated with a disposition towards partisan-

[10]We also checked whether the odds ratio was significantly influenced by race or gender and it was not. We added an interaction with High MAOA and gender (male=1) which was not significant ($p = 0.98$) and also an interaction with High MAOA and race (white=1) which was not significant ($p = 0.79$).

[11]We also checked whether the odds ratio was significantly influenced by race or gender and it was not. We added an interaction with Long 5HTT*Attend and gender (male=1) which was not significant ($p = 0.25$) and also an interaction with Long 5HTT*Attend and race (white=1) which was not significant ($p = 0.32$).

[12]We also tested whether there was a significant gene-gene (GXG) interaction between 5HTT and MAOA by regressing Long 5HTT, High MAOA, Long*High, race controls, gender, and age on turnout. The estimated parameter on the interaction was insignificant ($p = 0.89$).

[13]A variable M mediates the relationship between an independent variable X, in our case a genotype, and a dependent variable Y, in our case voting, if (1) X significantly predicts Y, (2) X significantly predicts M, and (3) M

	Model 1		*Model 2*		*Model 3*		*Model 4*		*Model 5*		*Model 6*	
	OR	*SE*	OR	*SE*	OR	*SE*	OR	*SE*	OR	*SE*	OR	*SE*
High MAOA	**1.26**	**0.10**			**1.29**	**0.11**			**1.28**	**0.11**	**1.28**	**0.12**
High*Attend					0.99	0.14						
Long 5HTT			1.00	0.13			0.97	0.13	0.96	0.13	1.04	0.15
Long*Attend							**1.58**	**0.17**	**1.46**	**0.17**	**1.51**	**0.20**
Attend					2.17	0.10	1.46	0.15	1.59	0.16	1.28	0.18
Black	1.59	0.14	1.45	0.13	1.33	0.14	1.19	0.14	1.28	0.14	1.54	0.18
Hispanic	0.76	0.17	0.76	0.17	0.75	0.17	0.73	0.17	0.73	0.17	0.93	0.20
Asian	0.83	0.22	0.79	0.22	0.75	0.22	0.72	0.22	0.74	0.22	0.91	0.25
Nat Am	0.81	0.37	0.82	0.36	0.81	0.38	0.82	0.36	0.80	0.37	0.96	0.43
Age	1.13	0.03	1.12	0.03	1.13	0.03	1.12	0.03	1.13	0.03	1.06	0.04
Male	0.96	0.10	1.04	0.10	1.06	0.10	1.15	0.10	1.07	0.10	1.09	0.12
Partisan											3.66	0.12
Income											1.02	0.02
Cognitive											1.01	0.00
College											2.34	0.13
Intercept	0.05	0.66	0.07	0.66	0.05	0.67	0.06	0.67	0.05	0.68	0.02	0.80
N	2273		2290		2283		2265		2254		1800	
Deviance	3059		3092		2966		2936		2918		2125	
NullDeviance	3091		3117		3109		3081		3067		2458	

Table 1: Models of Association Between MAOA, 5HTT, and Voter Turnout. Variable definitions are in the appendix. All results are expressed in odds ratios (OR). Standard errors (SE) are also presented.

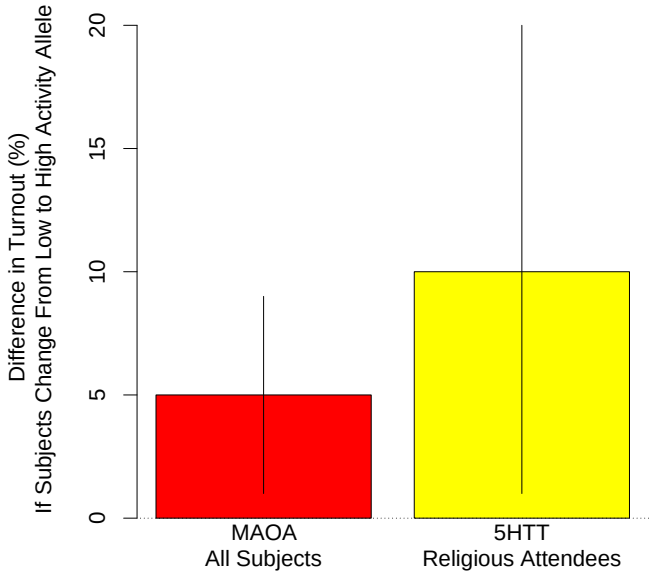

Figure 2: Changing MAOA and 5HTT to High Activity Allele Yields Significantly Higher Turnout. First differences in turnout simulated from the coefficient covariance matrix of *Model 1* and *Model 4*, assuming all other values are held at their means. Horizontal bar indicates 95% confidence interval.

ship, which is known to significantly influence political participation (Bartels 2000). We might also expect genes to contribute to variation in socioeconomic factors like income (Bowles & Gintis 2002), which in turn would yield greater participation. Also, several twin studies have suggested that variation in cognitive ability can be attributed to genetic factors (McGue & Bouchard 1998). If so, then variation in the ability to process political information, which has an impact on turnout (Verba, Schlozman & Brady 1995), may also be linked to genes. We can measure at least part of this ability using 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). Variation in educational attainment is another factor that has been found to be heritable (Baker, Treloar, Reynolds, Heath & Martin 1996, Heath, Berg, Eaves, Solaas, Corey, Sundet, Magnus & Nance 1985) and is frequently shown to influence turnout (Leighley & Nagler 1992*b*). In order to test whether these variables are potentially mediators, we regress each of them separately on High MAOA and Long 5HTT along with race, age, and gender controls. Since High MAOA and Long 5HTT are not significantly associated with any of these variables, we can rule them out as mediators.[14]

Even after including all of these variables in the model, both High MAOA and the interaction between Long 5HTT and attendance remain significant. We also observe something rather unexpected in *Model 6*—the main effect of religious service attendance ceases to be significant ($p = 0.17$). In other words, it appears that the direct effect of church attendance on voter turnout that has been reported in so many other studies (see Cassel 1999 for a review) may be driven by two factors: 1) a spurious association caused by the relationship between other correlates of turnout and religious service attendance, and 2) the previously unmodeled interaction between religious service attendance and the functioning of the serotonin transporter, 5HTT. To test this assertion we simply remove 5HTT from the *Model 6* specification (not shown). Religious attendance returns to exerting a significant direct effect on turnout ($p = 0.00001$). Our results show that individuals with the "short" 5HTT allele (the base category of the 5HTT variable) who are active in religious organizations are not more likely to vote. Similarly, individuals with the "long" allele who are not as active in religious organizations are not more likely to vote. In fact, voting is only higher among those who are *both* strongly exposed to the sense of community offered by religious groups

significantly predicts Y controlling for X (Baron & Kenny 1986).

[14]The p values for High MAOA and Long 5HTT in each regression are presented in the appendix.

(Cassel 1999) *and* potentially better equipped to handle the potential pain associated with social risks due to a fully-functioning serotonin metabolism conferred by a "long" 5HTT allele.

It is worthwhile pointing out how these results are reminiscent of the Caspi *et al.* (2002) findings on child abuse. Prior to that publication, scholars had reported a weak but significant relationship between receiving abuse as a child and abusing one's own children as an adult. What Caspi and his colleagues showed was that this weak effect was moderated by the MAOA gene. People with the "high" MAOA allele who suffered abuse as a child were not more likely to abuse their own children. Similarly, people with the "low" allele who had not been abused were not more likely to abuse their own children. It was only those who experienced the trauma of child abuse and who lacked the protective effect of a fully-functioning serotonin metabolism conferred by a "high" MAOA allele that continued the cycle of violence. In the case of both voting and child abuse, bringing genes into the study of social behavior not only highlights the role of biology—it clarifies and sharpens the effect of the environment.

Discussion

The results of this analysis are clear: we have found that two extensively studied genes are significantly associated with voter turnout. Further, these are the first two genes ever directly associated with political behavior. The empirical approach we employ in this paper improves on the twin study designs of Fowler, Baker & Dawes (2007) in a profoundly important way. Twin studies are valuable for determining the influence of genes over observed behavior, but they are agnostic about causality. By focusing on specific genes, our analysis is able to suggest potential causal pathways through which genes influence turnout. A significant body of research has found that the two genes we study, 5HTT and MAOA, influence social behavior via their impact on the serotonin metabolism and research within political science has identified prosociality as a significant determinant of turnout, thereby establishing a potential causal chain leading from these genes to observed political behavior. Again, we cannot test any causal pathway given our data so we are merely speculating based on previous work done in behavior genetics and political science.

More broadly, these results represent an important step for political science as a discipline. Specifically, they show that incorporating genetic information into our theories and analysis may

contribute to a greater understanding of political behavior. The environment-only approach used for so long in political science has frequently conceptualized human behavior as a "blank slate" on which any tendencies could be drawn, regardless of the unique biology of each individual (Pinker 2003). However, the results presented here refute the blank slate theory of political behavior. Although the environment is extremely important for turnout and other political acts, perhaps even more so than genes, we can no longer act as if genes do not matter at all. Genetic differences are likely to have important consequences for a whole range of political behaviors.

We believe the significant interaction we find between 5HTT and church attendance adds an important element missing from existing theories explaining the relationship between religious activity and turnout. Cassel (1999) suggests religious groups build a sense of belonging to a larger community, but any sense of belonging is likely mitigated by social anxiety or an aversion to potential social conflict. Brady, Verba & Schlozman (1995) argue that civic skills, acquired through church activities, increase the likelihood of voting. Specifically, they find that regular attendance prompts individuals to perform acts on behalf of their religious organization that enhance their civic skills, in turn better equipping them for involvement in the political process. These acts include writing a letter, taking part in decision-making, giving presentations or speeches, organizing or chairing a meeting, and/or contacting government officials on behalf of the church. However, the leap from church attendance to completing these acts is not a surety for every individual. These acts often require extensive interaction with others in a social setting, something that is likely to be uncomfortable for those with social anxiety and fear of social rejection. Even the simple act of writing a letter may be difficult when the person writing it knows others will evaluate it. Rosenstone & Hansen (1993) suggest that religious organizations provide a venue amenable to the discussion of politics, thus lowering the cost of gathering information. Since voting is less costly for regular attendees compared to those who do not attend, we should observe a higher turnout rate among this group. As in the case with the development of civic skills, engaging in political discussions with fellow members of one's church is likely difficult for those with antisocial tendencies. Therefore, if our hypothesis is correct, we would expect individuals with the "long" 5HTT allele to be most likely to benefit from political discussions in a church setting.

Even if one concedes genes do influence political behavior, it is tempting to assume that

since they are not causally proximate to observed behaviors they can be safely ignored for practical purposes. However, this thinking is mistaken. Genes are the institutions of the human body—they constrain individual behavior just as political institutions constrain the behavior of groups of people. In this article we demonstrate that possessing a particular gene is associated with voting activity. Even after controlling for factors known to influence turnout, having a high MAOA allele raises the likelihood of voting by about 5%. Among people active in their religious organizations, having a long 5HTT allele raises the likelihood of voting by about 10%. We theorize that since low efficiency MAOA and 5HTT alleles limit the degree to which individuals are socially oriented, these alleles inhibit their desire or ability to participate in the political process.

Our theory that genetic differences within a population, in part, explain variation in political behavior is in stark contrast to game-theoretic models of voter turnout that typically predict very little variation in participatory behavior (Aldrich 1993). In these models, when one person votes, everyone with the same preferences benefits from the increased likelihood that their preferred outcome will result. Yet those who do vote must bear the cost of time and effort required to learn about election alternatives and go to the polls. In large populations, the probability that a single vote will change the outcome of an election is miniscule (Riker & Ordeshook 1968, Gelman, King & Boscardin 1998), meaning that even very small costs to the individual typically outweigh the expected benefits he or she would receive from voting. As a result, classic models that assume individuals are self-interested and fully optimizing in their behavior show that the equilibrium amount of voter turnout approaches zero as the population becomes large (Palfrey & Rosenthal 1985). Yet in spite of this social dilemma, millions of people do vote, suggesting that something other than self-interest or optimizing behavior drives their decision (Bendor, Diermeier & Ting 2003, Feddersen & Sandroni 2006, Fowler 2006*b*, Fowler 2006*a*, Fowler & Kam 2007, Edlin, Gelman & Kaplan 2007, Jankowski 2002, Jankowski 2007). In addition, the fact that millions of people abstain concords with our finding here that there is inherent variation in the human tendency to participate. Future models of prospective voter behavior should account for this variation in predispositions.

One popular extension of these models is to assume that some individuals experience an extra benefit from voting that has nothing to do with the outcome (the "D" term as Riker &

Ordeshook (1968) called it). Instead, this benefit comes from the satisfaction of fulfilling a civic duty or of contributing to the democratic process (Blais & Young 1999). Alternatively, one might vote to fulfill a desire for expression (Schuessler 2000), which might be modelled as an 'E' term. In other words, these models posit that there is an additional factor that yields inherent heterogeneity in the desire to vote. While many scholars believe this line of argument is plausible (notably Aldrich (1993, page 266): "most of the action is probably in the intrinsic values of voting per se"), these models have failed to consider the possibility that this heterogeneity can be attributed to genes. Thus, our results suggest that a fruitful avenue for future research is to study whether or not expressiveness or feelings of civic duty intermediate the association between genes and political participation. In fact, at least one study suggests this may be the case for MAOA. Rosenberg, Templeton, Feigin, Lancet, Beckmann, Selig, Hamer & Skorecki (2006) show that the high activity allele of MAOA is associated with "straightforwardness" (frankness in expression) and "conscientiousness", suggesting two additional pathways between genes and voting.

In contrast to the theoretical literature, the empirical literature has embraced variation in turnout behavior with models that test dozens of explanatory variables (Plutzer 2002, Timpone 1998, Verba, Schlozman & Brady 1995). These models include: demographic factors like age (Strate, Parrish, Elder & Ford 1989), gender (Schlozman, Burns, Verba & Donahue 1995), race (Verba, Schlozman & Brady 1993), marital status (Stoker & Jennings 1995), education (Leighley & Nagler 1992*b*), income (Leighley & Nagler 1992*a*), occupational prestige (Nie, Powell & Prewitt 1969*a*, Nie, Powell & Prewitt 1969*b*), and home ownership (Highton & Wolfinger 2001); attitudinal and behavioral factors like interest in the campaign (Verba, Schlozman & Brady 1995), access to political information (DiMaggio, Hargittai & Neuman 2001), general political knowledge (Galston 2001), strength of partisanship (Huckfeldt & Sprague 1992), feelings of civic duty (Blais & Young 1999), internal and external efficacy (Finkel 1985), political trust (Hetherington 1999), church attendance (Cassel 1999), personal skill acquisition (Brady, Verba & Schlozman 1995), humanitarianism (Jankowski 2007), altruism (Fowler 2006*a*), and patience (Fowler & Kam 2006); social factors like interpersonal communication (McLeod, Scheufele & Moy 1999), social identification (Fowler & Kam 2007), group consciousness (Miller, Gurin & Gurin 1981), socialization (Cho 1999), the status of neighbors (Huckfeldt 1979), political disagreement (Mutz 2002), and so-

cial capital (Lake & Huckfeldt 1998); and institutional factors (Jackman & Miller 1995) like closeness of the election (Shachar & Nalebuff 1999), contact from political organizations (Wielhouwer & Lockerbie 1994), campaigns (Ansolabehere & Gerber 1994), civic education (Somit, Tanenhaus, Wilke & Cooley 1958), polling locations (Gimpel & Schuknecht 2003), and barriers to registration (Rosenstone & Wolfinger 1978). However, not one of these articles has considered the possibility that genes may account for this variation.

Genes may also help us to explain two well-known features of voting. First, parental turnout behavior has been shown to be one of the strongest predictors of turnout behavior in young adults (Plutzer 2002). Although this has previously been interpreted as the result of social influence, the findings here suggest it may also be due to the inheritance of particular alleles of genes like MAOA and 5HTT. Second, turnout behavior has been shown to be habitual—the majority of people either always vote or always abstain (Fowler 2006*b*, Gerber, Green & Shachar 2003, Green & Shachar 2000, Miller & Shanks 1996, Plutzer 2002, Verba & Nie 1972). Scholars previously interpreted this as the result of reinforcement learning, but given that genes like MAOA and 5HTT are fixed, it might also be largely due to inherent genetic variability within the population. Future longitudinal and family studies of voter turnout should investigate what role MAOA and 5HTT plays in the transmission of political behavior over time within individuals and between parents and children. In particular, it will be interesting to understand better why these two genes that affect the serotonin system behave differently—why MAOA is associated with behavior directly, while 5HTT interacts with exposure to social activity.

Future work should use genetic association studies to identify specific genes that are implicated in political behaviors and attitudes. Finding out which genes they are and what physical function they have will improve our understanding of the biological processes that underlie these complex social behaviors and may also shed light on their evolutionary origin (Fitzpatrick, Ben-Shahar, Smid, Vet, Robinson & Sokolowski 2005). It is important to emphasize that there is likely no single "voting gene"—the results presented here suggest that at least two genes do matter and there is some (likely large) set of genes whose expression, in combination with environmental factors, influences political participation. Finally, we offer a word of caution. Association studies like ours require further replication before their findings can be truly considered anything more than

suggestive, therefore more work needs to be done in order to verify and better understand the specific associations we have identified.

Appendix

Variable Definitions

High is an indicator variable for having two of the 336, 351, or 381bp alleles of the MAOA gene. *Long* refers to having at least one 528bp allele of the 5HTT gene. *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. We center this variable on the category at least a few times a month (never = -1, at least once a month = 0, more than once a month = 1). 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. *Income* is the log of the response to the question "Including all the income sources you reported above, what was your total personal income before taxes in [2000/2001]?" *Cognitive Ability* is the score on the Picture Vocabulary Test, which measures verbal intelligence. *College* is an indicator variable taking the value 1 if the respondent completed at least one year of college and 0 for no college. It is based on the question "What is the highest grade or year of regular school you completed?"

Summary Statistics

	Percent
Vote	45.7
White	72.3
Black	19.2
Native American	2.2
Hispanic	12.3
Male	47.8
High MAOA	48.1
Long 5HTT	81.4
Partisan	37.3
College	55.5

Table 2: Percentage of subjects exhibiting these characteristics.

	Mean	Std Dev
Age	21.9	1.7
Income	12912	13926
Cognitive Ability	99.12	13.94
Attend	2.1	0.8

Table 3: Sample means.

Race / Ethnicity		Percent
White		
	Vote	44.6
	Long 5HTT	81.6
	High MAOA	53.4
Black		
	Vote	52.5
	Long 5HTT	91.6
	High MAOA	33.2
Hispanic		
	Vote	39.6
	Long 5HTT	76.0
	High MAOA	46.1
Asian		
	Vote	40.9
	Long 5HTT	55.2
	High MAOA	31.9
Nat Am		
	Vote	63.5
	Long 5HTT	73.4
	High MAOA	55.8

Table 4: Percentage of subjects exhibiting these characteristics by race.

Potential Mediators

DV	*High MAOA* p	*Long 5HTT* p
Partisan	0.41	0.36
Income	0.84	0.77
Cognitive Ability	0.09	0.35
College	0.91	0.95

Table 5: Test of High MAOA and Long 5HTT as potential mediators. Table presents p values for High MAOA and Long 5HTT in models with partisanship, income, cognitive ability, and college attendance as dependent variables. Regressions also include race, age, and gender controls.

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