

The MAOA Gene Predicts Credit Card Debt *

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Abstract

Economists have long realized the importance of credit markets and borrowing behavior for household finance and economics more generally. However, none of this previous work has explored the role of biological constraints. Here we present the first evidence of a specific gene that may influence borrowing behavior. Using data from the National Longitudinal Study of Adolescent Health, we show that individuals with a polymorphism of the MAOA gene that has lower transcriptional efficiency are significantly more likely to report having credit card debt. Having one or both MAOA alleles of the low efficiency type raises the average likelihood of having credit card debt by 7.8% and 15.9% respectively. About half of our population has one or both MAOA alleles of the low type. The results suggest that economists should integrate innate propensities into economic models and consider the welfare consequences of possible discrimination by lenders on the basis of genotype.

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1 Introduction

The practical and theoretical importance of credit card debt cannot be overstated. In testimony to its widespread nature some 180 million Americans currently have a credit card (Lusardi and Tufano 2009) of which approximately half regularly carry unpaid credit card debt (Sprenger and Stavins 2008). Its importance has sparked renewed interest in the study of household finance and high-cost borrowers in particular (Campbell 2006, Agarwal, Driscoll, Gabaix and Laibson 2008, Tufano, Maynard and De Neve 2008, Lusardi and Tufano 2009, Zinman 2009). The variables used to explain variation in credit card usage revolve principally around age, gender, ethnicity, income levels, employment, and financial literacy.

Because credit card debt is generally viewed as a form of present-biased decision making it has also received the attention of those economists studying intertemporal choices and discounting (Laibson, Repetto and Tobacman forthcoming, Agarwal, Skiba and Tobacman 2009). They find that present-biased preferences correlate with credit card borrowing (Meier and Sprenger forthcoming). A number of such studies also find that individual variation in the propensity to make impulsive, present-biased decisions is associated with specific cognitive functions. For example, individual differences in valuing immediate and delayed monetary rewards can be traced to separate neural systems (McClure, Laibson, Loewenstein and Cohen 2004) and processes in the anterior prefrontal cortex, a region in the brain shown to support the integration of diverse information (Shamosh, DeYoung, Green, Reis, Johnson, Conway, Engle, Braver and Gray 2008).

While it is possible that differences in brain activity result from development or environmental factors, there is a growing body of evidence that suggests some of these differences are influenced by genes. Recent studies using twin design research techniques have been able to gauge the explanatory power of both genes and environment, and they have shown that genes play an important role in other-regarding preferences and risk-taking (Cesarini, Dawes, Johannesson, Lichtenstein and Wallace 2009), the tendency to cooperate (Cesarini, Dawes,

Fowler, Johannesson, Lichtenstein and Wallace 2008), investment decisions (Cesarini, Johannesson, Lichtenstein, Sandewall and Wallace forthcoming), political preferences (Alford, Funk and Hibbing 2005, Hatemi, Medland, Morley, Heath and Martin 2007), voting behavior (Fowler, Baker and Dawes 2008), and happiness levels (Weiss, Bates and Luciano 2008).

Although twin studies are an important first step in establishing the role of genes for a particular behavior, they do not identify the specific genes involved. But the rising availability of DNA analyses now allows us to test hypotheses about targeted genes and their effects. For example, social scientists have recently shown that specific gene variants are associated with dictator game giving (Knafo, Israel, Darvasi, Bachner-Melman, Uzevovsky, Cohen, Feldman, Lerer, Laiba, Raz and et al 2008), punishment behavior in public goods games (McDermott, Tingley, Cowden, Frazetto and Johnson 2009), and political behavior and attitudes (Fowler and Dawes 2008, Dawes and Fowler 2009, Settle, Dawes, Christakis and Fowler forthcoming).

For borrowing behavior, the natural place to start the search for such genes is among those that have already been shown to account for variation in related behaviors. Among these, MAOA is a prime candidate. The MAOA gene encodes monoamine oxidase A, an enzyme that degrades neurotransmitters such as serotonin, dopamine, and epinephrine (adrenaline) in parts of the brain that regulate impulsiveness and cognitive ability (Hariri, Drabant, Munoz, Kolachana, Mattay, Egan and Weinberger 2005, Meyer-Lindenberg, Buckholtz, Kolachana, Hariri, Pezawas, Blasi, Wabnitz, Honea, Verchinski, Callicott, Egan, Mattay and Weinberger 2006, Eisenberger, Way, Taylor, Welch and Lieberman 2007). MAOA has been studied for more than twenty years and much is known about the way different versions of this gene regulate transcription, metabolism, and signal transfers between neurons, all of which have behavioral effects (Craig 2007). In particular, the less transcriptionally efficient alleles of this gene have been associated with a variety of impulsive and addictive behaviors, as well as a lack of conscientiousness (Walderhaug, Lunde, Nordvik, Landro, Refsum and Magnusson 2002, Saito, Lachman, Diaz, Hallikainen, Kauhanen and et al 2002, Contini,

Marques, Garcia, Hutz and Bau 2006, Passamonti, Fera, Magariello, Cerasa, Gioia, Muglia, Nicoletti, Gallo, Provinciali and Quattrone 2006, Rosenberg, Templeton, Feigin, Lancet, Beckmann, Selig, Hamer and Skorecki 2006, Guo, Ou, Roettger and Shih 2008, McDermott et al. 2009). As a result, economists have specifically identified MAOA as a candidate gene for further study (Benjamin, Chabris, Glaeser, Gudnason, Harris, Laibson, Launer and Purcell 2007).

Because credit card debt is a relatively expensive form of debt, our prior intuition is that, all other things being equal, it would be used more by those individuals seeking immediate gratification, displaying less consideration of future consequences, and reduced information processing. Hence, we hypothesize that people with less transcriptionally efficient alleles of the MAOA gene are more likely to accrue credit card debt. Although recent studies have already shown that a large fraction of the variation in economic behavior 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 the goal of this article is to show association rather than causality. Using data from the National Longitudinal Study of Adolescent Health (Add Health), we conduct gene association tests on the relationship between MAOA and credit card debt. The results indicate that the MAOA gene is significantly associated with the reporting of credit card debt. To our knowledge, this is the first article to show a specific gene variant is associated with real world borrowing behavior.

2 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 (except for rare mutations), they represent the purest measure of biological inheritance

and they can 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 and other genes.

3 MAOA, Serotonin, and Behavior

In order to study the genetic component of a behavioral outcome, 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 transcriptional factors (Damberg, Garpenstrand, Hallman and Oreland 2001, Benjamin et al. 2007).

To study whether genes affect economic behavior we chose a candidate gene that has already received a great deal of attention for its association with behavioral traits. The MAOA gene is critical to the metabolism of serotonin and other neurological processes 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 and 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. 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 gene responsible for transcribing MAOA is eponymous—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 in response to social stressors (Kraemer, Ebert, Schmidt and McKinney 1989) and studies of rodents show that acute emotional stress affects the way MAOA breaks down serotonin in several areas of the brain (Popova, Voitenko and Maslova 1989, Virkkunen, Goldman, Nielsen and Linnoila 1995). In mice, knock-out studies that eliminate the MAOA gene in subjects cause enzymatic activity to come to a complete halt (Cases, Seif, Grimsby, Gaspar and Chen 1995). MAOA has also been shown to alter the structure of the brain in mice

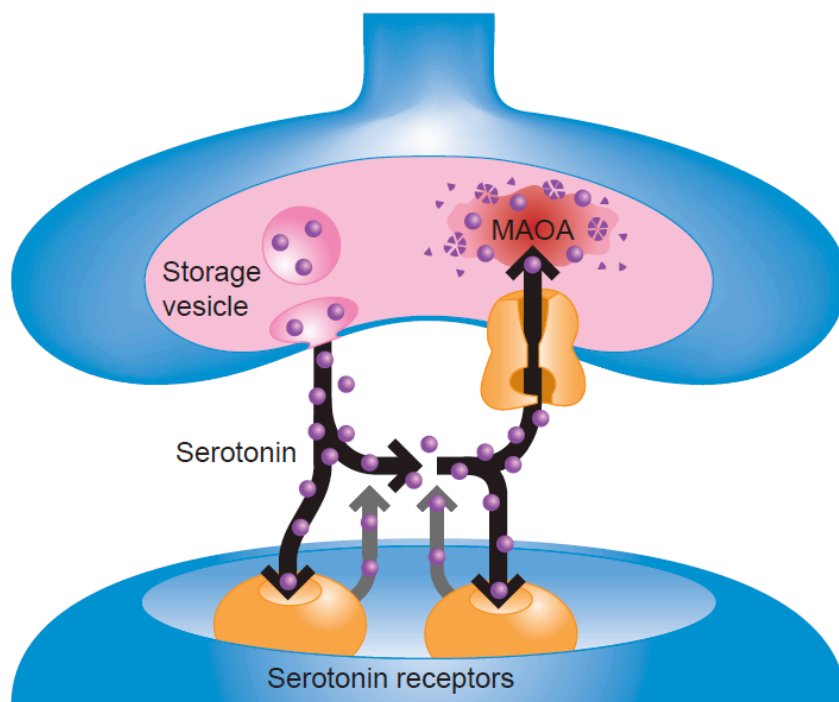


Figure 1: Simple representation of the release, reception, and recycling of serotonin in neurons

(Cases, Vitalis, Seif, De Maeyer, Sotelo and Gaspar 1996). There is strong evidence that the serotonin system affects complex social traits in humans (Balciuniene and Jazin 2001). For example, the serotonin function has been associated with aspects of impulsivity, such as reward sensitivity and inhibitory cognitive control (Walderhaug et al. 2002, Cools, Blackwell, Clark, Menzies, Cox and Robbins 2005), and is also related to prefrontal cortex activity (Rubia, Lee, Cleare, Tunstall, Fu, Brammer and McGuire 2005).

MAOA has a 30 base-pair VNTR polymorphism located in the promoter region. The “low” version of this polymorphism significantly decreases the transcriptional efficiency of MAOA (Sabol, Hu and Hamer 1998, Denney, Sharma, Dave and Waguespack 1994, Denney, Koch and Craig 1999). The less transcriptionally efficient alleles of MAOA have been linked to impulsive and addictive behavior, as well as attention deficit disorder, all of which appear to be mediated by certain parts of the brain (Lawson, Turic, Langley, Pay, Govan and et al. 2003, Domsche, Sheehan, Lowe, Kirley, Mullins and et al 2005, Contini et al. 2006). 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 and Thomas 2003).

Not all studies show a direct relationship between genetic variation 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 impulsive and violent behavior (Caspi, McClay, Moffitt, Mill, Martin, Craig, Taylor and Poulton 2002, Foley, Eaves, Wormley, Silberg, Maes and et al. 2004, Haberstick, Lessem, Hopfer, Smolen, Ehringer, Timberlake and Hewitt 2005, Kim-Cohen, Caspi, Taylor, Williams, Newcombe and 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. In these studies the gene itself was not associated with the behavior once the interaction with environment was included in the association test. Here we show evidence for a *direct* association between the number of low MAOA alleles and the reporting of credit

card debt. However, future studies may show that this direct association is moderated by environmental factors.

4 The Add Health Data

Our analysis is based on individual-level genetic and survey data collected as part of 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. It has been employed widely across disciplines and recently it has produced important contributions in economics (Echenique, Fryer and Kaufman 2006, Alcott, Karlan, Mobius, Rosenblat and Szeidl 2007, Norton and Han 2009). 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%.¹ 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 and Rowe 1998). Allelic information for six genetic markers are available for 2,574 individuals as part of Wave III, including markers that identify alleles of MAOA. Details of the DNA 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 (Denney et al. 1994, Sabol et al. 1998). 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.

In Wave III, subjects were asked “Do you have any credit card debt?” About 41% answered in the affirmative. While this question gives us a valuable opportunity to explore the genetic basis of credit card usage, we want to make clear two limitations of the data. First, it would be preferable to have verifiable information about the actual amount of credit card debt. Second, it would also be preferable to have information about the credit card use of older adults. The Add Health sample is restricted to individuals who are 18-26 years old during Wave III, so it is possible that our results apply only to financial decision-making by

¹A breakdown for those providing DNA samples is presented in the appendix.

young adults and not to people in different age categories.

5 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. However, a significant association can mean one of three things: (1) The allele itself influences credit card use; (2) the allele is in “linkage disequilibrium” with an allele at another locus that influences credit card use; 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. Financial decision-making in these groups may be the product of their environments, alleles other than the one of interest, or some unobserved reason. 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.² 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 handle the problem of population stratification by using family members, such as parents or siblings, as controls. Tests using family data compare

²Controls may be randomly selected from the population or those known not to exhibit the trait.

whether offspring exhibiting the trait receive a risk allele from their 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 and 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.

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

$$P[Y_{ij} = 1 | Z_{kij}, U_j] = \text{logit}(\beta_0 + \beta_G G_{ij} + \beta_k Z_{kij} + U_j)$$

where i and j index subject and family respectively. For the MAOA gene, $G = 2$ if the subject’s genotype is LL, $G = 1$ for genotypes HL or LH, and $G = 0$ if the subject’s genotype is HH (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). Z is a matrix of variables to control for underlying population structure of the Add Health samples as well as potentially mediating factors such as age, gender, income, and education that may all influence financial decision-making. U is a family random effect that accounts for multiple observations among siblings from the same family. The coefficient β_G tests the association between each additional low allele of the MAOA gene and the tendency to report credit card debt. The coefficients are reported as odds ratios, so the null hypothesis is that $\beta_G = 1$ (that is, low alleles of the MAOA gene do not increase the odds of reporting credit card debt).

To control for the effects of the underlying population structure, we include indicator variables for whether a subject self-reported as Black, Hispanic, or Asian (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.

	<i>Model 1</i>			<i>Model 2</i>			<i>Model 3</i>		
	OR	<i>SE</i>	P-value	OR	<i>SE</i>	P-value	OR	<i>SE</i>	P-value
MAOA Low	1.13	0.06	0.015	1.18	0.07	0.003	1.16	0.08	0.049
Black	0.86	0.11	0.248	0.93	0.14	0.609			
Hispanic	0.90	0.35	0.790	1.04	0.43	0.883			
Asian	0.96	0.22	0.826	0.91	0.19	0.727			
Age	1.25	0.03	0.000	1.22	0.04	0.000	1.28	0.05	0.000
Male	0.73	0.06	0.000	0.70	0.07	0.000	0.79	0.10	0.054
Income				1.00	0.00	0.048	1.00	0.00	0.130
College				1.75	0.18	0.000	1.46	0.20	0.006
Intercept	0.01	0.58	0.000	0.01	0.67	0.000	0.00	0.87	0.000
<i>N</i>	2528			2077			1197		
<i>PseudoR2</i>	0.030			0.049			0.044		

Table 1: Models of Association Between MAOA and Credit Card Debt. Variable definitions are in the appendix. All results are expressed in odds ratios (OR). Standard errors (SE) and P-values are also presented.

6 Results

Table 1 shows the results of several specifications of the models to test the hypothesis that the MAOA gene is associated with reporting credit card debt. Each of these specifications includes variables for age, gender, and race to control for population stratification. *Model 1* shows that the low allele of MAOA is significantly associated with increased credit card debt ($p = 0.015$). *Model 2* adds controls for income and education. This model suggests that the odds of a person reporting credit card debt are increased by a factor 1.18 for each additional low allele of the MAOA gene. *Model 2* also strengthens our confidence in these statistical results ($p = 0.003$).

Following Xu and Shete (2006), as a robustness test for population stratification, we also include *Model 3* that is a case-control association model for those subjects that uniquely identified themselves as being white. The coefficient on MAOA and its p-value ($p = 0.049$) suggest that population stratification between self-reported racial categories is not driving the association between MAOA and credit card debt.

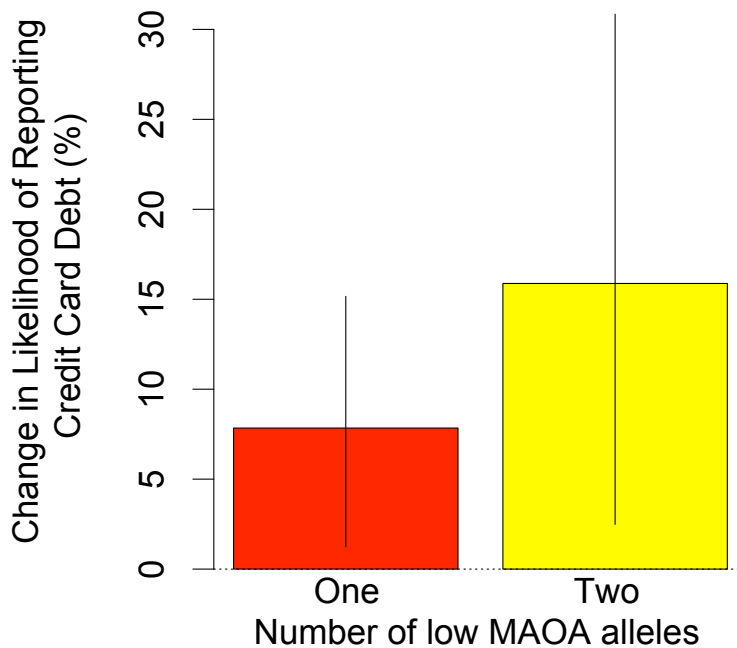


Figure 2: Increasing the number of low activity MAOA alleles yields significantly higher credit card debt. First differences, based on simulations of *Model 1* parameters, are presented along with 95% confidence intervals. All other variables are held at their means.

In Figure 2 we summarize our results for MAOA by simulating first differences from the coefficient covariance matrix of *Model 1*. Holding all else constant and changing the MAOA gene of all subjects from zero to one low allele would increase the reporting of credit card debt in this population by about 7.8%. Similarly, changing the MAOA gene from zero to two low alleles would increase the reporting of credit card debt by about 15.9%.

Model 2 includes a number of socio-economic factors that influence financial decision-making. Income and education may in fact *mediate* the relationship between the genes we have identified and credit card usage.³ We might also expect genes to contribute to variation in socioeconomic factors like income (Bowles and Gintis 2002), which in turn would impact financial decision-making. Also, several twin studies have suggested that variation

³A variable M mediates the relationship between an independent variable X , in our case a genotype, and a dependent variable Y , in our case credit card debt, if (1) X significantly predicts Y , (2) X significantly predicts M , and (3) M significantly predicts Y controlling for X (Baron and Kenny 1986).

in cognitive ability can be attributed to genetic factors (McGue and Bouchard 1998). If so, then variation in the ability to process financial information may also be linked to genes. Variation in educational attainment is a factor that has been found to be heritable (Baker, Treloar, Reynolds, Heath and Martin 1996, Heath, Berg, Eaves, Solaas, Corey, Sundet, Magnus and Nance 1985) and is frequently shown to influence household finance. In order to test whether these variables are potentially mediators, we regress each of them separately on MAOA low along with race, age, and gender controls. We perform the same mediation test for a set of additional controls that are married, divorced, religiosity, educational debt, having a job, and parents' income. Since MAOA low is not significantly associated with any of these variables, we can rule them out as mediators.⁴

7 Discussion: Theory and Welfare Implications

The above results on genetic variation in credit card debt may have important theoretical and welfare implications. The theoretical contribution of the MAOA finding lies in providing new explanatory power for estimating discount functions and understanding intertemporal choice. In the discounting literature, credit card debt is a favorite indicator as it presents a real-world measure of individuals' time preferences (Tobacman 2009, Laibson et al. forthcoming). For example, Meier and Sprenger (forthcoming) present the results of a large field study that shows that present-biased time preferences correlate with credit card borrowing. Earlier theoretical work in behavioral economics argued that greater present bias would predict levels of credit card borrowing as impatience leads to having higher discount rates for delayed rewards (Laibson 1997, Fehr 2002, Heidhues and Koszegi forthcoming). In particular, we believe that the MAOA finding builds on the neuroscientific work by McClure et al. (2004) that identified neural systems involved in valuing immediate and delayed monetary rewards. Our results suggest that some of the variation in these systems may result from differences in MAOA genotype. Genes are upstream from the neurological processes that McClure

⁴The p values for MAOA low in each regression are presented in the appendix.

et al. (2004) identified and may thus bring us even closer to understanding the sources of intertemporal choice.

More broadly, these results suggest that integrating the unique biology of each individual, in addition to studying the socio-economic environment, may fine-tune existing models of financial and other economic decisions. Of course, genes would have no influence on debt behavior if credit cards were not available. So in some sense, the environment should still be the primary focus of economic inquiry. However, the evidence we report here suggests that in the context of a particular environment (the availability of credit cards), different genotypes can yield different outcomes. Even if they could carry credit card debt, many people do not, and this is at least partly due to genetic differences. As a consequence, models that assume away inherent biological differences may suffer from omitted variable bias.

The results also suggest that genes may be used to identify consumers who are likely to be profitable clients, and economists should consider the welfare consequences of allowing credit companies to discriminate on the basis of a person's genotype. We hope that our finding stimulates a debate that will put in place a policy framework that prevents *any* kind of genetic discrimination prior to individuals' genomes being easily available. On 21 May 2008 the Genetic Information Nondiscrimination Act of 2008 became a federal law in the United States, but it only protects people from discrimination by health insurers and employers. The results here suggest that these protections should be extended to prevent discrimination by lenders as well.

8 Conclusion

In this article we have shown that an extensively studied gene is significantly associated with the reporting of credit card debt. Further, this is the first gene to be directly associated with real world borrowing behavior. The empirical approach we employ in this paper improves on twin study designs in a profoundly important way. Twin studies are valuable for measuring the influence genes have on an 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 economic decision-making. A significant body of research has found that the MAOA gene influences individual behavior via its impact on the serotonin metabolism and other neurological processes, thereby establishing a potential causal chain leading from this gene to observed economic behavior. Given prior research linking the low efficiency alleles of the MAOA gene to impulsivity and addiction we hypothesized that its carriers would be more susceptible to high-cost borrowing, and this intuition is verified in the data.

Future work should use genetic association studies to identify other specific genes that are implicated in economic behaviors. 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 and Sokolowski 2005). While the MAOA gene may explain a significant portion of the variation in credit card debt, it is important to emphasize that there is no single “credit card debt gene.” Instead, there is likely to be a set of genes whose expression, in combination with environmental factors, influences financial decision-making.

Finally, we offer a word of caution. Association studies like ours require replication before their findings can be truly considered anything more than indicative, therefore more work needs to be done in order to verify and better understand the specific association we have identified.

Appendix

Variable Definitions

MAOA Low is an indicator variable for having 0, 1, or 2 of the 291 or 321bp alleles of the MAOA gene. The *race/ethnicity* indicator variables are 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 response to the question “Including all the income sources you reported above, what was your total personal income before taxes in [2000/2001]?” *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?”

In Tables 2-5 we report on descriptive statistics and present a raw cross tabulation with a Pearson chi-squared test of the relationship between credit card debt and MAOA. The chi-squared test shows that the null hypothesis of no relationship is highly unlikely ($p = 0.003$). This test however is only meant to be illustrative since it does not take into account family structure or population stratification, both of which are dealt with in the mixed-effects logistic regression reported in the main text.

Descriptive Statistics

Table 2: Sample means

	Mean	Std Dev	Min	Max
MAOA Low	0.82	0.86	0	2
Age	21.9	1.7	18	26
Income	12,912	13,926	0	250,000

Table 3: Percentage of subjects exhibiting these characteristics

	Percent
Credit Card Debt	41.3
White	70.9
Black	19.0
Hispanic	14.7
Asian	8.2
Male	47.8
College	54.9

Table 4: Cross-tabs and chi-squared test

Credit Card Debt	MAOA Low			Total
	0	1	2	
No	751 <i>50.3%</i>	315 <i>21.1%</i>	427 <i>28.6%</i>	1,493 <i>100%</i>
Yes	459 <i>43.9%</i>	268 <i>25.6%</i>	319 <i>30.5%</i>	1,046 <i>100%</i>
Total	1,210 <i>47.6%</i>	583 <i>23.0%</i>	746 <i>29.4%</i>	2,539 <i>100%</i>

Pearson chi-squared = 11.5; Pr = 0.003

Table 5: Credit card debt and genotype, by race

Race		Mean
White	Credit Card Debt	0.42
	MAOA Low	0.69
Black	Credit Card Debt	0.38
	MAOA Low	1.09
Hispanic	Credit Card Debt	0.40
	MAOA Low	0.63
Asian	Credit Card Debt	0.44
	MAOA Low	1.16

Potential Mediators

Table 6: Tests of MAOA low as potential mediator

DV	<i>MAOA Low</i> <i>p – value</i>
Income	0.44
College	0.59
Job	0.21
Married	0.17
Divorced	0.20
Religious	0.27
Educational Debt	0.49
Parents’ Income	0.36

Note: table presents p values for MAOA low in models with income, college attendance, job, married, divorced, religious, educational debt, and parents’ income as dependent variables. Regressions also include race, age, and gender controls.

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