



Credit card borrowing and the monoamine oxidase A (MAOA) gene



Jan-Emmanuel De Neve^{a,b,*}, James H. Fowler^c

^a School of Public Policy, University College London, 29/30 Tavistock Square, London WC1H 9QU, UK

^b Centre for Economic Performance, London School of Economics, Houghton Street, London WC2A 2AE, UK

^c Division of Social Sciences and School of Medicine, University of California, San Diego, 9500 Gilman Drive, La Jolla, CA 92093-0521, USA

ARTICLE INFO

Article history:

Received 7 February 2013

Received in revised form 27 February 2014

Accepted 3 March 2014

Available online 18 April 2014

JEL classification:

D14

D91

G02

Keywords:

Credit card borrowing

Present-biased time preferences

MAOA gene

Genetic association study

ABSTRACT

Using a discovery and replication sample from a U.S. representative data set, we show that a functional polymorphism on the MAOA gene is associated with credit card borrowing behavior. For the combined sample of approximately 12,000 individuals we find that having one or both MAOA alleles of the less transcriptionally efficient type raises the average likelihood of reporting credit card debt by about 4%. These results suggest that behavioral models benefit from integrating genetic variation and that economists should consider the welfare consequences of possible discrimination by lenders on the basis of genotype.

© 2014 Elsevier B.V. All rights reserved.

1. Introduction

The practical and theoretical importance of credit card debt cannot be overstated. Some 180 million Americans currently have a credit card (Lusardi and Tufano, 2009) and approximately half regularly carry unpaid credit card debt (Sprenger and Stavins, 2008). The ubiquity of credit card debt has sparked renewed interest in the study of household finance and high-cost borrowers in particular (Campbell, 2006; Agarwal et al., 2008; Lusardi and Tufano, 2009; Zinman, 2009; Tufano et al., 2011). The variables used to explain variation in credit card usage typically include 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 (Agarwal et al., 2009; Laibson et al., 2007). They find that present-biased preferences correlate with credit card borrowing (Meier and Sprenger, 2010). 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

* Corresponding author at: School of Public Policy, University College London, 29/30 Tavistock Square, London WC1H 9QU, UK. Tel.: +44(0)20 7679 4949. E-mail address: jdeneve@post.harvard.edu (J.-E. De Neve).

to separate neural systems (McClure et al., 2004) and processes in the anterior prefrontal cortex, a region in the brain shown to support the integration of diverse information (Shamosh et al., 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 suggest that genetic variation plays an important role in a number of behaviors including financial choices (Cesarini et al., 2010; Kuhnen et al., 2013), other-regarding preferences and risk-taking (Cesarini et al., 2009), the tendency to cooperate (Cesarini et al., 2008), educational attainment (Rietveld et al., 2013), political behavior (Alford et al., 2005; Hatemi et al., 2007; Fowler et al., 2008), leadership role occupancy (De Neve et al., 2013), and psychological well-being (Weiss et al., 2008; De Neve, 2011; De Neve et al., 2012; Rietveld et al., 2013).

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. The increasing availability of DNA analyses now allows us to test hypotheses about targeted genes and their effects. For example, recent lab experiments have linked specific gene variants to dictator game giving (Knafo et al., 2008), cooperation (Mertins et al., 2011), financial risk-taking in men (Dreber et al., 2009; Kuhnen and Chiao, 2009; Frydman et al., 2011), sensitivity over gains and losses (Zhong et al., 2009), and attitudes towards long-shot risks (Zhong et al., 2009). Kuhnen et al. (2013) find a significant association between a functional polymorphism in the serotonin transporter gene (5-HTTLPR) and the number of credit lines opened in a sample of 31 participants for whom credit reports were obtained. These genetic associations remain only suggestive in light of the fact that they have not been shown robust to replication studies and remain open to confounding. These results are obtained in relatively small sample sizes that do not typically have sufficient statistical power to reliably detect the small effects of particular genetic variations (Beauchamp et al., 2011; Benjamin et al., 2012).

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 et al., 2005; Meyer-Lindenberg et al., 2006; Eisenberger et al., 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 et al., 2002; Saito et al., 2002; Contini et al., 2006; Passamonti et al., 2006; Rosenberg et al., 2006; Guo et al., 2008; McDermott et al., 2009). As a result, economists have specifically identified MAOA as a candidate gene for further study (Benjamin et al., 2007) and have studied its relationship with attitudes towards risk-taking in a lab setting (Zhong et al., 2009; Frydman et al., 2011).

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.

In line with the literature in behavioral genetics, it is crucial to point out at the outset that this article does not estimate causal coefficients. 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. Association studies like ours require independent replication before the findings can be truly considered anything more than suggestive. The staggered release of genotypical data by Add Health provided the opportunity to test the association in both a discovery and a replication stage. Generally, the results indicate that the MAOA gene is significantly associated with the reporting of credit card debt. To our knowledge, this is the first large-scale study to show a specific genotype is associated with real-world borrowing behavior yet more work needs to be done in order to verify and better understand the specific association we have identified.

2. Basic genetics concepts

Human DNA is composed of an estimated 21,000 genes that form the blueprint for molecules that regulate the development and function of the human body. Genes are distinct regions of human DNA that are placed in the 23 pairs of chains, or chromosomes, that make up all human DNA. Almost all human cells contain the same inherited DNA chains that develop from the moment of 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 or BA—order is irrelevant), 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). If an individual is heterozygous at a locus, a “dominant” allele may impose itself on the “recessive” allele and the expression of the latter allele will not be observed.

Genes transcribe proteins that 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, Huntington disease, and cystic fibrosis), the vast majority of phenotypes are “polygenic”, meaning they are influenced by multiple genes (Mackay, 2001; Plomin et al., 2008), and are shaped by a multitude of environmental forces. As a result, association models between genotypes and phenotypes are an important first step, 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 gene

In order to study the genetic component of a behavioral outcome, scientists often start with “candidate” genes that are known to influence related behaviors or processes in the body. For economic behavior, this means focusing on genes that affect brain development, neurotransmitter synthesis and reception, hormone regulation, and transcriptional factors (Damberg et al., 2001; Benjamin et al., 2007).

To study whether genes affect credit card borrowing behavior we chose a candidate gene that has already received much attention for its association with behavioral traits. The MAOA gene is responsible for transcribing an enzyme called monoamine oxidase A that is critical to the metabolism of serotonin and other neurological processes in the brain. In particular, the body’s homeostatic response to excess serotonin is to reabsorb it into the emitting or pre-synaptic neuron. Once the reuptake of serotonin is complete, MAOA degrades the serotonin so that its components can be reabsorbed in the cell.

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 et al., 1989) and studies of rodents show that acute emotional stress affects the way MAOA breaks down serotonin in several areas of the brain (Popova et al., 1989; Virkkunen et al., 1995). In mice, knock-out studies that eliminate the MAOA gene in subjects cause enzymatic activity to come to a complete halt (Cases et al., 1995). MAOA has also been shown to alter the structure of the brain in mice (Cases et al., 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 et al., 2005), and is also related to prefrontal cortex activity (Rubia et al., 2005).

The MAOA gene has a variable-number tandem repeat (VNTR) polymorphism¹ in its promoter region² that is responsible for variation in transcriptional efficiency. The VNTR on the MAOA gene consists of repeat variations that result in either a 291, 321, 336, 351, or 381 base-pair fragment size. The 291 and 321 base-pair alleles have lower transcriptional efficiency than the 336, 351, and 381 base-pair alleles (Denney et al., 1994; Sabol et al., 1998). Following the literature, we group the 291 and 321 base-pair alleles to form a “low” transcription group and the 336, 351, and 381 base-pair alleles to form a “high” transcription group (Caspi et al., 2002; Haberstick et al., 2005; Frazzetto et al., 2007; Fowler and Dawes, 2008; McDermott et al., 2009). This functional polymorphism of the MAOA gene produces the same protein but the short allele is associated with less basal activity than the high allele. Consequently, the short variant produces significantly less MAOA mRNA³ and protein (Sabol et al., 1998; Denney et al., 1994, 1999). Contrary to a single nucleotide polymorphism (SNP)—that relates to variation at a single base-pair location on the genome—a structural variation such as a VNTR covers a broader fragment of the genome and is understood to have potentially greater phenotypical influence (Redon et al., 2006; Can et al., 2011).

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 et al., 2003; Domsche 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 et al., 2003).

The MAOA gene is located on the X variant of chromosome 23. As such, the distribution of alleles is gender specific—unlike genes located on the other chromosomes. Males will have only one MAOA allele (their other chromosome is a Y variant), whereas females will have two MAOA alleles. As a result, males will necessarily be homozygous for either the high or low MAOA polymorphism. On the other hand, females can be heterozygous and have both a high and a low allele. The enzymatic activity of the number of alleles is not additive, hence the heterozygous females cannot be characterized with certainty (Caspi et al., 2002). We therefore follow the recent literature by grouping heterozygous females with the low transcription group (Fan et al., 2003; Frazzetto et al., 2007; Fowler and Dawes, 2008).

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 interaction

¹ A VNTR polymorphism is a repeated segment of DNA that varies among individuals in a population.

² 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.

³ Messenger ribonucleic acid (mRNA) is a type of RNA that carries information from DNA to ribosomes. In turn, these ribosomes “read” messenger RNAs and translate their information into proteins.

has been identified in many cases for impulsive and violent behavior (Caspi et al., 2002; Foley et al., 2004; Haberstick et al., 2005; Kim-Cohen 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 anti-social 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 MAOA genotype and the reporting of credit card debt. However, future studies may show that this direct association is also moderated by environmental factors (for example, variation in local credit markets).

4. Data and methods

4.1. Add Health data

This research is based on genetic and survey data collected as part of The National Longitudinal Study of Adolescent Health (Harris et al., 2009). Add Health is a study that explores the causes of health-related behavior of adolescents in grades 7–12 and their outcomes in adulthood. It has been employed widely across disciplines and it has produced important contributions in economics (Echenique and Fryer, 2007; Alcott et al., 2007). 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 14,738 students from the Wave I sample and a follow-up telephone survey of the school administrators. Wave III (2001–2002) consisted of an in-home interview of 15,170 Wave I participants. The result of this sampling design is that Add Health is a nationally representative study. Allelic information for a number of genetic markers were collected for 2574 individuals as part of Wave III. These particular candidate genes were chosen because they were known to affect brain development, neurotransmitter synthesis and reception, and hormone regulation. Details of the DNA collection and genotyping process are available at the Add Health website (Add Health Biomarker Team, 2007). Genotypical information includes markers that identify alleles of the MAOA polymorphism. The conventional grouping of these alleles (described above) results in a MAOA “low” group that represents 52% of our sample and the “high” group representing 48%. In 2012, Add Health released a second batch of genotypical data that extended to almost all Wave III participants and included the MAOA polymorphism. The distributional frequencies of this new sample (used here for replication purposes) aligns with the earlier discovery sample.⁴

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 antecedents 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 young adults and not to people in different age categories. Jassim and Taylor (2010) report using Sallie Mae data that a vast majority of college students held at least one credit card in 2008 with an average balance of \$3173. The study by Jassim and Taylor (2010) finds that undergraduate students’ credit card debt levels are similar to that of the general public. Furthermore, their study indicates that the student’s socio-economic family status had little or no effect on credit card debt use.

Table 1 presents the variable means for the whole sample and by the transcriptional efficiency grouping of the MAOA genotype. For the binary variables (credit card debt, gender, race dummies, and college) these means also represent column frequencies. The *p*-value is obtained from the Pearson chi-square test. For continuous variables (age and income) the *p*-value is the significance value of the *F*-test on a linear regression between these variables and the MAOA genotype.

4.2. Genetic association study

Genetic association studies test whether an allele or genotype occurs more frequently within a group exhibiting a particular trait than those without the trait.⁵ However, a significant association can be a false positive signal due to “population

⁴ For about 288 individuals of the MAOA discovery sample there is no newly sequenced MAOA genotypical data available as part of the newly released DNA sample. In the analyses reported in this paper we gave priority to the newly sequenced sample and hence why the full sample is slightly less large than the sum of the discovery and replication samples. Including these older observations does not alter the results reported in the paper.

⁵ A significant association means that a particular genotype—or one likely near it on the same gene that would be in “linkage disequilibrium”—significantly influences credit card debt borrowing behavior.

Table 1
Descriptive statistics (full sample, $N = 12,369$).

	Mean	MAOA		P-value
		High	Low	
Credit card debt (yes)	0.41	0.40	0.43	0.007
Race				
White	0.64	0.72	0.57	0.000
Black	0.23	0.16	0.29	0.000
Hispanic	0.16	0.16	0.15	0.089
Asian	0.07	0.05	0.08	0.000
Gender (male)	0.47	0.59	0.36	0.000
Age	21.9	21.8	22.0	0.123
Income	14,055	14,399	13,015	0.515
College (yes)	0.54	0.53	0.55	0.065

Note: Presented are the variable means for the whole sample and by the transcriptional efficiency grouping of the MAOA genotype. For the binary variables (credit card debt, gender, race dummies, and college) these means also represent column frequencies. The P -value is obtained from the Pearson chi-square test. For continuous variables (age and income) the P -value is the significance value of a linear regression between these variables and the MAOA genotype (controlling for age, gender, and race).

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 local adaptation or genetic drift these groups may develop different frequencies of a particular genotype. At the same time, the two groups may also develop divergent behaviors that are not influenced by the genotype but completely by the environment in which they live. Once these two groups mix in a larger population, simply comparing the frequency of the genotype 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 genotypes among subjects that exhibit a trait of interest to subjects who do not. As a result, case-control designs are vulnerable to population stratification if either group is especially prone to selection effects. A typical way to deal with this problem is to include controls for the race or ethnicity of the subject or to limit the analysis to a specific racial or ethnic group. This is the approach used in this paper. 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 whether offspring exhibiting the trait receive a risk allele from their parents more often than would be expected by chance. The family-based design is very powerful in minimizing type I error (false positive) but suffers from much lower power in detecting a true association and is thus prone to type II error (false negative).⁶ 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, $G = 1$ if the subject's genotype is L/L, L/H or H/L, and $G = 0$ if the subject's genotype is H/H (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 sample as well as potentially mediating factors such as age, gender, income, parental income and education that may influence financial decision-making. Finally, the variable U is a family random effect that controls for potential genetic and environmental correlation among family members. The coefficient β_G tests the association between the MAOA genotype 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, having a low efficiency allele of the MAOA gene does 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.

⁶ The availability of family clustered data in Add Health is limited to the smaller discovery sample. The results of family-based analyses are presented in Appendix.

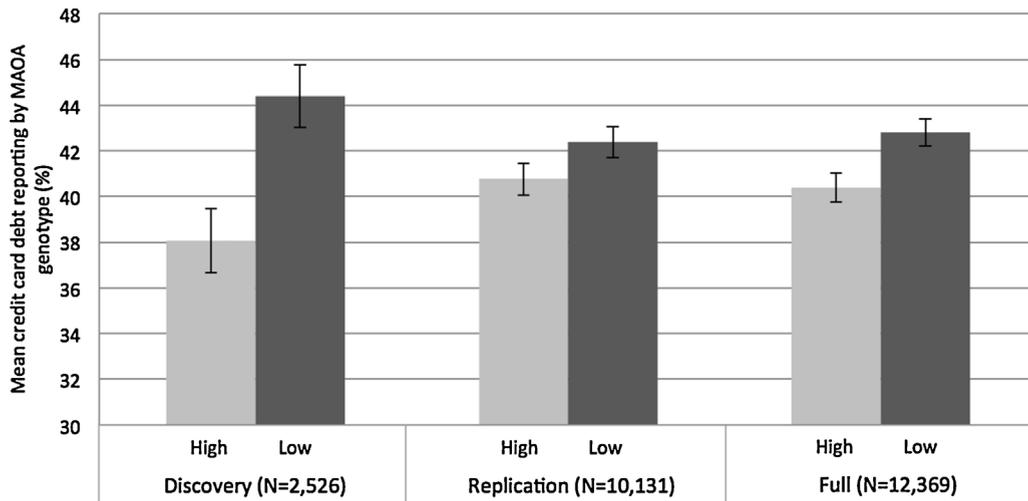


Fig. 1. Credit card debt by MAOA genotype for discovery, replication, and combined samples. Mean credit card debt reporting percentages are presented along with 95% confidence intervals.

5. Results

Prior research has linked the low efficiency alleles of the MAOA gene to impulsivity and reduced conscientiousness via its impact on the serotonin metabolism and other neurological processes. As such, we hypothesized that its carriers would be more susceptible to high-cost borrowing, as exemplified by credit card debt, and this intuition is verified in the data. In Figure 1 we summarize the descriptive results for mean credit card debt reporting by MAOA genotype in the discovery, replication, and combined samples. To our knowledge, this is the first specific genotype to be associated with real-world borrowing behavior.

In Table 2 we develop these descriptive statistics and show the results of three model specifications for each sample. Each of these specifications includes variables for age, gender, and race. It is also possible that socio-economic factors mediate the

Table 2
Models of association between MAOA and credit card debt (discovery, replication, and full samples).

	Discovery sample			Replication sample			Full sample		
	Model 1 OR	Model 2 OR	Model 3 OR	Model 1 OR	Model 2 OR	Model 3 OR	Model 1 OR	Model 2 OR	Model 3 OR
MAOA low	1.24** (0.11)	1.31** (0.15)	1.48*** (0.20)	1.04 (0.04)	1.12** (0.06)	1.17** (0.08)	1.07* (0.04)	1.15*** (0.06)	1.23*** (0.08)
Black	0.81* (0.10)	0.85 (0.14)		0.85*** (0.05)	0.91 (0.07)		0.84*** (0.04)	0.90 (0.06)	
Hispanic	0.86 (0.386)	0.80 (0.17)		1.20** (0.05)	1.23* (0.14)		1.15* (0.09)	1.14 (0.12)	
Asian	1.01 (0.19)	1.04 (0.27)		0.96 (0.08)	0.88 (0.11)		0.98 (0.08)	0.94 (0.11)	
Age	1.25*** (0.03)	1.22*** (0.04)	1.28*** (0.06)	1.21*** (0.01)	1.20*** (0.02)	1.23*** (0.02)	1.21*** (0.01)	1.20*** (0.02)	1.24*** (0.02)
Male	0.76*** (0.07)	0.74*** (0.08)	0.86 (0.12)	0.74*** (0.03)	0.74*** (0.04)	0.74*** (0.05)	0.74*** (0.03)	0.73*** (0.04)	0.77*** (0.05)
Income (log)		1.06*** (0.02)	1.06** (0.03)		1.06*** (0.01)	1.06*** (0.01)		1.05*** (0.01)	1.06*** (0.01)
Parental income (log)		1.03 (0.05)	0.98 (0.06)		1.01 (0.01)	0.99 (0.03)		1.01 (0.02)	0.99 (0.03)
College		1.64*** (0.19)	1.50*** (0.22)		1.63*** (0.09)	1.27*** (0.09)		1.62*** (0.08)	1.29*** (0.08)
Intercept	0.01*** (0.58)	0.01*** (0.67)	0.00*** (0.87)	0.02*** (0.26)	0.01*** (0.40)	0.00*** (0.53)	0.01*** (0.24)	0.01*** (0.37)	0.00*** (0.48)
N	2,523	1,628	1,001	10,113	6,213	3,589	12,349	7,676	4,504
Pseudo R ²	0.031	0.048	0.045	0.023	0.038	0.035	0.024	0.039	0.036

Note: Variable definitions are in the appendix. Model 3 presents association results for those subjects that uniquely identified themselves as being white. All results are expressed in odds ratios (OR). Standard errors in parentheses.

* Significant at 10%.
 ** Significant at 5%.
 *** Significant at 1%.

relationship between the gene we have identified and credit card usage. For example, we might expect genes to contribute to variation in socio-economic factors such as income (Bowles and Gintis, 2002), which in turn would impact financial decision-making. Also, several twin studies have suggested that variation 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 also a factor that has been found to be heritable (Baker et al., 1996; Heath et al., 1985; Rietveld et al., 2013) and is frequently shown to influence household finance. *Model 2* therefore includes income, parental income, and education as a basic set of socio-economic controls. If these variables were acting as mediators, then including them would weaken the association between MAOA and debt. We also regress each of these variables separately on MAOA low along with age, gender, and race controls in *Table A.4* in the Appendix. Since MAOA low is not significantly associated with any of these variables, we can further rule them out as mediators.

Across discovery, replication, and full samples, *Model 1* and *Model 2* show that having one or both MAOA low alleles is associated with increased credit card debt; controlling for gender, age, ethnicity (*Model 1*), as well as a basic set of socio-economic variables (*Model 2*). At the exception of the basic model in the replication sample, all specifications obtain statistical significance. To illustrate the strength of the relationship, *Model 2* suggests that the odds of a person reporting credit card debt are increased by a factor 1.15 (full sample) if moved from the high efficiency ($G = 0$) to the low efficiency allele grouping ($G = 1$) of the MAOA genotype. Identical analyses that incorporate the other available Add Health candidate genotype markers are given in *Table A.3* in Appendix. Only the MAOA genotype produces a significant coefficient on credit card borrowing behavior. Given the demographic limitation of the data in terms of age, a robustness check was carried out to test for whether debt is possibly due to investment in education rather than consumption. Introducing a measure for educational debt does not meaningfully alter the statistical significance or size on any of the odds ratios of the MAOA Low alleles.

Following Xu and Shete (2006), as a robustness test for population stratification, we also include association models for those subjects that uniquely identified themselves as being white in *Model 3* (for each sample). The MAOA genotype is robust to this specification across discovery, replication, and full samples. However, when considering the subsample of subjects that identified themselves as black or African-American no significance is obtained on the MAOA genotype (presented in *Table A.5* in Appendix). Furthermore, taking advantage of the available sibling clusters (available only for the discovery sample) and performing a family-based test through a variance-components based association analysis for sibling pairs (first suggested by Boehnke and Langefeld (1998) and Spielman and Ewens (1998)) also does not obtain significance. The results of the family-based test and a more detailed discussion on its methodology are given in *Table A.6* in Appendix. These insignificant results are obtained on samples that are substantially smaller, with less statistical power, than the samples on which the significant results are obtained but it remains that this research is inconclusive on the role of population stratification in the relationship between MAOA and credit card debt borrowing. When simulating the marginal effect from the coefficient covariance matrix of *Model 1* in *Table 2* (full sample) we find that varying the MAOA genotype of all subjects from high to low would increase the reporting of credit card debt in this population from 40.5% to 42.2% holding all other variables constant at their mean. This implies that the marginal effect of having one or both MAOA alleles of the low efficiency type raises the average likelihood of having credit card debt by about 4%.

6. Discussion

The staggered release of genotypical data by Add Health provides a rare opportunity to test the association between the MAOA genotype and the reporting of credit card debt in an independent replication sample as well as the larger combined sample. Replication efforts and increasing sample size are important ways to address the possibility that the original association would be a spurious result or false positive (Beauchamp et al., 2011; Benjamin et al., 2012). As shown in *Fig. 1* and *Table 2*, we observe mostly significant and large coefficients across the discovery, replication, and full samples. Generally, these results—combined with the relatively large sample sizes—lead us to believe that the original association result in the discovery sample is not spurious and that the MAOA gene is associated with the reporting of credit card debt. Prior research has linked the low efficiency alleles of the MAOA gene to impulsivity and reduced conscientiousness via its impact on the serotonin metabolism and other neurological processes. As such, we hypothesized that its carriers would be more susceptible to high-cost borrowing, as exemplified by credit card debt, and this intuition is verified in the data. To our knowledge, this is the first large-scale study to show a specific genotype is associated with real-world borrowing behavior.

It is important to once more stress the limitations of our design and results with regards to population stratification. It remains unclear whether or not population stratification is driving the results on the relationship between MAOA and credit card borrowing. The relationship was robust to introducing race controls and limiting the analyses to the whites-only subsample. However, no significant relationship was obtained when considering the African-American subsample nor when using the available sibling clusters. These insignificant results are obtained on samples that are substantially smaller, and with less statistical power, than the samples on which the significant results are obtained but it remains that this research is inconclusive on the role of population stratification. Another limitation of our study is that the add Health data covers the 18–26 age group, although Jassim and Taylor (2010) suggest that credit card debt levels at those ages are representative of the general public.

If genetic variation influences credit card borrowing behavior, then there are likely to be important theoretical and welfare implications. The theoretical contribution of the MAOA finding lies in providing new explanatory power for 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., 2007). For example, Meier and Sprenger (2010) present the results of a large field study that shows that present-biased time preferences correlate with credit card borrowing and 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, 2010). In particular, this MAOA finding may build on 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, since genes are upstream from the neurological processes that McClure et al. (2004) identified.

Economists should especially consider the welfare consequences of allowing credit companies to discriminate on the basis of a person's genotype, and more broadly, the welfare consequences of any kind of genetic discrimination. On 21 May 2008 the Genetic Information Nondiscrimination Act 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. As the cost of genotyping plummets, millions of individuals will soon have their genomes sequenced, so we hope our results will stimulate consumer education (Kim and DeVaney, 2001) and inform an appropriate policy framework to prevent *any* kind of genetic discrimination.

Finally, we offer a word of caution. While the MAOA gene may show significant association with credit card debt, it is important to emphasize that there is no single "debt gene." Instead, there is likely to be a set of genes whose expression, in combination with environmental factors, influences financial decision-making. Association studies like ours require further investigation 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 association we have identified.

Acknowledgments

We thank Dan Benjamin, Chris Dawes, Steve Pischke, Jaime Settle, Piero Stanig, Jeremy Tobacman and anonymous referees for their help and comments. Seminar participants at the Institute for Empirical Research in Economics (University of Zurich) provided helpful comments. The usual disclaimer applies. An earlier working paper version of this research circulated under the title "The MAOA Gene Predicts Credit Card Debt." This research uses data from Add Health, a program project directed by Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. No direct support was received from grant P01-HD31921 for this analysis. The authors declare that they have no relevant or material financial interests that relate to the research described in this paper.

Appendix A.

Variable definitions

MAOA Low is an indicator variable for whether the subject's genotype is L/L, L/H or H/L (where L represents having a copy of a 291 or 321 base-pair "low" allele and H represents having a copy of a 336, 351, or 381 base-pair "high" allele). 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]?" *Parental income* is asked of the parents in Wave I. It is the numeric answer to the question "About how much total income, before taxes did your family receive in 1994? Include your own income, the income of everyone else in your household, and income from welfare benefits, dividends, and all other sources." The natural logarithm of both income variables is used in all reported regression tables. *College* is a dummy 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?" *Educational debt* is a dummy variable taking the value 1 if the respondent answered the following question affirmatively: "Do you have any student loans or other educational loans that have not yet been paid?" For information regarding the other Add Health genes used in the Appendix please refer to the cited Add Health document (Add Health Biomarker Team, 2007).

See Tables A.1–A.5.

Family-based test

Estimating sibling fixed effects is one way of guarding against population stratification. The behavior genetics literature does not consider it to be a method for establishing causality. Both case-control and family-based methods test genetic association that would be consistent with a causal theory, but they do not establish a causal link.

We specify the family-based test through a variance-components based association analysis for sibling pairs (first suggested by Boehnke and Langefeld (1998) and Spielman and Ewens (1998)). By decomposing genotype scores into between-family (b) and within-family (w) components it is possible to control for spurious results due to population

Table A.1

Distribution and frequency of MAOA variable-number tandem repeat (VNTR) alleles across discovery sample population (each individual has two alleles).

MAOA genotype (VNTR)	Allele distribution	Frequency (%)
291 bp (low)	59	1.1
321 bp (low)	2016	39.7
336 bp (high)	45	0.9
351 bp (high)	2897	57.1
381 bp (high)	61	1.2
Total	5078	100

Table A.2

Association models between MAOA genotype (VNTR) variations and credit card debt with controls for gender, age, and race (discovery sample).

	Model 1		Model 2		Model 3		Model 4		Model 5	
	OR	P-value								
MAOA 291 bp	0.94	0.784								
MAOA 321 bp			1.14	0.014						
MAOA 336 bp					1.43	0.186				
MAOA 351 bp							0.87	0.007		
MAOA 381 bp									1.04	0.849
Male	0.74	0.001	0.74	0.014	0.74	0.001	0.83	0.14	0.74	0.001
Age	1.25	0.000	1.25	0.000	1.25	0.000	1.25	0.000	1.25	0.000
Black	0.91	0.462	0.87	0.289	0.90	0.433	0.86	0.248	0.90	0.426
Hispanic	0.89	0.762	0.90	0.789	0.88	0.730	0.90	0.788	0.89	0.765
Asian	1.01	0.944	0.96	0.819	1.02	0.915	0.96	0.831	1.01	0.938
N		2528		2528		2528		2528		2528
Pseudo R ²		0.028		0.030		0.029		0.031		0.028

Note: All results are expressed in odds ratios (OR) and P-values are presented.

Table A.3

Association model between available Add Health genotypes and credit card debt with controls for gender, age, and race.

	OR	P-value
MAOA: low	1.25	0.021
DRD4: r4	0.97	0.674
DRD2: a1	0.87	0.063
DAT1: r10	1.08	0.295
CHRNA6: rs892413	1.11	0.265
CHRN3: rs13280604	0.92	0.362
CYP2A6B: inactive	0.75	0.200
5-HTTLPR: short	1.05	0.436
Male	0.78	0.010
Age	1.23	0.002
Black	0.89	0.533
Hispanic	1.16	0.743
Asian	0.89	0.658
Intercept	0.01	0.000
N		2132
Pseudo R ²		0.030

Note: The full set of Add Health candidate genotype markers is only available for the discovery sample. All results are expressed in odds ratios (OR) and P-values are presented.

Table A.4

Tests for potential mediator variables.

DV	MAOA low p-value
Income	0.51
College	0.59
Married	0.09
Divorced	0.73
Religious	0.27
Educational debt	0.49
Parental income	0.82

Note: Presented are 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.

Table A.5

Models of association between MAOA and credit card debt for those subjects that identified themselves as black or African-American (discovery, replication, and full samples).

	Discovery sample OR	Replication sample OR	Full sample OR
MAOA low	1.13 (0.37)	1.04 (0.16)	1.04 (0.14)
Age	1.16 (0.11)	1.10** (0.04)	1.12*** (0.04)
Male	0.50** (0.16)	0.50*** (0.07)	0.50*** (0.06)
Income (log)	1.10 (0.06)	1.11*** (0.03)	1.11*** (0.02)
Parental income (log)	1.50** (0.29)	1.17** (0.08)	1.20*** (0.08)
College	2.61*** (0.86)	2.75*** (0.36)	2.67*** (0.33)
N	245	1122	1338
Pseudo R ²	0.128	0.094	0.097

Note: Variable definitions are in the appendix. All results are expressed in odds ratios (OR). Standard errors in parentheses.

* Significant at 10%.

** Significant at 5%.

*** Significant at 1%.

Table A.6

Family-based association model between MAOA low and credit card debt.

	OR	P-value
MAOA low: within-family variance (G_w)	0.92	0.599
MAOA low: between-family variance (G_b)	0.15	0.374
N	483	
Pseudo R ²	0.002	

Note: Sibling clusters are only available for the discovery sample. All results are expressed in odds ratios (OR) and P-values are presented.

stratification because only the coefficient on the between-family variance (β_b) will be affected. The association result is determined by the coefficient on the within-family variance (β_w) which, in essence, shows whether the allele frequencies among siblings with the phenotype differ significantly from the frequencies among their siblings without the phenotype. In the behavior genetics literature this approach is called a “sib TDT” (sibling transmission/disequilibrium) test. Given the binary nature of our dependent variable we employ a logistic regression model to perform this family-based association test:

$$P[Y_{ij} = 1 | Z_{kij}, U_j] = \text{logit}(\beta_0 + \beta_w G_{wij} + \beta_b G_{bj} + U_j + \epsilon_{ij})$$

where i and j index subject and family respectively. G_w is the within-family variance component of the individual’s MAOA genotype, G_b is the between-family variance component of the individual’s genotype, U is a family random effect that controls for potential genetic and environmental correlation among family members, and ϵ is an individual-specific error.

Table A.6.

The result on G_w is not significant. Please note that the sample size is relatively small and that many observations in the family based test are lost because it only includes individuals who have a different genotype than their sibling. As a result, this method suffers from much lower power in detecting a true association and is thus prone to false negatives. [Xu and Shete \(2006\)](#) show, based on extensive simulation work, that a case-control association study (with race controls) using a mixed-effects analysis outperforms family-based designs in detecting an association while at the same time effectively limiting type I error. This is the method that we have followed throughout this work, and all regressions account for repeated observations within families by clustering standard errors on family identification number (“famid”). Please note that the newly released genetic data (replication sample) does not allow for running a family fixed-effects estimation because the original genetic sample was selected precisely on the basis of comprising all the Add Health sibling clusters. Hence, none of the individuals in the replication sample are in a family relationship.

References

Add Health Biomarker Team, 2007. Biomarkers in Wave III of the Add Health Study. <http://www.cpc.unc.edu/projects/addhealth/files/biomark.pdf>
 Agarwal, S., Driscoll, J., Gabaix, X., Laibson, D.I., 2008. Learning in the credit card market. National Bureau of Economic Research Working Paper (13822).
 Agarwal, S., Skiba, P.M., Tobacman, J., 2009. Payday loans and credit cards: New liquidity and credit scoring puzzles. *Am. Econ. Rev.* 99 (2), 412–418.
 Alcott, H., Karlan, D., Mobius, M., Rosenblat, T., Szeidl, A., 2007. Community size and network closure. *Am. Econ. Rev.* 97 (2), 80–85.
 Alford, J., Funk, C., Hibbing, J., 2005. Are political orientations genetically transmitted? *Am. Polit. Sci. Rev.* 99 (2), 153–167.

- Baker, L., Treloar, S., Reynolds, C., Heath, A., Martin, N., 1996. Genetics of educational attainment in Australian twins: sex differences and secular changes. *Behav. Genet.* 26 (2), 89–102.
- Balciuniene, J., Jazin, E., 2001. Human monoamine oxidase: from genetic variation to complex human phenotypes. *Gene Funct. Dis.* 2 (1), 26–37.
- Beauchamp, J., Cesarini, D., Johannesson, M., van der Loos, M., Koellinger, P., Groenen, P., Fowler, J., Rosenquist, J., Thurik, A., Christakis, N., 2011. Molecular genetics and economics. *J. Econ. Perspect.* 25 (4), 57–82.
- Benjamin, D.J., Cesarini, D., Chabris, C.F., Glaeser, E.L., Laibson, D.I., Gunason, V., Harris, T.B., Launer, L.J., Purcell, S., Smith, A.V., Johannesson, M., Magnusson, P.K., Beauchamp, J.P., Christakis, N.A., Atwood, C.S., Hebert, B., Freese, J., Hauser, R.M., Hauser, T.S., Grankvist, A., Hultman, C.M., Lichtenstein, P., 2012. The promises and pitfalls of geneoconomics. *Ann. Rev. Econ.* 4 (September).
- Benjamin, D.J., Chabris, C.F., Glaeser, E.L., Gudnason, V., Harris, T.B., Laibson, D.I., Launer, L., Purcell, S., 2007. Geneoconomics. In: Weinstein, M., Vaupel, J.W., Wachter, K.W. (Eds.), *Biosocial Surveys*. The National Academies Press, Washington, DC.
- Boehnke, M., Langefeld, C., 1998. Genetic association mapping based on discordant sib pairs: the discordant-alleles test. *Am. J. Hum. Genet.* 62, 950–961.
- Bowles, S., Gintis, H., 2002. The inheritance of inequality. *J. Econ. Perspect.* 16 (3), 3–30.
- Campbell, J., 2006. Household finance. *J. Finan.* 61 (4), 1553–1604.
- Can, A., Coe, B.P., Eichler, E.E., 2011. Genome structural variation discovery and genotyping. *Nat. Rev. Genet.* 12 (5), 363–376.
- Cases, O., Seif, I., Grimsby, J., Gaspar, P., Chen, K., 1995. Aggressive behaviour and altered amounts of brain serotonin and norepinephrine in mice lacking *maoa*. *Science* 268 (5218), 1763–1766.
- Cases, O., Vitalis, T., Seif, I., De Maeyer, E., Sotelo, C., Gaspar, P., 1996. Lack of barrels in the somatosensory cortex of monoamine oxidase a—deficient mice: role of a serotonin excess during the critical period. *Neuron* 16 (2), 297–307.
- Caspi, A., McClay, J., Moffitt, T., Mill, J., Martin, J., Craig, I., Taylor, A., Poulton, R., 2002, August. Role of genotype in the cycle of violence in maltreated children. *Science* 297 (5582), 851–854.
- Cesarini, D., Dawes, C.T., Fowler, J., Johannesson, M., Lichtenstein, P., Wallace, B., 2008. Heritability of cooperative behavior in the trust game. *Proc. Natl. Acad. Sci. U.S.A.* 105 (10), 3721.
- Cesarini, D., Dawes, C.T., Johannesson, M., Lichtenstein, P., Wallace, B., 2009. Genetic variation in preferences for giving and risk-taking. *Quart. J. Econ.* 124 (2), 809–842.
- Cesarini, D., Johannesson, M., Lichtenstein, P., Sandewall, Ö., Wallace, B., 2010. Genetic variation in financial decision making. *J. Fin.* 65 (5), 1725–1754.
- Contini, V., Marques, F., Garcia, C., Hutz, M., Bau, C., 2006. *Maoa*-*uvntr* polymorphism in a brazilian sample: further support for the association with impulsive behaviours and alcohol dependence. *Am. J. Med. Genet. B Neuropsychiat. Genet.* 141B (3), 305–308.
- Cools, R., Blackwell, A., Clark, L., Menzies, L., Cox, S., Robbins, T., 2005. Tryptophan depletion disrupts the motivational guidance of goal-directed behavior as a function of trait impulsivity. *Neuropsychopharmacology* 30, 1362–1373.
- Craig, I., 2007. The importance of stress and genetic variation in human aggression. *Bioessays* 29 (3), 227–236.
- Damberg, M., Garpenstrand, H., Hallman, J., Orelund, L., 2001. Genetic mechanisms of behavior: don't forget about the transcription factors. *Mol. Psychiat.* 6 (5), 503–510.
- De Neve, J.-E., 2011. Functional polymorphism (5-*httlpr*) in the serotonin transporter gene is associated with subjective well-being: Evidence from a u.s. nationally representative sample. *J. Hum. Genet.* 56 (6), 456–459.
- De Neve, J.-E., Christakis, N.A., Fowler, J.H., Frey, B.S., 2012. Genes, economics, and happiness. *J. Neurosci. Psychol. Econ.* 5 (4), 193–211.
- De Neve, J.-E., Mikhaylov, S., Dawes, C.T., Christakis, N.A., Fowler, J.H., 2013. Born to lead? A twin design and genetic association study of leadership role occupancy. *Leader. Quart.* 24 (1), 45–60.
- Denney, R., Koch, H., Craig, I., 1999. Association between monoamine oxidase a activity in human male skin fibroblasts and genotype of the MAOA promoter-associated variable number tandem repeat. *Hum. Genet.* 105 (6), 542–551.
- Denney, R., Sharma, A., Dave, S., Waguespack, A., 1994. A new look at the promoter of the human monoamine oxidase a gene: mapping transcription initiation sites and capacity to drive luciferase expression. *J. Neurochem.* 63 (3), 843–856.
- Domsche, K., Sheehan, K., Lowe, N., Kirley, A., Mullins, C., et al., 2005. Association analysis of the monoamine oxidase a and b genes with attention deficit hyperactivity disorder (ADHD) in an irish sample: preferential transmission of the *maoa*-a 941 g allele to affected children. *Am. J. Me. Genet.* 134, 110–114.
- Dreber, A., Apicella, C.L., Eisenberg, D.T., Garcia, J.R., Zamore, R.S., Lum, J.K., Campbell, B., 2009. The 7r polymorphism in the dopamine receptor d4 gene (*DRD4*) is associated with financial risk taking in men. *Evol. Hum. Behav.* 30, 85–92.
- Echenique, F., Fryer, R.G., 2007. A measure of segregation based on social interactions. *Quart. J. Econ.* 122 (2), 441–485.
- Eisenberger, N., Way, B., Taylor, S., Welch, W., Lieberman, M., 2007. Understanding genetic risk to aggression: clues from the brain's response to social exclusion. *Biol. Psychiat.* 61, 1100–1108.
- Fan, J., Fossella, J., Sommer, T., Wu, Y., Posner, M., 2003. Mapping the genetic variation of executive attention onto brain activity. *Proc. Natl. Acad. Sci. U.S.A.* 100, 7406–7411.
- Fehr, E., 2002. The economics of impatience. *Nature* 415, 269–272.
- Foley, D., Eaves, L., Wormley, B., Silberg, J., Maes, H., et al., 2004. Childhood adversity, monoamine oxidase a genotype, and risk for conduct disorder. *Arch. Gen. Psychiat.* 61, 738–744.
- Fowler, J.H., Baker, L.A., Dawes, C.T., 2008. Genetic variation in political participation. *Am. Polit. Sci. Rev.* 101 (2), 233–248.
- Fowler, J.H., Dawes, C.T., 2008. Two genes predict voter turnout. *J. Poli.* 70 (03), 579–594.
- Frazzetto, G., Di Lorenzo, G., Carola, V., Proietti, L., Sokolowska, E., Siracusano, A., Gross, C., Troisi, A., 2007. Early trauma and increased risk of physical aggression during adulthood: The moderating role of *maoa* genotype. *PLoS One* 2 (5).
- Frydman, C., Camerer, C., Bossaerts, P., Rangel, A., 2011. MAOA-l carriers are better at making optimal financial decisions under risk. *Proc. R. Soc. B* 278 (1714), 2053–2059.
- Good, C., Lawrence, K., Thomas, N., 2003. Dosage-sensitive X-linked locus influences the development of amygdala and orbitofrontal cortex, and fear recognition in humans. *Brain* 126, 2431–2446.
- Guo, G., Ou, X.-M., Roettger, M., Shih, J.C., 2008. The *vntr* 2-repeat in *maoa* and delinquent behavior in adolescence and young adulthood: associations and *maoa* promoter activity. *Eur. J. Hum. Genet.* 16 (5), 626–634.
- Guo, G., Zhao, H., 2000. Multilevel modeling for binary data. *Ann. Rev. Sociol.* 26 (1), 441–462.
- Haberstick, B., Lessem, J., Hopfer, C., Smolen, A., Ehringer, M., Timberlake, D., Hewitt, J., 2005. Monoamine oxidase a (*maoa*) and antisocial behaviors in the presence of childhood and adolescent maltreatment. *Am. J. Med. Genet. B* 135B, 59–64.
- Hariri, A., Drabant, E., Munoz, K., Kolachana, B., Mattay, V., Egan, M., Weinberger, D., 2005. A susceptibility gene for affective disorders and the response of the human amygdala. *Arch. Gen. Psychiat.* 62, 146–152.
- Harris, K., Halpern, C., Whitsel, E., Hussey, J., Tabor, J., Entzel, P., Udry, J., 2009. The National Longitudinal Study of Adolescent Health: Research Design. <http://www.cpc.unc.edu/projects/addhealth/design>
- Hatemi, P., Medland, S., Morley, K., Heath, A., Martin, N., 2007. The genetics of voting: An australian twin study. *Behav. Genet.* 37 (3), 435–448.
- Heath, A., Berg, K., Eaves, L., Solaas, M., Corey, L., Sundet, J., Magnus, P., Nance, W., 1985. Education policy and the heritability of educational attainment. *Nature* 314 (6013), 734–736.
- Heidhues, P., Koszegi, B., 2010. Exploiting naivete about self-control in the credit market. *Am. Econ. Rev.* 100 (5), 2279–2303.
- Jassim, A.A., Taylor, J.C., 2010. College students' credit card usage and debt. *Compet. Forum* 8 (1), 101–110.
- Kim, H., DeVaney, S.A., 2001. The determinants of outstanding balances among credit card revolvers. *Finan. Counsel. Plan.* 12 (1), 67–77.
- Kim-Cohen, J., Caspi, A., Taylor, A., Williams, B., Newcombe, R., et al., 2006. *Maoa*, maltreatment, and gene environment interaction predicting children's mental health: new evidence and a meta analysis. *Mol. Psychiat.* 11 (10), 903–913.

- Knafo, A., Israel, S., Darvasi, A., Bachner-Melman, R., Uzevovsky, F., Cohen, L., Feldman, E., Lerer, E., Laiba, E., Raz, Y., et al., 2008. Individual differences in allocation of funds in the dictator game associated with length of the arginine vasopressin 1 a receptor RS 3 promoter region and correlation between RS 3 length and hippocampal mRNA. *Genes, Brain and Behavior* 7 (3), 266–275.
- Kraemer, G., Ebert, M., Schmidt, D., McKinney, W., 1989. A longitudinal study of the effect of different social rearing conditions on cerebrospinal fluid norepinephrine and biogenic amine metabolites in rhesus monkeys. *Neuropsychopharmacology* 2 (3), 175–189.
- Kuhnen, C.M., Chiao, J.Y., 2009. Genetic determinants of financial risk taking. *PLoS One* 4 (2), 1–4.
- Kuhnen, C.M., Samanez-Larkin, G.R., Knutson, B., 2013. Serotonergic genotypes, neuroticism, and financial choices. *PLoS One* 8 (1), e54632.
- Laibson, D.I., 1997. Golden eggs and hyperbolic discounting. *Quart. J. Econ.* 112 (2), 443–477.
- Laibson, D.I., Repetto, A., Tobacman, J., 2007. Estimating Discount Functions With Consumption Choices Over the Lifecycle. NBER Working Paper Series, 13314.
- Lawson, D., Turic, D., Langley, K., Pay, H., Govan, C., et al., 2003. Association of monoamine oxidase a and attention deficit hyperactivity disorder. *Am. J. Med. Genet. B Neuropsychiat. Genet.* 116B (1), 84–89.
- Lusardi, A., Tufano, P., 2009. Debt Literacy, Financial Experiences and Overindebtedness. NBER Working Paper Series (No. 14808).
- Mackay, T., 2001. The genetic architecture of quantitative traits. *Ann. Rev. Genet.* 35, 303–339.
- McClure, S., Laibson, D.I., Loewenstein, G., Cohen, J., 2004. Separate neural systems value immediate and delayed monetary rewards. *Science* 306 (5695), 503–507.
- McDermott, R., Tingley, D., Cowden, J., Frazetto, G., Johnson, D.D., 2009. Monoamine oxidase a gene (maoa) predicts behavioral aggression following provocation. *Proc. Natl. Acad. Sci. U.S.A.* 106 (7), 2118–2123.
- McGue, M., Bouchard, T., 1998. Genetic and environmental influences on human behavioral differences. *Ann. Rev. Neurosci.* 21 (1), 1–24.
- Meier, S., Sprenger, C., 2010. Present-biased preferences and credit card borrowing. *Am. Econ. J. Appl. Econ.* 2 (1), 193–210.
- Mertins, V., Schote, A.B., Hoffeld, W., Griessmair, M., Meyer, J., 2011. Genetic susceptibility for individual cooperation preferences: The role of monoamine oxidase a gene (MAOA) in the voluntary provision of public goods. *PLoS One*, <http://dx.doi.org/10.1371/journal.pone.0020959>.
- Meyer-Lindenberg, A., Buckholtz, J., Kolachana, B., Hariri, A., Pezawas, L., Blasi, G., Wabnitz, A., Honea, R., Verchinski, B., Callicott, J., Egan, M., Mattay, V., Weinberger, D., 2006. Neural mechanisms of genetic risk for impulsivity and violence in humans. *Proc. Natl. Acad. Sci. U.S.A.* 103 (16), 6269–6274.
- Passamonti, L., Fera, F., Magariello, A., Cerasa, A., Gioia, M., Muglia, M., Nicoletti, G., Gallo, O., Provinciali, L., Quattrone, A., 2006. Mao-a genetic variations influence brain activity associated with inhibitory control: new insight into the neural correlates of impulsivity. *Biol. Psychiat.* 59 (4), 334–340.
- Plomin, R., DeFries, J.C., McClearn, G.E., McGuffin, P., 2008. *Behavioral Genetics*, 5th ed. Worth Publishers, New York, NY.
- Popova, N., Voitenko, N., Maslova, L., 1989. The effect of emotional stress and corticosterone on the activity of monoaminoxidase in the rat brain. *Voprosy Med. Khim.* 35 (4), 114–119.
- Redon, R., Ishikawa, S., Fitch, K.R., Feuk, L., Perry, G.H., Andrews, T.D., Fiegler, H., et al., 2006. Global variation in copy number in the human genome. *Nature* 444 (7118), 444–454.
- Rietveld, C.A., Cesarini, D., Benjamin, D., Koellinger, P., De Neve, J.-E., Johannesson, M., Magnusson, P., Tiemeier, H., Pedersen, N., Krueger, R., Bartels, M., 2013. Molecular genetics and subjective well-being. *Proc. Natl. Acad. Sci. U.S.A.* 110 (24), 9692–9697.
- Rietveld, C.A., Medland, S., Derringer, J., Yan, J., Esko, T., Martin, N.W., Westra, H.-J., Shakbazov, K., Abdellaoui, A., et al., 2013. Gwas of 126,559 individuals identifies genetic variants associated with educational attainment. *Science* 340 (6139), 1467–1471.
- Rosenberg, S., Templeton, A.R., Feigin, P.D., Lancet, D., Beckmann, J.S., Selig, S., Hamer, D.H., Skorecki, K., 2006. The association of dna sequence variation at the MAOA genetic locus with quantitative behavioural traits in normal males. *Hum. Genet.* 120 (4), 447–459.
- Rubia, K., Lee, F., Cleare, A., Tunstall, N., Fu, C., Brammer, M., McGuire, P., 2005. Tryptophan depletion reduces right inferior prefrontal activation during response inhibition in fast, event-related FMRI. *Psychopharmacology* 179 (4), 791–803.
- Sabol, S., Hu, S., Hamer, D., 1998. A functional polymorphism in the monoamine oxidase a gene promoter. *Hum. Genet.* 103 (3), 273–279.
- Saito, T., Lachman, H., Diaz, L., Hallikainen, T., Kauhanen, J., et al., 2002. Analysis of monoamine oxidase a (maoa) promoter polymorphism in finnish male alcoholics. *Psychiat. Res.* 109 (2), 113–119.
- Shamosh, N.A., DeYoung, C.G., Green, A.E., Reis, D.L., Johnson, M.R., Conway, A.R., Engle, R.W., Braver, T.S., Gray, J.R., 2008. Individual differences in delay discounting. *Psychol. Sci.* 19 (9), 904–911.
- Spielman, R., Ewens, W., 1998. A sibship test for linkage in the presence of association: the sib transmission/disequilibrium test. *Am. J. Hum. Genet.* 62, 450–458.
- Sprenger, C., Stavins, J., 2008. Credit card debt and payment use. Federal Reserve Bank of Boston Working Paper (08-2).
- Tobacman, J., 2009. Endogenous effective discounting, credit constraints, and wealth inequality. *Am. Econ. Rev.* 99 (2), 369–373.
- Tufano, P., De Neve, J.-E., Maynard, N., 2011. U.S. consumer demand for prize-linked savings: New evidence on a new product. *Econ. Lett.* 111 (2), 116–118.
- Virkkunen, M., Goldman, D., Nielsen, D., Linnoila, M., 1995. Low brain serotonin turnover rate (low csf 5h-iaa) and impulsive violence. *J. Psychiat. Neurosci.* 20 (4), 271–275.
- Walderhaug, E., Lunde, H., Nordvik, J., Landro, N., Refsum, H., Magnusson, A., 2002. Lowering of serotonin by rapid tryptophan depletion increases impulsiveness in normal individuals. *Psychopharmacology* 164 (4), 385–391.
- Weiss, A., Bates, T., Luciano, M., 2008. Happiness is a personal(ity) thing. *Psychol. Sci.* 19 (3), 205–210.
- Xu, H., Shete, S., 2006. Mixed-effects logistic approach for association following linkage scan for complex disorders. *Ann. Hum. Genet.* 71 (2), 230–237.
- Zhong, S., Israel, S., Xue, H., Ebstein, R.P., Chew, S.H., 2009. Monoamine oxidase a gene (maoa) associated with attitude towards longshot risks. *PLoS One* 4 (12).
- Zhong, S., Israel, S., Xue, H., Sham, P., Ebstein, R.P., Chew, S., 2009. A neurochemical approach to valuation sensitivity over gains and losses. *Proc. R. Soc. B* 276 (1676), 4181–4188.
- Zinman, J., 2009. Debit or credit? *J. Bank. Finan.* 33 (2), 358–366.