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Kuhnen, Camelia M. and Chiao, Joan Y.

Northwestern University – Kellogg School of Management,
Northwestern University

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Genetic determinants of financial risk taking

Camelia M. Kuhnen¹

Joan Y. Chiao^{2,3}

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Abstract

Individuals vary in their willingness to take financial risks. Here we show that variants of two genes that regulate dopamine and serotonin neurotransmission and have been previously linked to emotional behavior, anxiety and addiction (5-HTTLPR and DRD4) are significant determinants of risk taking in investment decisions. These findings provide novel evidence of a genetic basis for financial choices.

¹Kellogg School of Management, Northwestern University, Evanston, IL

²Department of Psychology, Northwestern University, Evanston, IL; ³Northwestern University Interdepartmental Neuroscience Program, Evanston, IL;

Please address correspondence to: Camelia M. Kuhnen, Department of Finance, Kellogg School of Management, Northwestern University, 2001 Sheridan Rd. , Evanston, IL 60208, email: c-kuhnen@kellogg.northwestern.edu, phone: +1 847 467 1841 or to Joan Y. Chiao, Department of Psychology, Interdepartmental Neuroscience Program, Northwestern University, 2029 Sheridan Rd., Evanston, IL 60208, email: jchiao@northwestern.edu, phone: +1 847 467 0481.

Risk preferences describe individuals' willingness to take or avoid risk in a variety of settings, including financial choice, and are an essential component of any model of economic behavior. Individuals vary in the extent to which they are willing to take financial risks, which may be explained in part by individual differences in heritable traits. Classical twin design studies estimate that genetic effects account for 20% variation in risk taking in experimental lottery choices (1) and between 35-54% of the liability for developing symptoms of pathological gambling (2). However, identification of specific genes underlying financial risk preferences has remained elusive.

Recent findings in neuroeconomics indicate that brain regions containing a high density of dopamine and serotonin neurotransmitters play an important role in financial decision-making. In particular, activity within the anterior insula and the nucleus accumbens, brain regions innervated by serotonergic and dopaminergic neural pathways, has been shown to relate to individuals' financial risk taking behavior (3). Given their role in regulating serotonergic and dopaminergic neurotransmission and prior association with anxiety (4) and novelty seeking (5), the serotonin transporter polymorphism (5-HTTLPR) and dopamine D4 receptor (DRD4) exon III polymorphism may contribute to the genetic basis of financial risk-taking behavior. The 5-HTTLPR consists of a 44-base pair insertion or deletion, generating either a long (l) or a short (s) allele. The short variant of the polymorphism reduces the transcriptional efficiency of the 5-HTT gene promoter and is associated with higher scores on neuroticism and harm avoidance (4). The dopamine D4 receptor (DRD4) exon III polymorphism has been linked to novelty seeking and pathological gambling. Individuals with the 7-repeat allele have higher

novelty seeking scores than those with other DRD4 variants (5) and are more likely to be pathological gamblers (6).

Here we investigated whether or not genetic variations in these two candidate functional polymorphisms, 5-HTTLPR and DRD4, contribute to individual differences in financial risk taking preferences. Based on prior behavioral genetics work, we hypothesized that individuals carrying two copies of the s allele of the 5-HTTLPR would be significantly more risk averse relative to individuals carrying one or two copies of the l allele. Additionally, we hypothesized that individuals with a 7-repeat allele of the DRD4 polymorphism would be significantly more risk seeking relative to those individuals without the 7-repeat allele. We elicited financial risk preferences in an experimental setting where participants made multiple investment decisions allocating funds between a risky and a riskless asset, and were compensated based on the performance of their chosen financial portfolio (Fig. 1A). We subsequently genotyped participants for 5-HTTLPR and DRD4 functional polymorphisms (see Supplement).

We found that individuals carrying two copies of the short allele of the 5-HTTLPR are significantly more risk averse relative to individuals carrying one or two copies of the long allele. Additionally, individuals with the 7-repeat allele of DRD4 are significantly more risk seeking relative to those individuals without the 7-repeat allele. These findings provide novel evidence of a genetic basis for financial choices.

Experimental Methods

65 subjects (26 male; M age = 22.4 yrs; SD age: 4.9 yrs) completed the investment task and were subsequently genotyped for the 5-HTTLPR and DRD4 functional polymorphisms. Participants gave informed consent prior to participating and

the study was approved by the IRB committee at Northwestern University. The entire experiment took 1.5 hours to complete and the average pay per subject was \$25. The entire sample consisted of 21 carriers homozygous for the s allele and 44 carriers with one or two copies of the l allele of the 5-HTTLPR polymorphism, and 15 carriers of the 7-repeat allele and 50 non-carriers of 7-repeat allele variant of DRD4.

Participants first completed the investment task and were then genotyped. On each of the 96 trials of the task subjects were given an amount of money \$T. Subjects could invest \$T + \$15 (the show-up fee) in two assets, a riskless and a risky one. The amount not invested in the risky asset was automatically invested in the riskless asset (shorting and borrowing were not allowed). In one version of the task, subjects were informed that the risky asset would pay either of two possible returns with equal probability, and these two possible outcomes for the return were known by the subject in each trial. In another version of the task, subjects were provided with the expected return and standard deviation of the risky asset. These two ways of presenting information about the payoffs of the risky investment are equivalent if subjects have mean-variance preferences (i.e. they like higher expected returns and lower variance), a common assumption in the finance literature which is also supported by our data. The riskless asset paid a known rate of return. Subjects' choices did not differ across these versions of the task, and therefore we combine the data from the two versions.

At the time of making a choice, subjects knew the actual rate of return of the risk-free asset and the two possible outcomes of the risky security, or, equivalently, the expected value and standard deviation of the risky return. The actual rate of return for the risky asset on any trial was not revealed until the end of the experiment. At the end of

the experiment, each subject selected a random number between 1 and 96 by picking a ball from an urn. That number determined the trial for which the subject would receive payment. If on any trial a subject chose to invest in the risky asset an amount larger than the maximum investment allowed ($\$T + \15), or if they did not respond, that trial was marked as invalid. If an invalid trial was selected from the urn, the final payment was only the show-up fee of \$15. Subjects therefore had incentives to always enter their choice for the risky investment, and to treat each of the 96 trials as the one that would determine their pay. By deferring information about earnings until the end of the experiment we eliminate wealth effects that may change subjects' choices depending on past outcomes.

In each trial subjects had six seconds to learn the information about the return distribution of the risky security and the return of the safe asset. They had six additional seconds to enter the dollar amount they wished to invest in the risky asset, which was an integer that could range from zero to the maximum investment of $\$T + \15 . A 2-second fixation screen indicated a new trial was about to begin.

Results

The amount of money participants invested in the risky security (M risky allocation: \$9.78; SD risky allocation: \$7.16) on each trial depended on the characteristics of the two investment choices, as would be predicted by standard models of economic choice where individuals have mean-variance preferences (7). Our benchmark model of investment decisions (see Table 1) indicates that all else equal, participants invested significantly more money in the risky asset if its expected return was higher, the standard deviation of its return was lower, or if the return of the safe asset

was lower. Moreover, the higher the amount available to participants, the more money they invested in the risky asset.. For each portfolio allocation decision of our subjects we calculated the risky investment in excess of the amount predicted by the benchmark model (i.e. the residual term in the regression model in Table 1). This *excess risky investment* measures how risk seeking an individual is relative to the average person in the subject pool.

Results demonstrate that financial risk seeking is correlated with the 5-HTTLPR and DRD4 functional polymorphisms. As shown in Fig. 1B, individuals who carry two copies of the short allele of the 5-HTTLPR polymorphism invest \$2.69 (about 28% of the average risky allocation) less in the risky asset than those carrying one or two copies of the long allele of the genotype ($p < 0.02$), in excess of the benchmark model. Similarly, individuals who carry the 7-repeat allele in the DRD4 gene invest \$2.46 (about 25% of the average risky allocation) more in the risky asset than those lacking the 7-repeat allele (Fig. 1C, $p < 0.04$).

Our findings show for the first time that functional polymorphisms known to regulate serotonergic and dopaminergic activity in the brain are associated with individual differences in financial risk-seeking behavior. The current work compliments a growing body of work demonstrating the heritability of economic decision-making (1-2) and reveals specific genetic determinants of financial choices.

References

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Figure 1. (A) Trial structure of the investment task. For 6 seconds subjects observe the two possible and equally-likely values of the return of the risky asset, the return of the safe asset and the amount they have to invest that trial. When the word "Choice" appears on the screen, subjects have 6 seconds to enter the amount they wish to invest in the risky asset. Their remaining funds are automatically invested in the safe asset. A 2-second fixation screen precedes a new trial. (B) 5-HTTLPR and risk taking propensity. Individuals carrying one or two copies of the l allele demonstrated significantly greater risk taking relative to individuals carrying two copies of the s allele ($p < 0.02$). (C) DRD4 and risk taking propensity. Individuals carrying the 7-repeat allele demonstrated significantly greater risk taking relative to individuals without the 7-repeat allele ($p < 0.04$).

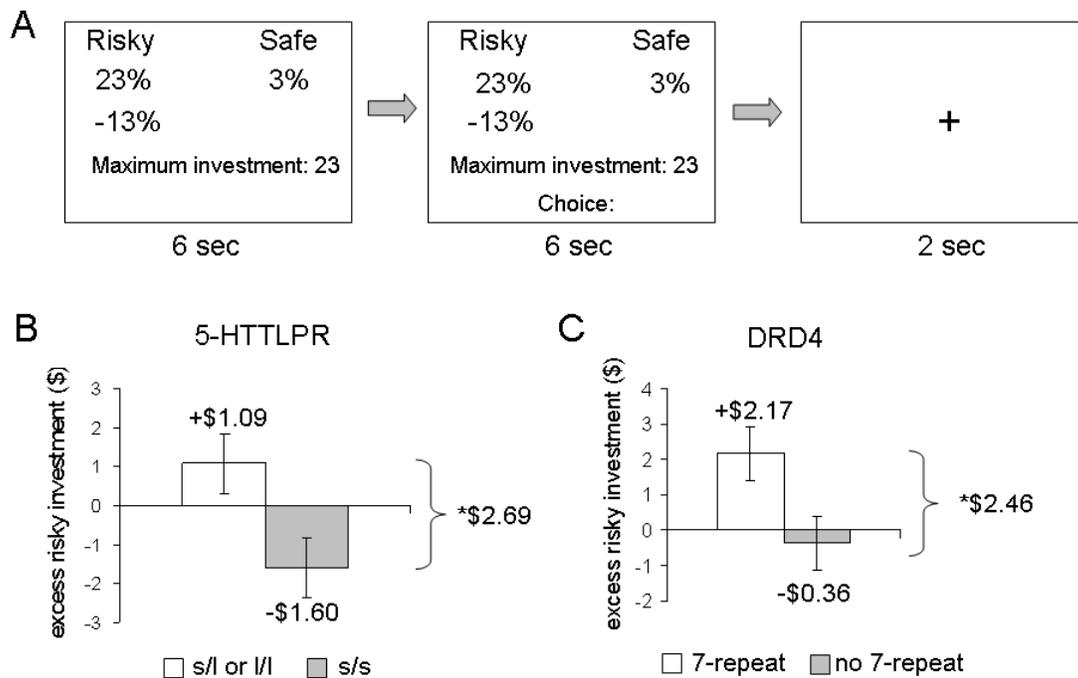


Table 1. Benchmark model of amount invested in risky asset. The dependent variable is the amount invested in the risky asset in each trial. Independent variables include the characteristics of the two investment options in a given trial, the amount of money available to the subject, as well as a task version indicator variable. Standard errors are robust to heteroscedasticity and correlation among error terms in observations belonging to the same subject. T-statistics are in parentheses.

Dependent Variable:	<i>Amount invested in risky asset</i>
	Coefficient/t-stat
<i>Risky Asset Expected Return</i>	42.89 (9.20) ^{***}
<i>Risky Asset Std. Dev. of Return</i>	-3.92 (-2.55) ^{**}
<i>Safe Asset Return</i>	-70.01 (-7.81) ^{***}
<i>Available funds</i>	0.39 (7.34) ^{***}
<i>Trial number</i>	-0.01 (-1.42)
<i>Constant</i>	-2.66 (-1.64)
Adj. R2	0.13
Observations	5987

** p < 0.05, *** p < 0.01

Supplementary Materials and Methods

Genotyping Procedure

Genomic DNA purification. Saliva was collected in the Oragene DNA collection kit (DNA Genotek). The DNA was extracted using the manufacturer's protocol. Briefly, samples were incubated at 50°C for 1 hr to inactivate enzymes. To each sample, Oragene DNA purifier (1/25 original volume) was added, incubated on ice for 10 min and centrifuged at 22,000xg for 10 min. The supernatant was removed and mixed with an equal volume of 100% ethanol by gentle inversion of the tubes. The sample incubated at room temperature for 10 min and was centrifuged at 22,000xg for 10 min. The pellet was washed twice with 70% ethanol, air-dried and resuspended in nuclease-free water. The quality and quantity of the extracted DNA was evaluated by ultraviolet spectroscopy.

DNA repeat element genotyping. The presence of repeat DNA elements in the human serotonin transporter (5HTT) and the dopamine-D4-receptor exon III cytosolic loop were determined by PCR amplification across these regions of genomic DNA and evaluating the size of the resulting DNA products by agarose gel electrophoresis. The PCR amplification primers for the 5HHT assay were 5'-ATGCCAGCACCTAACCCCTAATGT -3' (forward) and 5'-GGACCGCAAGGTGGGCGGGA-3' (reverse) (Steiger, H. et al, 2007), and those for the DRD4 assay were 5'- GCGACTACGTGGTCTACTCG -3' (forward) and 5'-AGGACCCTCATGGCCTTG -3' (reverse) (Lichter, JB et al, 1993). A PCR reaction (25 µl) containing 50 ng purified genomic DNA from each subject, 0.5 µM each of forward and reverse primers, 400 µM deoxyribonucleotides, PCR buffer, 1X Q-solution and Taq

polymerase (1.25 units, Qiagen) was performed in a 7900 thermocycler (Applied Biosystems, Inc.). The samples were subjected to 95°C/5 min, 35 cycles of 94°C/30 sec, 60°C/30 sec and 72°C/1 min, and 72°C/4 min. After amplification, the PCR products were separated on a 2% agarose gel that was subsequently stained with ethidium bromide and visualized under ultraviolet light. Included in each assay was water as a negative control. Between 15-20% of the test samples were re-analyzed for data verification.

Supplementary References and Notes

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