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# The Genetics of Economic Risk Preferences

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ABSTRACT

We examine the influence of genetics on economic risk preferences by administering a measure of these preferences to monozygotic (MZ) (i.e., identical) and dizygotic (DZ) (i.e., non-identical) twin pairs. Our analysis supports a dominant genetic effect and virtually no additive genetic effect on economic risk preferences, with the heritability of preferences estimated at 0.63. These findings suggest that over half of the variation in such preferences can be explained by genetic factors, with the remainder of the variance explained by environmental influences not shared among sibling twins. We discuss the implications of our findings for the study of individual differences in economic risk preferences. Copyright © 2009 John Wiley & Sons, Ltd.

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We live in an uncertain world where our choices always involve an element of risk. Indeed, Starmer (2000, p. 377) states that ''. . .theories of choice lie at the very heart of economics.'' Since the modern formulation of expected utility theory (EUT) by Neumann and Morgenstern (1944), EUT has largely dominated the study of economic risk and risky choice.[1] According to EUT, given a choice between multiple options, people will choose the option that maximizes their expected utility. There has, however, been a systematic documentation of evidence showing that people's behavior deviates from these normative models in predictive ways (for discussion see Lowenstein, Weber, Hsee, & Welch, 2001; Starmer, 2000).

In the domain of risky choice, EUT predicts that people should always accept gambles that increase their expected utility. However, prospect theory, an alternative to EUT, suggests that people do not evaluate

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[1]Utility theory has its roots in the St. Petersburg Paradox, posed by Nichols Bernoulli in 1728 and resolved by Daniel Bernouilli in 1738 with the introduction of the notion of expected utility maximization. See, e.g., Machina (1987), Schoemaker (1982), and Starmer (2000).

potential losses and gains in the deliberative rational manner of an expected utility maximizer. Instead, people are subject to various cognitive limitations that, for example, lead to the use of utility functions where people are risk averse when it comes to gains but risk seeking when it comes to losses (see Kahneman & Tversky, 1979). Hence, individuals will choose a certain $85 000 instead of a gamble with an 85% chance of winning $100 000 and a 15% chance of winning nothing, but will take a gamble with an 85% chance of losing $100 000 and a 15% chance of losing nothing rather than taking a certain loss of $85 000 (see Tversky, 1990).

Although more traditional theories of choice from the field of finance differ from prospect theory in important ways, the principle of risk aversion when it comes to evaluating investment alternatives lies at the core of risk-return models such as Markowitz's (1959) mean-variance portfolio theory as well as Kahneman and Tversky's (Kahneman & Tversky, 1979) prospect theory.[2] Consider a choice between options A and B that each has two outcomes, where one outcome is a zero return and the other is a positive return. In this scenario, if a person chooses a bet A that has a higher expected return (with a lower probability of winning) over bet B that has a lower expected return (with a higher probability of winning), then such a choice indicates that the person is willing to take on more risk compared to a person who chooses bet B over A. The risk preferences associated with the former choice (i.e., A) is one that embodies a higher degree of risk-taking behavior compared to the latter (i.e., B) under either the model of Markowitz or of Kahneman–Tversky.

Although a number of studies explore the consequences of risk preferences in determining behavior (e.g., Odean, 1998; Shefrin & Statman, 1985), there is much less research investigating the underlying causes of individual differences in such preferences than there is investigating situational factors that influence these preferences (for discussion, see Lopes, 1994). Since Edward O.Wilson's suggestion that science has paid insufficient attention to the role of biological factors in the production of human behavior, the ''nature vs. nurture'' debate has raged on in the social sciences (Wilson, 1975). In recent years, scholars have begun to examine the biological factors contributing to human behavior. In this paper, we examine how genetic factors might influence economic risk preferences. If a genetic basis for differences in economic risk preferences can be determined, this will provide support for the proposition that risk preferences are a dispositional construct.

The idea that economic risk preference might be dispositional has received some indirect support. For example, a study by Weber and Hsee (1998) showed that perceptions of risk and risk preferences might vary across cultures, suggesting that there might be factors that influence risk preferences in individuals as well as groups. Also, there is some direct evidence suggesting that dispositional factors may be associated with preferences towards risk. For example, Levin, Hart, Weller, and Harshman, (2007) recently found correlations ranging between .20 and .38 in risky decision making over a 3-year period. Additionally, loss aversion and other factors relevant to risky decision making have been shown to be related to various personality dimensions (see Cohen, Narayanan, Johnson, & Weber, 2006), such as surgency, shyness, and impulsivity (e.g., Levin & Hart, 2003; Levin et al., 2007).

Although these relationships provide preliminary evidence that risk preferences may be dispositional, there have yet to be any systematic examinations of the genetic basis of risk preferences, and economic risk preferences more specifically. In the current study, we examined the degree of genetic influence (i.e., heritability) on economic risk preferences by conducting a twin study and using three measures of such preferences.

Although many studies assessing heritability have investigated the genetic basis of very basic human traits such as pitch perception (see Drayna, Manichaikul, de Lange, Snieder, & Spector, 2001), the study of genetic factors that may affect human behavior in decision-making contexts is not without precedent. For example, personality researchers such as Loehlin, McCrae, Costa, and John (1998) have shown that around half of the

[2]We are not suggesting here that modern finance and prospect theory view risk aversion identically.



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variation across people along many of the Big Five personality dimensions may be attributed to genetic causes. Further, studies in organizational behavior have pointed to the genetic components of a variety of constructs, such as the finding that job satisfaction is heritable (e.g., Arvey, Bouchard, Segal, & Abraham, 1989). As in our study, most of these studies use a twin methodology to examine the genetic versus environmental influences on various human attributes.

Section ''Twin Models'' begins with a discussion of twin models, followed by a discussion of our survey methodology in Section ''Methodology''. Section ''Statistical Analysis and Results'' presents the statistical analysis and results of our measure of economic risk preferences. Section ''Discussion and Conclusion'' discusses these results and provides concluding remarks.

## TWIN MODELS

Twin studies are based on the logic that phenotypic variation in a population is due to either additive genetic effects ($A$), dominant genetic effects ($D$), shared environmental effects ($C$), or unshared environmental effects ($E$). With these variance components, heritability is defined as the ratio of genetic variation ($A^2 + D^2$) to total variation ($A^2 + D^2 + C^2 + E^2$). By sampling monozygotic (MZ) and dizygotic (DZ) twins that are raised in the same household as their respective twin, the variation in an observed variable can be decomposed into the $A$, $D$, $C$, and $E$ components. This decomposition is based on the following logic (see Plomin, DeFries, McClearn, & McGuffin, 2001).

First, any similarity across MZ and DZ twins in the covariation among an observed variable may be attributed to shared environmental factors ($C$) because such factors should equally influence both members of a twin pair (e.g., shared experiences such as parenting style, early socio-economic status, and the like). Second, if there is covariation among the observed variables that is larger for MZ twins than DZ twins, the difference may be attributed to genetic effects because MZ twins share all of their genetic structure and DZ twins share, on average, half of their genetic structure. Third, any lack of covariation among both types of twins may be attributed to unshared environmental factors ($E$), such as unique experiences with friends, romantic partners, higher education, and life circumstances more generally.

Additive genetic effects ($A$) are those that are additively passed down from parent to offspring, perhaps due to the complexity of the phenotype. An example is the case with individuals' heights, which vary continuously in the population and are an additive function of the many genes that code for leg length, back curvature, and the like. As noted by Galton (1886 P. 249) ''stature is not a simple element, but a sum of the accumulated lengths of thicknesses of more than a hundred bodily parts''. Because MZ twins share all of their genetic structure and DZ twins share half, an additive genetic effect is modeled as being twice as large for MZ twins compared to DZ twins—additive traits will account for 50% of the covariance in DZ twins compared to MZ twins. Thus, an additive genetic effect can account for larger amounts of covariation among MZ twins than DZ twins when the degree of covariation is no larger than twice the size for MZ twins when compared to DZ twins (see Neale & Cardon, 1992).

Alternatively, dominant genetic effects ($D$) are those that are passed down from parent to offspring in a dominant/recessive fashion, perhaps due to a more precise genetic structure than additive genetic effects—although a simple genetic structure is not a requirement for dominant/recessive traits to exist. For example, consider the trait of eye color where alleles code for either dark or light eyes. Since dark eyes are dominant and light eyes are recessive, two alleles coding for light eyes are required to produce light colored eyes; all other combinations produce dark colored eyes. Given having light or dark eyes is a simple, dominant/recessive trait, the covariation among MZ twins along eye color will always be very high. However, because offspring can only receive a single allele from each parent instead of an additive genetic mixture as is the case in more complex traits such as height, Plomin et al. (2001) note that the covariation among DZ twins along eye color will always be much lower than that found in MZ twins. Specifically, dominant/recessive traits will



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account for 25% of the covariance in DZ twins compared to MZ twins. Therefore, dominant genetic effects are present when covariation along an observed variable for MZ twins is more than twice as large as the covariation observed among DZ twins. In such a case, shared environmental effects are assumed to be null.

With this logic, the twin model decomposes variance into *A*, *D*, *C*, and *E* components as follows:

$$\mathrm{VAR} = A^2 + D^2 + C^2 + E^2 \tag{1}$$

$$\mathrm{COVAR(MZ)} = A^2 + D^2 + C^2 \tag{2}$$

$$\mathrm{COVAR(DZ)} = A^2 * .5 + D^2 * .25 + C^2 \tag{3}$$

$$H = (A^2 + D^2)/(A^2 + D^2 + C^2 + E^2) \tag{4}$$

where VAR is the variation of the variable of interest, COVAR(MZ) is the covariation among MZ twins, COVAR(DZ) is the covariation among DZ twins, and *H* is the heritability coefficient. With these equality constraints, any difference between MZ and DZ twins in covariance will be attributed to *A* or *D*, while similarity in covariance will be attributed to *C*.

To illustrate the logic of this model, a few examples are helpful. Consider a case where MZ and DZ twins are only slightly different in their degree of covariance, such that MZ twins have covariance of 1.0 and DZ twins have covariance of .80. In this case, the difference in covariance of .20 means that there is a genetic effect present. Because additive genetic effects (*A*) are half as large in DZ twins as in MZ twins, the amount of covariance in MZ twins attributable to genetic factors can be fully captured by *A* such that $A^2 = .40$. By removing .40 from the MZ covariance of 1.0, and by removing .20 from DZ covariance of .80 (because in DZ twins $A^2$ is reduced by half), the remaining covariance is .60 for *both* MZ and DZ twins. With this similarity in covariance for MZ and DZ twins, the *C* factor will account fully for the remaining covariance (because, as noted above, shared environment creates similar amounts of covariance among twins). In this case, Heritability will be .40/1.0, or 40% (assuming that $E = 0$).

Next, consider a model where there is a larger difference in covariance, such that MZ twins have a covariance of 1.0 and DZ twins have a covariance of .50. In this case the difference in covariance is .50, again indicating a genetic effect is present. With the same logic as above, the A factor will fully capture the genetic effect, such that $A^2 = 1.0$. By removing 1.0 from the MZ covariance of 1.0 and by removing .50 from the DZ covariance of .50, there is no remaining covariance to attribute to C. In this case, Heritability will be 1.0/1.0, or 100% (again, assuming that $E = 0$). It is notable that this example shows how with *at least* twice as much covariance in MZ twins as in DZ twins, there is no additional covariance to attribute to *C* (i.e., shared-environmental effects will be null; because MZ twins are so much more similar than DZ twins, all covariation is attributed to a genetic effect).

Finally, consider a model where there is a substantial difference in covariance, such that MZ twins have a covariance of 1.0 and DZ twins have a covariance of .25. In this case the difference in covariance is .75, indicating a genetic effect is present. In this case, the difference between the two covariances is so large (i.e., the genetic effect is more than twice as large in MZ twins than DZ twins) that the A factor cannot account for the magnitude of this difference. In this case, the *D* factor will fully account for the covariance in MZ and DZ twins because the DZ covariance is one-quarter the magnitude of the MZ covariance. In this case $D^2 = 1.0$, so by removing 1.0 from the MZ covariance of 1.0 and by removing .25 from the DZ covariance of .25 there is no additional covariance to attribute to *A* or *C*.[3]

[3]As noted earlier, dominant/recessive traits account for 25% of the covariance in DZ twins compared to MZ twins. Hence, $D^2$ is reduced by three-fourths in DZ twins (Plomin et al., 2001).



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## METHODOLOGY

### Participants

Our sample was drawn from the Minnesota Twin Registry as a part of the Minnesota Parenting Project, which holds age, sex, and birth cohort constant by sampling male twins raised together and born between 1961 and 1964. Protocol for the Minnesota Twin Registry attempts to add additional control to data collection by indicating that surveys should be filled out independently by all respondents (i.e., without collusion among twin pairs). In 1999, surveys were sent to 558 male twin pairs (1116 men); 646 surveys were returned for a response rate of 57.9%. Of these, there were 631 that contained responses to the portion of the survey that measured economic risk preferences. After matching twins along measures of these preferences—a twin pair represents a single case in the data—our final sample included 111 identical (monozygotic, MZ) and 89 non-identical (dizygotic, DZ) twin pairs (400 individuals).

Similar to their Minnesota birth cohort, the sample was 98% white with an average age of 36.7 years (SD = 1.12). The zygosity of twins was previously established as part of the Minnesota Parenting Project, wherein a five-item questionnaire was used that has been previously shown to exceed 95% accuracy compared to serological (i.e., DNA-based) methods for establishing twin type (see Lykken, Bouchard, McGue, & Tellegen, 1990).

### Measuring economic risk preferences

We measured economic risk preferences with three questionnaire items that were ordered-categorical, each with three response options (endorsement proportions for the MZ and DZ groups are provided in parentheses).[4] The first item was taken from a questionnaire commonly administered to new customers being consulted for their economic risk preferences at major portfolio management companies (see Hube, 1998). This measure involved informing participants that they had won a ''big prize,'' but had to choose among one of three lotteries for their prize. The first lottery was a \$2000 prize with a probability of 1.0, resulting in an expected payoff $E[p\mathrm{X}] = \$2000$ (MZ = .71; DZ = .71), the second was a \$5000 prize with a probability of 0.5 with a 0.5 probability of receiving nothing, resulting in $E[p\mathrm{X}] = \$2500$ (MZ = .21; DZ = .21), and the third was a \$15 000 prize with a probability of 0.2 with a 0.8 probability of receiving nothing, resulting in $E[p\mathrm{X}] = \$3000$ (MZ = .08; DZ = .08). The difference in expected payoffs across these lotteries mirrors real-world investment decisions, where the three lotteries have, successively, higher expected payoffs and risks (see similar measures in other literature on risk, such as that by Murnighan, Alvin, & Francoise, 1988).

The second item informed participants that they were investing in retirement, which was 15 years away. They were asked to choose one of three different forms of investment. In order of least to most risky, the first was ''a money-market fund or guaranteed investment contract, giving up the possibility of major gains, but virtually assuring the safety of your principal'' (MZ = .09; DZ = .11), the second was, ''a 50–50 mix of bond funds and stock funds, in hopes of getting some growth,but also giving yourself some protection in the form of steady income'' (MZ = .44; DZ = .51), and the third was, ''aggressive growth mutual funds whose value will probably fluctuate significantly during the years, but have potential for impressive gains in the long term'' (MZ = .48; DZ = .38).

The third item informed participants that their employer—a private entity—is selling stock to employees, and that management has plans to take the company public in roughly three years. Until the company is taken public, there is no possibility of selling shares or receiving dividends, but if the company goes public, increases in stock valuation could be quite large. Participants were then asked how much they would invest in

[4]To conduct a meaningful comparison of answers to the questionnaire one must assume that the respondents have either similar levels of wealth or constant absolute risk aversion; the latter is a typical assumption in economics because of its tractability. See http://economics.about.com/library/glossary/bldef-cara-utility.htm.



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the stock where they could choose either ''none'' (MZ = .10; DZ = .12), ''two months' salary'' (MZ = .50; DZ = .49), or ''four months' salary'' (MZ = .39; DZ = .39), noting that additional investment is associated with additional risk.

## STATISTICAL ANALYSIS AND RESULTS

The latent variable modeling program *Mplus* version 5.1 (see Muthén & Muthén, 1998–2008) was used to apply genetic model-fitting techniques. These techniques specify an equation that decomposes observed phenotypic variance into the genetic and environmental *A*, *D*, *C*, and *E* parts. However, traditional analytic methods for such a model, which use estimation algorithms based on maximum likelihood, are only appropriate when variables may be assumed continuous and normally distributed. As noted above, our measures are ordered-categorical in nature. Therefore, as recommended in literature addressing genetic model-fitting techniques with such data (see Neale & Cardon, 1992), we used a weighted least squares (WLS) estimator to fit our models (for details see Muthén, 1998–2004). As noted by Muthén (1983), such an estimation technique assumes an underlying continuous normal distribution that produces the observed responses. The underlying continuous normal distribution is said to be composed of ''liabilities'' that produce the observed responses, and in the context of twin analyses the variance in the liability distributions across the MZ and DZ groups is decomposed into genetic and environmental parts (see Neale & Cardon, 1992). Given our relatively modest sample size and the critique that WLS estimators may be inappropriate with small samples (e.g., Bollen, 1989), we used a non-parametric bootstrapping technique with 10 000 draws when estimating all parameters (including standard errors and confidence intervals).

In order to allow each individual to have an overall measure of economic risk preferences, we estimated a latent composite variable to account for responses across all three questionnaire items. This was possible by setting the communality for each observed variable to 1.0, which is equivalent to a principal component, thereby allowing the latent composite variable to capture all of the variance in the liabilities across all twin pairs. Thus, in this model the relationship of interest is the difference in the correlation among the latent composite variables for MZ versus DZ twins, where a genetic effect would be indicated by a larger correlation among the latent variables for MZ twins than DZ twins.

Estimating the relationship among the composite variables revealed a correlation among the latent liabilities of .63 for the MZ group and .16 for the DZ group, suggesting a dominant genetic effect on economic risk preferences. Therefore, we did not pursue an ''ACE'' model, instead opting to estimate an ''ADE'' model, because, as mentioned above, dominant genetic effects preclude the possibility of a shared environmental effect (see Plomin et al., 2001). To test the effects of *A* and *D* on economic risk preferences, we first freely estimated an ADE model (see Table 1 for model $\chi^2$ values; see Table 2 for all parameter estimates). Then, we removed the *A* and *D* components to test for decrements in model fit associated with the removal of

Table 1. Model fit statistics

| | $\chi^2$ | df | $\Delta\chi^2$ | $\Delta$df |
|---|---|---|---|---|
| Model ADE | 359.51* | 43 | | |
| Model AE | 381.89* | 44 | 22.38* | 1 |
| Model DE | 359.51* | 44 | <.01 | 1 |

*Note*: All $\chi^2$ difference testing compares the fit of Model ADE to subsequent models; df = degrees of freedom; $\Delta\chi^2 = \chi^2$ difference; $\Delta$df = degrees of freedom difference.
*$p < .01$.



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Table 2. Parameter estimates

| | Parameter estimate | Standard error | *t*-value |
|---|---|---|---|
| Model ADE | | | |
| *A* | (−.37) <.01 (.37) | .19 | <.01 |
| *D* | (.24) .82 (1.40) | .30 | 2.77 |
| *E* | (.16) .62 (1.08) | .23 | 2.66 |
| *H* | (.15) .63 (1.00) | .25 | 2.57 |
| Model AE | | | |
| *A* | (.04) .67 (1.31) | .32 | 2.08 |
| *E* | (.40) .75 (1.10) | .18 | 4.23 |
| *H* | (−.06) .45 (.95) | .26 | 1.74 |
| Model DE | | | |
| *D* | (.29) .82 (1.35) | .27 | 3.05 |
| *E* | (.16) .62 (1.08) | .24 | 2.65 |
| *H* | (.15) .63 (1.00) | .25 | 2.54 |

*Note*: $A$ = additive genetic effect; $D$ = dominant genetic effect; $E$ = unshared environment; $H$ = heritability coefficient; all parameters were bootstrapped with 10 000 draws using a weighted least squares estimator; values in parentheses represent the boundaries of a 95% confidence interval.

these model parts.[5] Reductions in model fit were assessed with a traditional chi-square difference test after adjusting statistical significance values to account for the mixture of chi-squares that occurs when testing variances at the boundaries of their spaces (i.e., when variances are constrained to zero; see Dominicus, Skrondal, Gjessing, Pedersen, & Palmgren, 2006). Results indicated that removing $D$ significantly decreased model fit ($\Delta\chi^2$ (1) = 22.38, $p < .01$) but that removing $A$ did not influence model fit ($\Delta\chi^2$ (1) < .01, $p > .05$). This indicates that a dominant genetic effect was present, while an additive effect was not supported by the data.

Although the estimate for $A$ in the ADE model was virtually zero and removing $A$ from the ADE model had no impact on model fit, it is generally considered inappropriate to exclude additive genetic effects from twin studies when the goal is to estimate heritability (Hammon et al., 2001). Therefore, the original ADE model was retained in favor of either an AE or DE model. As shown in Table 2, $D$'s bootstrapped confidence interval did not encompass zero, while $A$'s was virtually *centered* at zero. Additionally, a dummy variable that estimated the heritability of economic risk preferences—99.99% of which was composed of variance associated with D—was estimated at 0.63, with a bootstrapped 95% confidence interval ranging between 0.15 and 1.00. These results indicate that there is a genetic effect on economic risk preferences, and that this genetic effect is associated almost entirely with a dominant genetic structure.[6] Additionally, these results indicate that a substantial proportion of variance in economic risk preferences may be attributed to unshared environmental factors, while no variance in these preferences may be attributed to shared environmental factors.

[5]Neale and Cardon (1992) note that such tests inform on the appropriateness of each nested model specification in relation to a less-constrained model.

[6]We conducted heritability analyses for the three items separately. In order of their presentation in the text, Item 1: $A = .00$, $D = .44$, $C = .00$, $E = .81$, $H = .20$; Item 2: $A = .00$, $D = .48$, $C = .00$, $E = .77$, $H = .23$; Item 3: $A = .00$, $D = .44$, $C = .00$, $E = .81$, $H = .19$. As these results show, the pattern of a dominant genetic effect exists across all items.



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## DISCUSSION AND CONCLUSION

The study of individual differences in economic risk preferences is far less prevalent than the study of situational or framing effects on such preferences; much of the extant decision-making literature assumes that economic risk preferences are largely determined by situational and environmental cues. The findings from our analyses support the hypothesis that there is a significant genetic component to economic risk preferences, suggesting that these preferences, and risk preferences more generally, may not only be considered dispositional in nature, but also as being in-born. That is, risk preferences appear to be coded in the human genome as our results show that economic risk preferences are two-thirds genetically and one-third environmentally determined. This result fits well with other work on individual difference variables (e.g., Finkel & McGue, 1997), where sizable heritabilities have been found for variables such as harm avoidance and stress reactions.

However, unlike many other findings of heritability, an important feature of our results is that virtually all of the variance in economic risk preferences attributable to genetic causes is a function of a dominant genetic effect. Although the rarity of a dominant genetic effect in behavior genetics research does indicate the need to replicate the current findings, the current results suggest that economic risk preferences are not a genetically additive trait, but instead are genetically coded in a fashion that makes them dominant/recessive. Although this finding opens the door to an understanding of the nature of economic risk preferences' place in the human genome, future studies assessing the specific genes associated with these preferences will pave the way towards a more complete understanding of exactly how nature determines economic risk preferences.

For example, it is well known that both serotonergic and dopaminergic neurological systems play important roles in a variety of human functions (for a discussion related to economics, see Camerer, Loewenstein, & Prelec, 2005). More specifically, both of these neural substrates have been linked to various cognitive processes relevant to differences in risk preferences, such as impulsivity (Carver & Miller, 2006) and the extent to which emotion-based information is used in making decisions (Sevy et al., 2006). Additionally, there is evidence that dominant/recessive modes of genetic transmission account for differences in serotonergic and dopaminergic systems that have been shown to be related to risk preferences, such as impulsivity (e.g., Oades et al., 2008). Given the current findings, researchers may be motivated to examine those aspects of these neurological systems that are transmitted in a dominant/recessive fashion when exploring the physiological underpinnings of risk preferences.

Regarding the ''nurture'' effect on economic risk preferences, our findings suggest that these preferences are roughly one-third environmentally determined. Importantly, for the variance in economic risk preferences attributable to twins' environment, there was no effect of shared environmental factors, but instead a sizable effect of unshared environmental factors was found. In tandem, these results suggest that, irrespective of shared child-rearing techniques by parents and shared socio-economic status, economic risk preferences diverge between siblings by the time they are middle-aged in a way that nullifies the effect of a shared environment during childhood. Such a result might seem to suggest that, for example, parental training regarding risk taking and, perhaps, monetary investment strategies might have little effect on an offspring's economic risk preferences later in life.

However, because we did not measure parenting behavior during childhood it is possible that parents often simply fail to train their children in the domain of risk taking. Without such training, a shared environmental effect on economic risk preferences would be difficult to find and, therefore, we are hesitant to claim that shared environmental effects cannot influence economic risk preferences in adults. Future studies should employ designs capable of more thoroughly addressing the question of shared environmental effects on economic risk preferences. For example, by investigating the heritability of risk preferences across a person's lifespan, researchers might discover that during childhood and very early adulthood, there is a significant effect of shared environment on economic risk preferences. However, consistent with literature showing that



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parental influence on child risk preferences decreases with age (see Levin & Hart, 2003), this effect might dissipate to null by middle adulthood—as we observe here.

Additionally, it is important to note that our study design is incapable of examining the heritability of risk preferences in relation to shared-environment effects that are common to all participants. As we note above, Weber and Hsee (1998) provide evidence that risk preferences may vary across cultures, which would suggest an effect of shared environment on such preferences. However, because all individuals within our sample share a common culture, we do not see our failure to find an effect of shared environment as inconsistent with cultural differences in risk preferences. Instead, it is likely the case that culture exerts a common influence on individuals across households, while shared-environmental effects caused by being raised in a common household are null for middle-aged adults.

Finally, in relation to the lack of a shared-environment effect, it is notable that MZ twins may experience a more similar environment than DZ twins. For example, MZ twins experience the same uterine environment during development, and they are also much more likely to be treated similarly by others than DZ twins (Plomin et al., 2001). Accordingly, higher levels of covariance among MZ twins could exist that are actually due to environmental factors, although such similarities would masquerade as genetic effects in the traditional twin statistical model.

A primary limitation of our study was that all of the twin pairs in our sample were male, disabling us from making strong inferences regarding the heritability of economic risk preferences for humans in general. Although theoretical and empirical work supports the view that males are more risk-prone than females across a variety of social domains, such as mate selection (Byrnes, Miller, & Schafer, 1999), such differences do not preclude similar levels of economic risk preference heritability across males and females. For example, research by Finkel and McGue (1997) has shown similar levels of heritability across a variety of attitudinal variables in males and females, even when mean levels along some of the variables differed across males and females. Future studies should be conducted with both male and female twin pairs to examine for similarity in heritabilities, paying close attention to attempt to replicate the dominant genetic effect observed here across both genders.[7]

Additionally, although it was the focus of our study to examine the heritability of economic risk preferences, this focus necessarily limits our ability to generalize our results to risk preferences in other domains, as well as risk preferences more generally. As shown in a variety of studies (e.g., Hanoch, Johnson, & Wilke, 2006; Johnson, Wilke, & Weber, 2004; Mellers, Schwartz, & Weber, 1997; Weber, Blais, & Betz, 2002), risk preferences are often domain-specific. Hence, domain-specific scales such as the DOSPERT (Domain Specific Risk Taking; Blais & Weber, 2006) may be usefully employed to measure specific aspects of an individual's risk preferences. Given these findings, it is possible that risk preferences may be differentially heritable across different decision-making domains. Only future empirical analyses can uncover the extent to which our results generalize to such domains.

In summary, the current study opens avenues for research into individual differences in economic risk preferences and the genetic causes underlying these preferences. Although we find that such preferences have a significant heritable component, we are unable to address important issues with our data, such as the number of genes involved in determining economic risk preferences and their relative effects on these preferences, as well as the heritability of risk preferences specific to other domains (e.g., social or health-related risk preferences). However, barring this shortcoming, our results do indicate that a genetically oriented approach to understanding the underlying causes of risk preferences has the potential to bear significant fruits in the study of human decision-making. Future research should endeavor to uncover the many relevant biological

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[7]Barber and Odean (2001, p. 285–286), citing relevant studies, note that there "is considerable evidence that men and women have different attitudes towards risk." However, the cited studies do not examine the issue of differential heritability of economic risk preferences across males and females.



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and psychological processes that determine everyday decisions involving an element of risk in relation to how these processes may be genetically determined.[8]

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[8]For a review of the literature on the biological basis of economic decision-making, see Robson (2001).



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