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Abstract

In this paper, we use a sample of almost 30,000 Swedish mono- and dizygotic twins to study the heritability of financial risk-taking. Following a major pension reform in the year 2000, virtually all Swedish adults had to simultaneously make a finnancial decision aaecting post-retirement wealth. We take this event as a field experiment to infer risk preferences. We use standard techniques from behavior genetics to partition variation in risk-taking into environmental and genetic components. Our findings suggest that genetic variation is an important source of individual heterogeneity in financial risk-taking.

Keywords: genetics, risk-taking, portfolio investment, twins.

JEL codes: D01, G11

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Introduction

Parents and their children exhibit considerable similarity in self-reported attitudes toward risk (Charles and Hurst (2003); Dohmen *et al.* (2006); Hryshko *et al.* (2007)), as well as in their choice of what assets to hold (Chiteji and Stafford (1999)). Yet, little is known about the mechanisms generating these correlations. Do they arise because parents pass on genes for certain traits associated with risk preferences to their children, or is it, as often postulated, merely a reflection of parental socializing influences? In a recent string of papers (Wallace *et al.* (2007); Cesarini *et al.* (2008a, 2009)), laboratory experiments designed to elicit preferences were run on a sample of twins. Comparing the behavior of monozygotic (MZ) twins to that of dizygotic (DZ) twins is a form of quasi-experiment. MZ twins reared together share the same environment and the same genes, and while DZ twins reared together also share the same environment, their degree of genetic relatedness is no greater than that of ordinary siblings. A significantly higher observed correlation for MZ twins than DZ twins is therefore usually taken as evidence that a trait is under genetic influence. Estimating fairly standard behavior genetic models, the results in Wallace *et al.* (2007) and Cesarini *et al.* (2008a, 2009) suggest that heritability - the share of individual variation that can be explained by genetic influences - for a number of economic preferences, including risk preferences, is typically somewhere between 20 and 40 %.

Yet, eliciting preferences experimentally has at least two distinct disadvantages. First, there is genuine uncertainty about the extent to which laboratory behavior generalizes to the field (Levitt and List (2007)). Second, the sample sizes in the above cited studies, though very large by behavioral economic standards, still do not allow for precise inference. In this paper, we use microdata from the Swedish individualized pension savings account introduced in 2000 to extend the previous literature from the laboratory to the field. As part of the transition to a new pension system, virtually all adult Swedes born after 1938 had to make simultaneous investment decisions with potentially far-reaching effects on their post-retirement wealth. In particular, they had to compose an investment portfolio from a menu of more than six

hundred funds. We take this event, which is known as the "Big Bang" of the Swedish financial sector, as a field experiment to infer risk preferences. Matching individual portfolio data to the Swedish Twin Registry, we then employ standard methods from behavior genetics and estimate the heritability of preferences for financial risk-taking. Unlike small stake gambles in the laboratory, or attitudinal risk questions, the investment decisions made in the pension savings accounts are real financial decisions that have real economic consequences. Moreover, our dataset is very large, allowing us to estimate parameters with much greater precision than previous experimental studies.

To our knowledge, this paper is the first to use behavior genetic techniques to document the heritability of risk-taking in the financial market, as well as outside the laboratory. There is, however, a related literature in economics which has considered economic outcome variables such as educational attainment, income and socioeconomic status (Taubman (1976); Behrman and Taubman (1989); Lichtenstein *et al.* (1992); Plug and Vijverberg (2003); Björklund, Lindahl and Plug (2006); Björklund, Jäntti and Solon (2007); Sacerdote (2007)). The general idea behind these papers is to make reasonable assumptions about the genetic relationships of relatives to separate the effects of genetic and environmental variation (Sacerdote (forthcoming)). Behavior genetic techniques are in no way restricted to twins, and many of the above studies also include adoptees, as well as other sibling types. Taken together, adoption, sibling and twin studies point to a role for both genetic and cultural transmission of economic outcomes.

The estimates of heritability that we obtain match the laboratory evidence in Cesarini *et al.* (2009) very closely, and suggest that approximately 25 % of the individual variation in financial risk-taking is due to genetic influences. This implies that a significant portion of the previously observed parent-child resemblance in risk attitudes is due to genetic transmission. Furthermore, besides establishing that a key economic preference is heritable, an important result in and of itself, we believe that our findings have broader implications. For instance, the share of individual variation explained by genes is much higher than the $R^2 s$ typically

obtained in standard empirical models of financial risk-taking (Cohn *et al.* (1975); Pålsson (1996); Palme *et al.* (2007)). In an early microdata study of portfolio choice Cohn *et al.* (1975) found R^2*s* of approximately 0.10 using a set of demographic and socio-economic controls, while Pålsson (1996) report substantially lower estimates for Swedish registry based data. Importantly, these results for individual portfolios of total asset holdings are close to the R^2's found when considering portfolio choice for the Swedish individualized pension savings accounts in isolation (Säve-Söderbergh (2005); Palme *et al.* (2007)).

Our findings may also be relevant for research on the intergenerational transmission of economic status. Reviewing the literature, Bowles and Gintis (2002) suggest that further study of non-cognitive behavioral traits and preferences may help explain the fact that even though income is heritable (Taubman (1976)), simple calibration exercises show that the genetic transmission of intelligence can account for at most a moderate share of the parent-child correlation. Poterba *et al.* (2000) show the substantial effects risk-preferences can have on accumulation of post-retirement wealth and thus potentially on intergenerational transmission of economic status.[1]

The paper is structured as follows. In Section 2, we describe the Swedish Pension reform and our dataset. In Section 3, we describe twin methodology. In Section 4, we present our results, and relate them to previous findings. In section 5 we investigate and discuss the robustness and generalizability of our findings, and in section 6 we relate them to the literature on behavior genetics and the biological basis of risk preferences. Section 7 concludes.

I. Data

A. The Swedish Pension Reform

In 1994, legislation gradually introducing a new pension system was passed by the Swedish parliament in response to demographic challenges and underfinancing of the pay-as-you-go system that had been in place since the 1960s.[2] The new system is based on

a contribution rate of 18.5 percent on earnings, whereof 2.5 percentage points accrue to mandatory individual self-directed accounts, one of the system's key features.

As part of the introduction of the new system, a government body - the Premium Pension Agency - was set up and assigned the responsibility of handling the individual investment accounts. Most adult Swedes born after 1938 were invited to decide how to invest the balance on their individualized pension savings accounts, but the system only fully applied to individuals born 1954 and onwards.[3] The "Big Bang" occurred toward the end of 2000, when all participants in the new system had to simultaneously decide how to invest their balances. Some 68 percent of the eligible population made an active decision. Individuals who did not make an active choice had their money invested in a default fund.

Participants could compose a portfolio consisting of no more than 5 funds from a very large menu of options comprising more than 600 different funds.[4] All eligible Swedes were sent a catalogue in which available funds were listed with information on management fees and the investment strategy of each fund. For the approximately 400 funds that had a historical record, returns and standard deviation of returns for the preceding three years were also given. These funds were also color-coded by risk level: from red (high risk) to green (low risk). The circumstances under which these investment decisions were made make the experiment uniquely suitable for inferring risk preferences among individuals with little or no financial fluency.

B. Portfolio Risk Data

Our primary measure of portfolio risk, which we denote Risk 1, is the average risk level of the funds invested in by the individual, with the risk of each fund measured as the standard deviation of the rate of return over the previous three years. In cases where historical returns were not available, these values were imputed by assigning the average value of risk for similar types of funds in the sample.[5] This measure is similar to that employed in Säve-Söderbergh (2008) and Palme *et al.* (2007), with the one notable exception that we also include twins

whose money was invested in the default fund.[6] As a robustness check, we also calculated a second risk measure, Risk 2, as the weighted share of high-risk funds in an individual's portfolio.[7]

C. The Swedish Twin Registry

The Swedish Twin Registry, the largest in the world, contains information on nearly all twin births in Sweden since 1886, and has been described in detail elsewhere (Lichtenstein *et al.* (2006)). Our sample includes individuals who have participated in at least one of the Twin Registry's surveys. For these respondents, we can establish zygosity with reasonable confidence based on survey questions with proven reliability (Lichtenstein *et al.* (2006)). In practice, roughly 90 % of the twins in our dataset come from one of two sources. The primary source is the web-based survey STAGE (The Study of Twin Adults: Genes and Environment). This survey was administered between November 2005 and March 2006 to all twins born in Sweden between 1959 and 1985, and it attained a response rate of 60 %. Data on individuals born between 1938 and 1958 were obtained from SALT (Screening Across the Lifespan Twin study), a survey conducted by telephone in 1998. SALT attained a response rate of 74 % (Lichtenstein *et al.* (2006)).[8] Though these response rates are not alarmingly low, we acknowledge that our sample is not fully representative of the population of twins. Considering all complete same sex twin pairs born after 1938 gives a total of 7224 female pairs, of which 3346 are monozygotic, and 6338 male pairs, of which 2747 are monozygotic.

II. Method

Our analysis uses [a proxy for] the portfolio risks chosen by twins to estimate the degree to which variation is influenced by additive genetic factors (A), environmental factors shared or common to the two twins in a pair (C), and unshared environmental (E) factors which are specific to each twin. Additive genetic effects are defined as the sum of the effects of individual

alleles influencing a trait. Common environment effects are those environmental influences shared by both twins. Examples include childhood diet, schooling, parental socialization and shared peer influences. Unshared environmental effects include influences not shared by the co-twins as well as measurement and response error.

The basic idea behind a behavior genetic decomposition is simple. MZ and DZ twins differ in their genetic relatedness. If one is willing to assume that the common environment does not exert greater influence in MZ twins, then a greater similarity between MZ twins can be taken as evidence that the trait is under genetic influence.

Several authors, most recently Sacerdote (forthcoming), have noted that moving from a crude comparison of correlations to a full-fledged variance decomposition requires making strong independence and functional form assumptions. Therefore, our empirical analysis proceeds in two steps. We first abstain from imposing any structural assumptions, and simply compare the correlations of MZ and DZ twins using the bootstrap. Letting N_{MZ} be the number of MZ pairs without missing data, we draw N_{MZ} pairs with replacement 1000 times and calculate the non-parametric correlation each time. We proceed analogously for DZ twins, and then create a 1000 by 1 vector where the DZ correlation is subtracted from the MZ correlation for each draw. This gives a distribution for the difference in correlation between the two samples. The p-value for the test of the hypothesis that the two correlations are equal is then easily computed by counting the number of instances where the vector of differences takes a negative value and dividing by ten.

We then proceed to a standard behavior genetic variance decomposition. The workhorse model in the behavior genetics literature, known as the ACE model, posits that additive genetic factors (A), common environmental factors (C), and specific environmental factors (E) account for all individual differences in the trait of interest. Start with the case of MZ twins. Let all variables, including the trait, be expressed as deviations from zero and standardize them to have unit variance. Consider a pair of MZ twins and suppose first that the outcome variable can be written as the sum of two independent influences: additive

genetic effects, A, and environmental influences, U. We then have that,

$$P = aA + uU,$$

and, using a superscript to denote the variables for twin 2 in a pair,

$$P' = aA' + uU'.$$

Since for MZ twins $A = A'$, the covariance (which, due to our normalization, is also a correlation) between the outcome variables of the two twins is given by,

$$\rho_{MZ} = a^2 + u^2 COV(U, U')_{MZ}.$$

Now consider a DZ pair. Under the assumptions of random-assortative mating with respect to the trait of interest, it will be the case that $COV(A, A') = 0.5$.[9] We then have that,

$$\rho_{DZ} = \frac{1}{2}a^2 + u^2 COV(U, U')_{DZ}.$$

Finally, we impose the equal environment assumption, namely that,

$$COV(U, U')_{MZ} = COV(U, U')_{DZ}.$$

Under these, admittedly strong, assumptions it is easy to see that heritability, the fraction of variance explained by genetic factors, is identified as $a^2 = 2(\rho_{MZ} - \rho_{DZ})$. In the standard behavior genetics framework, environmental influences are generally written as the sum of a common environmental component (C) and a non-shared environmental component (E) such that,

$$P = aA + cC + eE.$$

With this terminology, the environmental covariance component of the trait correlation,

$u^2COV(U, U')$, can be written as c^2, since by definition any covariance must derive only from the common component. This allows us to write the individual variation as the sum of three components a^2, c^2, and e^2; a^2 is the share of variance explained by genetic differences, c^2 is the share of variance explained by common environmental influences, and e^2 the share of variance explained by non-shared environmental influences. There are a number of ways in which the parameters of this model can be estimated. We follow standard practice in using maximum likelihood under the assumption that the outcome variables come from a bivariate normal distribution.[10]. In particular, following directly from the above derivation, the likelihood is maximized under the restriction that the variance-covariance matrix is of the form,

$$\sum = \begin{bmatrix} a^2 + c^2 + e^2 & R_i a^2 + c^2 \\ R_i a^2 + c^2 & a^2 + c^2 + e^2 \end{bmatrix},$$

where R_i takes the value 1 if the observation is of an MZ pair, and 0.5 otherwise. The analyses are run in MPLUS (Muthén and Muthén (2006)), a numerical optimizer often used in behavior genetics.

III. Results

A first diagnostic of genetic influences comes from examining the MZ and DZ correlations. These are reported in Table I. Interestingly, there are no major differences between men and women in the patterns of correlations, with MZ correlations being consistently higher than the DZ correlations. In women the correlations are 0.27 and 0.16. In men, the correlations are 0.29 and 0.13. An MZ correlation, as we have noted, captures all determinants of financial risktaking that identical twins share; that is, genotype and shared environmental influences. In other words, the joint influence of genes and shared environment explains nearly 30 percent of the variation portfolio risk. The correlations for our second risk measure, Risk 2, are very similar, which demonstrates that most variation in risk

is driven by differences in the share of equity in the portfolio. Some summary statistics are reported in Table II.

In the two columns of Table III we report results from the basic model, without age controls (Column 1) and with age controls (Column 2).[11] In the top panel, we report results from a model where variance components are allowed to differ by gender. Similar patterns hold for men and women. Consider for example the results from Model 1. In women, heritability is estimated at 0.22 (99% CI, 0.07-0.31) and in men, heritability is estimated at 0.28 (99 % CI, 0.15-0.32). In both cases, most of the remaining variation comes from non-shared environment.

The lower panel reports results from a model where the restriction that variance components are the same in men and women has been imposed. Whether this restriction entails a significant deterioration in fit can be tested using a likelihood-ratio test. We can reject the hypothesis that the variance components are the same in men and women ($\Delta\chi^2 = 10.14$, $df = 3$, $p < 0.05$) but this is probably a consequence of the large sample size rather than of economically interesting differences. In the pooled model, heritability is estimated at 0.26 (99 % CI, 0.15-0.31) and common environment is estimated at 0.01 (99 % CI, 0.00-0.10). The estimates are very similar when risk residualized on age is used as the dependent variable. Heritability is estimated at 0.22 in women (99 % CI, 0.07-0.31), 0.28 in men (99 % CI, 0.15-0.32) and the pooled model produces a heritability estimate of 0.23 (0.17-0.23).

A. Relationship to Previous Findings

As noted above, previous studies have shown that there is moderate parent-child correlation both in attitudes toward risk (Charles and Hurst (2003); Dohmen *et al.* (2006); Hryshko *et al.* (2007)), and in choice of asset holdings (Chiteji and Stafford (1999)), but a parent-child correlation in isolation cannot inform us about the relative importance of genetic and environmental influences.The magnitude of our estimates can easily be reconciled with this existing literature on intergenerational transmission. For instance, the parent-child cor-

relation found in Dohmen *et al.*'s (2006) representative German sample imply upper bounds on heritability of approximately 0.35, and the point estimates of heritability in Cesarini *et al.* (2009) range from 0.14 to 0.35.[12] This convergence of results across different methodologies is reassuring because it suggests that the findings are not driven by confounding factors particular to our study. Such include the fact that our sample is not fully representative (unlike the sample in Dohmen *et al.* (2006)), or the fact that we cannot rule out that twins have communicated about their choice of portfolio (unlike the experimental evidence in Cesarini *et al.* (2009) where twins always participated in the same session).

Furthermore, it is interesting to note that the share of individual variation that is explained by genes as reported above is much higher than the R^2 typically found in standard empirical models of financial risk-taking (Cohn *et al.* (1975); Pålsson (1996); Palme *et al.* (2007)). In an early microdata study of portfolio choice using a non-representative sample Cohn *et al.* (1975) obtained R^2's of approximately 0.10 using a set of demographic and socio-economic controls, while Pålsson (1996) report substantially lower estimates for Swedish registry based data. Perhaps most strikingly, our single variable A typically explains a substantially larger fraction of individual variation in risk-taking for the Swedish individualized pension accounts than the up to 8 controls in Palme *et al.* (2007), $R^2 \leq 0.042$, and the approximately 20 controls in Säve-Söderbergh (2005) (whose highest reported R^2 is 0.112). A fairly robust finding is that there are differences between men and women in their average propensity to take financial risk (Eckel and Grossman (forthcoming); Sunden and Surette (1998); Säve-Söderbergh (2008)): In this context it is interesting to note that these sex differences are small relative to the genetic differences within-sex suggested by our estimates.

IV. Robustness and Generalizeability

To establish how sensitive our results are to variations in the underlying assumptions, we now turn to an examination of the numerous potential sources of bias, their direction,

and the extent to which they might be expected to impact our findings.

A. Representativeness and Generealizability

In order to ascertain how representative our sample is of the population at large, we compare it disaggregated on zygosity and sex to the Swedish population born between 1938 and 1978 on a number of demographic background variables. The results are reported in Table IV.[13] Respondents tend to have slightly higher incomes than the population average, but unlike other studies (Behrman *et al.* (1994); Ashenfelter and Krueger (1994); Rouse (1999)), we do not find any economically interesting attrition with respect to education. There is however a slight tendency for participants to have higher marriage rates than the population as a whole. Finally, STAGE and SALT respondents are also somewhat older than the average for the 1938-1978 cohorts.

Obviously, it is impossible to fully establish the "selectivity" of our sample. The propensity to respond to a survey is likely associated with a number of background characteristics which are not readily measurable but which may nevertheless be influencing our findings, such as general motivational factors. If people with certain background characteristics are overrepresented, and if heritability is associated with these background characteristics, then the heritability estimate will be biased in the direction of this association.

In addition to asking how representative our sample of twins is, it is also important to consider whether twins as a group differ from the population as a whole with respect to unobservables. Few variables have been found to differ between twins and non-twins (Kendler *et al.* (1996)) and we can think of no good reason why the experience of having grown up with a twin should have idiosyncratically affected financial decisionmaking in adult life.

B. Equal Environment Assumption

Critics of the classical twin design cite a number of alleged failures of the equal environment assumption which states that shared environmental influences are not more important for monozygotic twins than for dizygotic twins. A number of objections have been raised, including that parents, on average, give MZ twins more similar treatment.[14] It is important to emphasize that even if MZ twins receive more similar treatment from their parents, this does not in and of itself constitute a violation of the assumption; greater similarity in environment may be caused by the greater similarity in genotypes (Plomin *et al.* (2001)). In the context of research on personality and IQ, where the equal environment assumption has been tested most rigorously, the evidence is fairly convincing that any bias that arises from this restriction is not of first order (Bouchard (1998)).

Most importantly, for measures of personality and cognitive ability, studies of MZ and DZ twins who were reared apart tend to produce estimates of heritability similar to those using twins reared together (Bouchard (1998)). Since studies of twins reared apart do not rely on the equal environments assumption, findings from such studies seem to validate the basic model. Also, in the relatively rare cases where parents miscategorize their twins as MZ instead of DZ (or the converse), differences in correlations of cognitive ability and personality persist (Bouchard and McGue (2003)).

C. Reciprocal Influences

The basic model assumes an absence of reciprocal influences between twins. If twins influence each other's choices positively, their degree of similarity will be inflated. Moreover, if this effect is stronger in MZ twins than in DZ twins, it will bias upward the estimate of heritability. The STAGE and SALT datasets both contain information on the frequency of contact between twins. As is commonly found in twin studies, monozygotic twins do interact more than dizygotic twins. On average, MZ pairs reported 3.3 interactions per week at the time of the survey, whereas DZ pairs reported an average of 1.8 interactions per week.[15]

Running separate regressions by gender, where the dependent variable is the squared within-pair difference in portfolio risk, and the independent variables are frequency of contact and zygosity, frequency of contact is a significant predictor of within twin-pair squared difference in portfolio risk, for both men and women. The presence of a statistically significant effect does not, however, prove that the frequency of contact is causing increased similarity. Much research has been devoted to establishing the direction of causality. Lykken *et al.* (1990) and Posner *et al.* (1996) offer some evidence suggesting that twins similar in personality tend to stay in contact with one another, and not the other way round.

One crude way of examining whether twins have communicated about their choice of funds is to ask how common it is for both twins to choose the same portfolio. Excluding pairs where both twins selected the default portfolio, of the remaining MZ twins, 8 % choose the same portfolio as their co-twin. In DZ twins the corresponding figure is 3 %. To further examine the sensitivity of our results to this source of bias, we conduct two robustness checks, the results of which are reported in Table V.

First, we drop all pairs in which both individuals chose the same portfolio, and rerun the analyses. Obviously, by discarding these observations, both MZ and DZ correlations will drop. Furthermore, these adjusted correlations will be downward biased if twins choosing identical portfolios are more similar than average with respect to risk-preferences. This sample restriction produces a pooled heritability estimate of 0.20 (99% CI, 0.11-0.23) which, under the assumption that communication only affects choices through identical portfolios, can serve as a lower bound to our heritability estimate in the presence of reciprocal action.

Second, we make use of our frequency of contact variable. Specifically, we stratify frequency of contact into 15 groups, and for each sex and level of contact we then randomly drop the required number of either MZ or DZ pairs to make the number of MZ and DZ pairs equal. In this restricted sample, the distribution of frequency of contact is, by construction, virtually the same in the MZ and DZ groups. Rerunning the analyses on this subset of the data, the heritability estimate in the pooled model falls to 0.19 (99% CI, 0.07-0.28). The

finding that the heritability estimates only fall marginally is reassuring since it demonstrates that frequency of contact is not a major influence on our main result.[16]

Our interpretation of these results is that the twins who opted for the same retirement fund would generally have chosen portfolios with similar levels of risk even without the opportunity to consult each other.

D. Misclassification and Measurement Error

We use the Swedish Twin Registry's standard algorithm to establish zygosity. The algorithm has been validated against DNA-based evidence, and studies show that misclassification is typically of the order 2-5 % (Lichtenstein *et al. (*2006)). Purely random assignment error would bias heritability downward, since the difference in genetic relatedness between pairs assigned as MZ or DZ would diminish to less than one half. However, misclassification is probably not random, but related to physical similarity (notice that the questions we use to establish zygosity are solely based on assessments of physical similarity). The relevant question is then if physical similarity is somehow related to the similarity with respect to behavior. The classical reference on this topic is Matheny *et al* (1976), who administered two intelligence tests, two perceptual tests, one reading test, one test of speech articulation, and one personality inventory to twins and found that "correlations revealed no systematic relation between the similarity of appearance and the similarity of behaviors for either the identical twin pairs or the same-sex fraternal twin pairs."[17] We conclude that the bias which arises due to misclassification is likely small and leads to an understatement of heritability.

As in the case of misclassification, measurement errors tend bias a^2 and c^2 downwards since any such error will be subsumed under the estimate of e^2. In the simplest case where the preference is observed with a mean zero random error with variance σ_ϵ^2, it is easy to show that the estimates of a^2 and c^2 need to be scaled up by a factor of $\frac{1}{1-\sigma_\epsilon^2}$. But, whereas measurement error is easy to conceptualize in psychometric research as the test-retest reliability of some instrument designed to measure a personality trait, it is less clear in the present case where

it presumably would involve the choice of actual portfolio risk to be related to factors other than risk prefereces. While this is certainly likely to be the case, it is far from obvious how the reliability of actual observed risk-taking in the field convincingly could be tested.

V. Heritability and the Biological Basis of Risk-Taking

It is important to emphasize two features of **this** behavior genetic model when interpreting our findings. First, the model produces estimates of the proportion of variance explained. Thus, it does not shed any direct light on the determinants of the average proclivity to take risks. This distinction is important. For instance, if genetic transmission in a studied population is uniform, then a trait that is primarily acquired through genes might actually show a low, or zero, estimate of heritability. Alternatively, consider a culturally homogenous environment with little variation in how parents, whether consciously or not, instill certain beliefs and values in their children. In such an environment, it is quite possible that common environmental influences are important determinants of the average propensity to take financial risks, but that differences in common environmental influences are not an important source of variation. Second, the model is based on strong functional form and independence assumptions.

It is interesting to note, however, that our results are in line with the very voluminous and closely related behavior genetic literature on personality and attitudes (Bouchard (1998); Bouchard and McGue (2003); Plomin *et al.* (2006)), much of which has employed other types of sibling relations. While not measuring risk preferences per se, several suggested dimensions of personality are thought to be correlated with actual risk-taking. In a recent metastudy of parent-child resemblance in personality, Loehlin (2005) report average correlations of 0.13 for personality and 0.26 for attitudes in families with children reared by their biological parents.[18] As with our findings, twin and adoption studies strongly suggest that the primary explanation for these correlations is genetic transmission (Bouchard and McGue (2003); Loehlin (2005)). For instance, the correlations for personality and attitudes are 0.04 and

0.07 respectively between adopted children and their non-biological parents, but 0.13 and 0.20 between adopted children and their biological parents (Loehlin (2005)). Thus, seen in the context of the behavior genetic literature there is nothing anomalous about the finding of moderate heritability, a low effect of shared environment, and a large effect of non-shared environment for financial risk-taking. Indeed, these findings match Turkheimer's (2000) three laws of behavior genetics perfectly.[19]

The fact that a trait is heritable does not imply that there are genes with a direct effect on the trait. However, sensation and novelty seeking are both heritable and presumably correlated with risk-taking, and molecular genetic studies have implicated a number of particular genes associated with these traits (Koopmans *et al.* (1995); Zuckerman and Kuhlman (2000); Munafò *et al.* (2002); Kreek *et al.* (2005)). In addition to particular genes, several studies have found significant relationships between risk-taking and other biological factors such as patterns of brain activation and testosterone levels (Kuhnen and Knutson (2005); Cardinal (2006); Apicella *et al.* (2008)). It is worth noting that hormone levels (Harris et al. (1998)) and brain structure (Toga and Thompson (2005)) are both heritable, providing some indirect support for our hypothesis.

Yet, it seems very likely that some of the genetic effects may operate through genome-wide influences on variables which in turn affect risk-taking. For instance, one early paper found that participants' education and income levels were related to asset allocation decisions in mandatory private savings accounts, with less educated and lower income participants being less inclined to invest in equity securities (Poterba and Wise (1996)), although this finding is not supported by Palme *et al.* (2007). Differences in financial fluency (Bhandari and Deaves (2007) and health (Berkowitz and Qiu (2006)) are other candidate variables.[20] There are also evidence of small differences between men and women in their average propensity to take financial risk (Sunden and Surette (1998); Säve-Söderbergh (2008)), though it has been suggested that the magnitude of the difference is sensitive to the inclusion of covariates (Sung and Hanna (1998); VanDerhei and Olsen (2000)).

VI. Conclusion

In this paper, we have matched data on the mandatory pension investment decisions made in the fall 2000 to the Swedish Twin Registry in an attempt to estimate the genetic influence on variation in financial risk-taking. Relative to the experimental and survey evidence reported in Cesarini *et al.* (2009), a distinct advantage of our approach is that we examine risk-taking behavior in a field setting with large financial incentives attached to performance. Furthermore, relative to Dohmen *et al.* (2006), a second advantage of our approach is that the use of twin data allows us to shed light on the relative importance of environmental and genetic differences as sources of variation. Our finding that approximately 25% of variation in portfolio risk is due to genetic influences is in line with this previous, but small, experimental and intergenerational literature as well as the behavior genetics literature in general. The explanatory power of the genetic effect that we find is also typically at least twice as large as the R^2s found in previous non-twin studies using the same data and up to as many as 20 controls. In short, this paper is the first to use behavior genetic techniques to document the heritability of risk-taking in financial markets, as well as outside the laboratory, and the results strongly suggest that genetic variation is an important source of individual heterogeneity.

In addition to exploring specific mechanisms, we can think of a number of avenues for further work along the lines of this paper. Constructing a dataset similar to ours but with adoptees instead of twins would provide more precise estimates of the relative importance of common environmental influences. Also, augmenting the twin dataset with other sibling types of varying degrees of genetic relatedness, and, ideally, rearing environments, would allow researchers to explore the possibility of non-additivity or to test the equal environment assumption (See, for example, Björklund *et al.* (2005)) . If other work on attitudes and personality provides any guidance, we would expect that some of the genetic influences reported in this paper are in fact non-additive.[21] Regardless of what evolutionary dynamics led to the genetic variation that we observe for preferences in financial risk-taking[22], the

fact is that genetic differences explain a large share of individual variation in risk-taking. In light of these findings, we suggest that the further study of the biological and genetic basis of human risk-taking behaviors will lead to a more comprehensive theory of financial decision-making.

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Notes

[1]However, preferences are notoriously diffucult to measure, and attitudes toward risk is only one of many dimensions of preferences, whose individual effects may be small, but whose combined effect might be substantial. Moreover, attitudes towards risk may well interact with other variables and form a non-linear relationship with socio-economic status. Such non-linearities have been documented by Turner and Martinez (1977) in the context of scores on the Mach V scale, which measures the degree of Macchiavellian personality traits. They provide evidence that individual scores on this personality test have differential effects on income depending on social stratum.

[2]See Palmer (2000) for a detailed exposition of the new system.

[3]Only Swedes whose income exceeded SEK 36000 ($1 is roughly 6 SEK) in 1995, 36800 in 1996, 37000 in 1997 and 37100 in 1998 were eligible for fund selection in the year 2000.

[4]The official justification for this policy was that individuals should be able to select a portfolio that suited their preferences. For a criticism of this feature of the system, see Cronqvist and Thaler (2004) and Palme and Sundén (2004).

[5]The classification of funds was made by the Premium Pension Agency. Examples of types are "New Markets", "IT and Communication", and "Europe Small Enterprises". Our method of imputing missing values has no interesting effects on the estimates we report in this paper.

[6]Säve Söderbergh (2008) excludes individuals with the default portfolio on the grounds that its investment profile was not fully known when investment decisions were made in the fall of the year 2000. The reason its risk profile was not known is that it was constructed to reflect the profile of an average investor. On the other hand, it seems reasonable to assume that people had some expectation about the future level of risk in the default fund. In practice, none of the results reported in this paper are sensitive to this inclusion. This supports the notion that individuals not actively choosing a portfolio nevertheless conveyed some information about their risk preferences.

[7]A high risk fund was defined by the Premium Pension Agency as one holding at least 75% equity investments.

[8]Additionally, a small number of individuals in our sample responded to a survey sent out in 1973 (See, again, Lichtenstein *et al.*, 2002). These are also included.

[9]A full derivation of the latter result can be found in any text on quantitative genetics, for instance Falconer (1996) or Mather and Jinds (1982).

[10]Estimation of variance and covariances by maximum likelihood is consistent even if the normality assumption does not hold. However, standard errors will be biased, even though simulations show that small departures from normality are not too great a concern (Enders, 2001). As a robustness check, we have also estimated the model using the estimator of DeFries and Fulker (1985). They propose regressing twin 1's phenotype on: a constant, twin 2's phenotype and twin 2's phenotype interacted with the coefficient of genetic relatedness for the pair in question. DeFries and Fulker (1985) demonstrate that, under the additive genetic model, this produces unbiased estimates of the variance components. Kohler and Rodgers (2001) establish the asymptotic properties of this least square estimator with double entry. Computing standard errors using their method, we obtain heritability estimates that are extremely similar to those reported in the main body of the text.

[11]It is common in behavior genetics studies to residualize the phenotype on age, but interpretational issues arise. For example, age is obviously confounded with cohort effects, so removing age-related variation might actually remove environmental variation inadvertently. Or, gene expression might vary with age, in which case purging the outcome variable from age-related variation might actually have the unintended consequence of removing genetic variation.

[12]If the coefficient of genetic relatedness is 0.5, and only genes explain parent-child resemblance, then doubling the correlation will produce an estimate of heritability. If there are other, non-genetic, forces that can account for the correlation, then heritability estimated from parent offspring correlations will be upward biased.

[13]As is common in twin studies, women are slightly overrrepresented (McGue and Tellegen, 1980) in both STAGE and SALT, comprising 53 % of our sample.

[14]For further criticisms of the equal environment assumption, see Joseph (2002) and Pam et al., (1996), and the references therein.

[15]We construct the frequency of contact variable as follows. Subjects who report seven or more interactions (by e-mail, telephone or letter) per week are assigned a value of 7. All other subjects are assigned the number of interactions per week that they report. If we have data on both twins, we use the mean of the two reports.

[16]A significant drop in estimated heritability is, however, a necessary but not sufficient condition for frequency of contact to be the cause of greater similarity.

[17]More recently, Hettema, Neale and Kendler (1995) report no significant associations between physical similarity and phenotypic resemblance in four out of the five psychological disorders they consider (the one exception is bulimia.)

[18]Loehlin (2005) distinguishes young children from other children. When he only considers young children, the association between non-biological, but rearing, parents and their children is stronger. This finding that is consistent with the literature documenting increasing heratibility in adolescence (Bouchard and McGue 2003). Notice also that Loehlin's parent-offspring correlations yield considerably lower estimates of heritability than estimates based on samples of twins. He suggests that the difference is accounted for by non-additivity.

[19]Turkheimer's three laws are the following. First, all human behavioral traits are heritable. Second, the effect of being raised in the same family is smaller than the effect of genes. Third, a substantial portion of variation in complex human behavioral traits is not accounted for by the effects of genes and family.

[20]See Benartzi and Thaler (2001) for some evidence suggesting that individuals apply a diversification heuristic which is inconsistent with mean-variance optimizing behavior. In particular, individuals overinvest in asset types that are overrrepresented in the menu of funds.

[21]The basic ACE-model - like most behavior genetic models - assumes that genes influence the trait in an additive manner. That is to say, the genetic effect is simply the sum of all individual effects. This rules out epistasis (interaction between alleles at different loci) and dominance (interaction between alleles at a locus). A possible way to test for this would be to extend the dataset to include also sibling, parent-child, or even cousin data. The correlation between siblings, under an additive model, ought to be at least half the heritability obtained from a twin study. Were this assumption to fail, it would be diagnostic of some non-additivity being present in the data. This issue is explored in Loehlin (2005) in the context of the heritability of personality.

[22]Dall *et al.* (2004) and Penke *et al.* (2007) are two recent papers exploring the issue of how genetic variation can be maintained.

VIII. Tables and Figures

TABLE I.

WITHIN PAIR CORRELATIONS

		Women		p-value of diff	Men		p-value of diff
		MZ	DZ		MZ	DZ	
Risk 1	Pearson	0.27	0.16	<0.01	0.29	0.13	<0.01
	Spearman	0.28	0.16	<0.01	0.30	0.13	<0.01
	# pairs	3346	3878		2747	3591	
Risk 2	Pearson	0.26	0.13	<0.01	0.24	0.11	<0.01
	Spearman	0.26	0.14	<0.01	0.23	0.10	<0.01
	# pairs	3346	3878		2747	3591	

Note. Within twin pair correlations for Risk 1 and Risk 2. One sided p-values testing the equality of MZ and DZ correlations are reported.

TABLE II.

SUMMARY STATISTICS FOR RISK MEASURES

	Women		Men		Total	
	MZ	DZ	MZ	DZ	MZ	DZ
Risk 1	19.0 (4.2)	18.7 (4.4)	19.3 (4.4)	19.1 (4.6)	19.2 (4.3)	18.9 (4.5)
Risk 2	0.77 (0.34)	0.77 (0.34)	0.81 (0.32)	0.80 (0.33)	0.79 (0.33)	0.78 (0.34)
Active Choice	0.72 (0.36)	0.69 (0.36)	0.71 (0.36)	0.67 (0.35)	0.71 (0.36)	0.68 (0.36)
# observations	6692	7756	5494	7182	12186	14938

Note. Standard deviations in parenthesis. Active Choice is a binary variable taking the value 1 if individual made an active portfolio investment decision and 0 otherwise.

TABLE III.

RESULTS OF THE ACE MODEL, 99 % CONFIDENCE INTERVALS IN PARENTHESES

			Model 1	Model 2
Separate				
		a^2	0.22** (0.07-0.31)	0.22** (0.08-0.28)
	$Women$	c^2	0.04 (0.00-0.17)	0.00 (0.00-0.10)
		e^2	0.73** (0.68-0-78)	0.78** (0.72-0.83)
		a^2	0.28** (0.15-0.32)	0.24** (0.17-0.29)
	Men	c^2	0.00 (0.00-0.08)	0.00 (0.00-0.02)
		e^2	0.72** (0.68-0.78)	0.76** (0.71-0.82)
	$ln(L)$		-180051.48	-121625.98
Pooled		a^2	0.26** (0.15-0.31)	0.23** (0.17-0.27)
		c^2	0.01 (0.00-0.10)	0.00 (0.00-0.04)
		e^2	0.73**(0.69-0.76)	0.77** (0.74-0.80)
	$ln(L)$		-180056.55	-121636.13

* A is the genetic contribution; C is the common environment contribution; E is the unique environment contribution. Two stars denote statistical significance at the 1 % level, and one star denotes statistical significance at the 5 % level. Dependent variable is Risk 1. The top panel contains results from a model where separate variance components are estimated for women (subscript w) and men (subscript m). The lower panel reports a restricted model where $a_w^2 = a_m^2$, $c_w^2 = c_m^2$ and $e_w^2 = e_m^2$. All models are estimated allowing the mean and the variance to differ by gender. Confidence intervals are constructed using the bootstrap with 1000 draws. Model 1 is the baseline

model without age moderation. Model 2 is the baseline model where the risk measure has been residualized on age.

TABLE IV.

BACKGROUND VARIABLES

	Women		Men		Population		
	MZ	DZ	MZ	DZ	Women	Men	Total
Income	234363	230560	326272	324824	210022	288012	250995
S.D.	111145	107722	216235	292757	-	-	-
Education (years)	12.3	11.9	12.0	11.6	12.25	11.93	12.11
S.D.	2.6	2.7	2.8	2.9	-	-	-
Marital Status	0.52	0.55	0.55	0.56	0.52	0.48	0.50
S.D.	0.50	0.50	0.50	0.50	-	-	-
Age	48.7	51.8	50.1	52.8	46.6	46.5	46.6
S.D.	11.3	10.0	10.9	9.2	-	-	-

Note. Population mean is defined as the average for individuals born 1938 to 1978. Education refers to years of education. Marital status is a variable taking the value 1 if the individual is married. All data is for the year 2005 and population means were computed using data from Statistics Sweden.

TABLE V.

ROBUSTNESS CHECKS OF THE ACE MODEL, 99 % CONFIDENCE INTERVALS IN PARENTHESES

			Dropped	Matched
Separate				
		a^2	0.16** (0.01-0.22	0.15* (0.00-0.29)
	$Women$	c^2	0.01 (0.00-0.11)	0.10 (0.00-0.23)
		e^2	0.83** (0.78-0.88)	0.75 (0.69-0.82)
		a^2	0.23** (0.13-0.28)	0.23* (0.00 -0.32)
	Men	c^2	0.00 (0.00-0.06)	0.03 (0.00-0.17)
		e^2	0.77 (0.72-0.83)	0.74 (0.68-.0.82)
	$ln(L)$		-147933.61	-91029.09
Pooled		a^2	0.20** (0.11-0.23)	0.19** (0.07-0.28)
		c^2	0.00 (0.00-0.06)	0.07 (0.00-0.17)
		e^2	0.80** (0.78-0.84)	0.75** (0.70-0.79)
	$ln(L)$		-147937.36	-91033.66

* A is the genetic contribution; C is the common environment contribution; E is the unique environment contribution. Two stars denote statistical significance at the 1 % level, and one star denotes statistical significance at the 5 % level. In the "Dropped" column, pairs where both twins selected identical portfolios are excluded. In the "Matched" column, we stratified the data by frequency of contact into 15 groups, and for each sex and level of contact we then randomly dropped the required number of either MZ or DZ pairs to make the number of MZ and DZ pairs equal. In this restricted sample, the distribution of frequency of contact is, by construction, virtually the

same in the MZ and DZ groups. The top panel contains results from a model where separate variance components are estimated for women (subscript w) and men (subscript m). The lower panel reports a restricted model where $a_w^2 = a_m^2$, $c_w^2 = c_m^2$ and $e_w^2 = e_m^2$. All models are estimated allowing the mean and the variance to differ by gender. Confidence intervals are constructed using the bootstrap with 1000 draws.

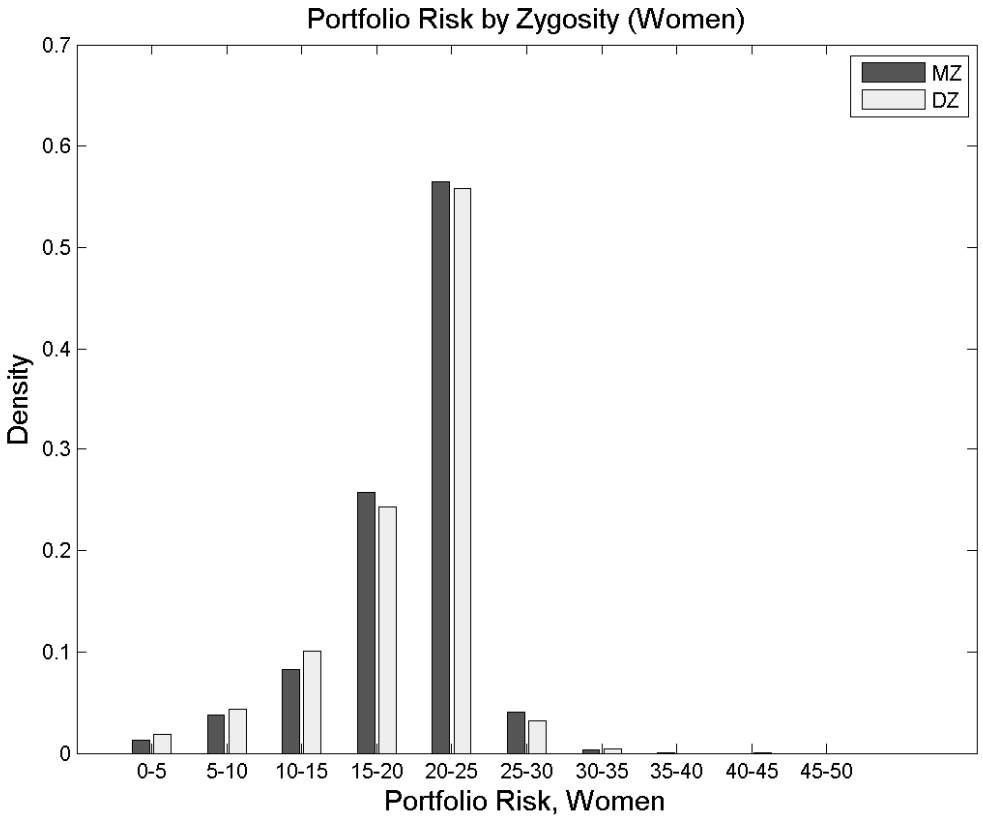

FIGURE 1. Portfolio Risk Distribution in Women.

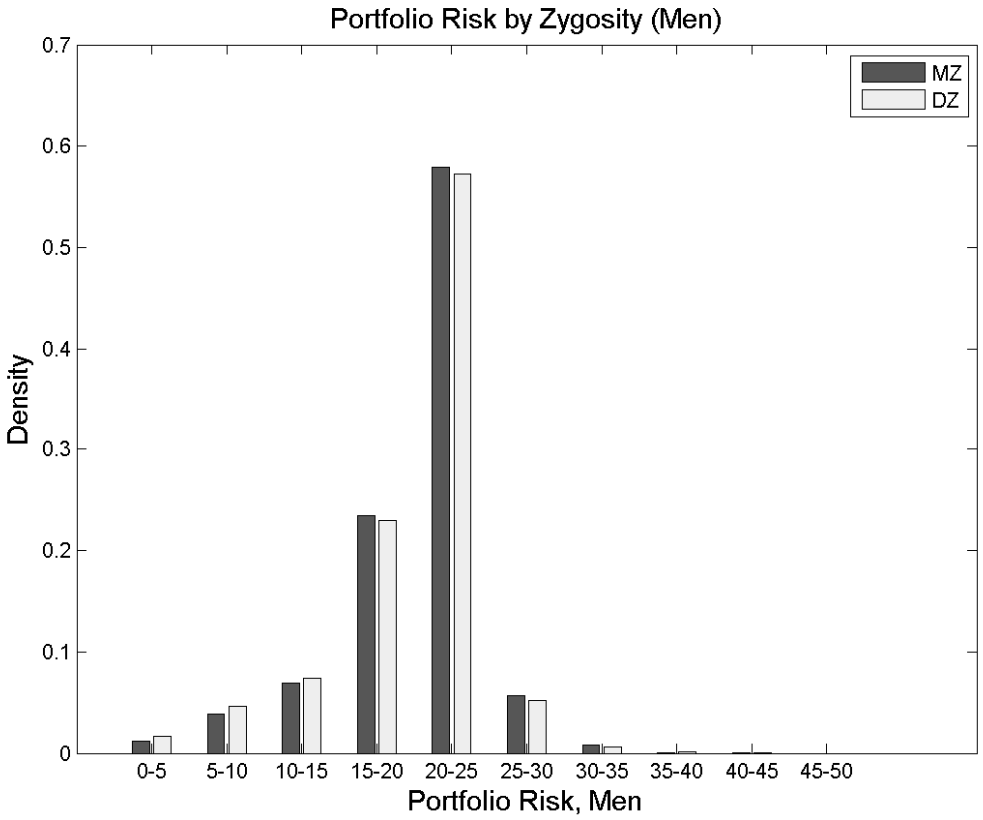

FIGURE 2. Portfolio Risk Distribution in Men.