Genetic Variation in Preferences for Giving and Risk-Taking*

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Abstract

In this paper, we use the classical twin design to provide estimates of genetic and environmental influences on experimentally elicited preferences for risk and giving. Using standard methods from behavior genetics, we find strong prima facie evidence that these preferences are broadly heritable and our estimates suggest that genetic differences explain approximately twenty percent of individual variation. The results thus shed light on an important source of individual variation in preferences, a source which has hitherto been largely neglected in the economics literature.

*This paper has benefited from discussions with Coren Apicella, Samuel Bowles, Terry Burnham, Bryan Caplan, Tore Ellingsen, James Fowler, Moses Ndulu, Matthew Notowidigdo, Niels Rosenquist, Paul Schrimpf, Steven Pinker, Tino Sanandaji, Örjan Sandewall, Vernon Smith and Robert Östling. Thanks to Larry Katz and six anonymous referees for very helpful comments. We thank the Jan Wallander and Tom Hedelius Foundation, the Swedish Research Council, and the Swedish Council for Working Life and Social Research for financial support. The Swedish Twin Registry is supported by grants from the Swedish Research Council, The Ministry for Higher Education and Astra Zeneca. Rozita Broumandi and Camilla Björk at the Swedish Twin Registry responded to a number of queries, for which we are grateful. Patrik Ivert, Niklas Kaunitz and Benjamin Katzeff provided excellent research assistance. We are grateful to a number of colleagues, especially Håkan Jerker Holm and Fredrik Carlsson, for help with subject recruitment outside Stockholm. The paper was completed whilst David Cesarini was visiting the Research Institute of Industrial Economics in Stockholm; he gratefully acknowledges their hospitality.

1 INTRODUCTION

Writing in 1875, the prolific Francis Galton concluded the first scientific inquiry into the behavior of twins by remarking that "There is no escape from the conclusion that nature prevails enormously over nurture" (Galton 1875, p. 576). In fact, Galton was so taken by his results that he continued "My only fear is that my evidence seems to prove too much and may be discredited on that account, as it seems contrary to all experience that nurture should go for so little." Although his methodology would be considered dubious, if not flawed, by modern standards, Galton's work laid the conceptual basis for behavior genetics (Bouchard and Propping 1993; Plomin et al. 2001b), the study of genetic and environmental influences on variation in human behavior. Today ample evidence for the importance of genetic influences ('nature') on variation in human behavioral traits has amassed. However, the debate about the rather nebulous concepts 'nature' and 'nurture' still rages.

In economics, there is a small but growing research field using behavior genetic techniques. The seminal paper is due to Taubman (1976), who employed the twin design to estimate the heritability of earnings for US males. Later papers in this procession, based on either twins or adoptees, include Behrman and Taubman (1989), Plug and Vijverberg (2003), Björklund, Lindahl and Plug (2006), Björklund, Jäntti and Solon (2007) and Sacerdote (2002, 2007). In short, these studies find that both 'nature' and 'nurture' are important determinants of life outcomes and uniformly corroborate the importance of genetic influences on educational attainment and earnings.[1]

Some recent work in economics also focuses on the issue of intergenerational transmission of preferences. Cipriani, Giuliani and Jeanne (2007) report mother-son correlations for contributions in a standard public goods game, and find no significant associations, interpreting this as evidence that peer-effects influence contributions. Dohmen et al. (2006), on the other hand, use survey evidence on attitudinal questions and find modest intergenerational correlations in self-reported trust and risk attitudes. Naturally, these papers suffer from the limitation that it is impossible to separately identify genetic (parents passing on genes for a certain trait to their biological children) and cultural transmission.

In this paper, we move beyond the computation of intergenerational correlations and offer a direct test of the hypothesis that economic preferences are under genetic influence. We elicit preferences experimentally with a subject pool of twins recruited from the population based Swedish Twin Registry. The virtue of this approach is that by comparing monozygotic (MZ) twins, who share the same set of genes, to dizygotic (DZ) twins, whose genes are imperfectly correlated, we can estimate the proportion of variance in experimental behavior due to genetic, shared and unique environmental effects. The measures of economic preferences that we use are based on de facto observed experimental behavior under controlled circumstances with financial incentives attached to performance. For risk-taking, we also present some supplementary survey-based evidence derived from hypothetical questions that have been behaviorally validated (Dohmen et al. 2005; Dohmen et al. 2006).

This paper is the first to use the twin methodology to study experimentally elicited risk preferences and giving behavior in a dictator game. Outside economics, two papers have used the twin methodology to shed light on individual variation in the ultimatum game (Wallace et al. 2007) and the trust game (Cesarini et al. 2008). Two other previous papers used twins as a subject pool (Segal and Hershberger 1999; Loh and Elliott 1998), but the experiments therein were designed to test whether cooperation varied by genetic relatedness, as predicted by inclusive fitness theory (Hamilton 1964). Therefore, twins played against their co-twin, and consequently it is not possible to estimate heritability from these studies.

We find strong evidence that preferences for risk-taking and giving are broadly heritable. Our point estimates from the best fitting models suggest that approximately twenty percent of individual variation can be explained by genetic differences. Furthermore, our results suggest only a modest role for common environment as a source of variation. We argue that the significance of these results extends well beyond documenting an important, but, hitherto largely ignored, source of preference heterogeneity. For example, although it is widely accepted that parent-offspring correlations in isolation cannot be used to discriminate between theories of genetic and cultural transmission, much economic research is carried out under the presumption that genetic transmission is small enough that it can be safely ignored. Such an assumption is not consistent with our findings.

Importantly, the estimates we report are in line with the behavior genetics literature, where survey based studies have documented substantial genetic influences on variation in economically relevant abilities, preferences and behaviors such as intelligence (Bouchard et al. 1990), personality (Jang, Livesley and Vernon 1996), addiction (True et al. 1997), pro-sociality (Rushton et al. 1986; Rushton 2004), sensation seeking (Stoel, De Geus and Boomsma 2006), religiosity (Bouchard et al. 1999; Kirk et al. 1999; Koenig et al. 2005), political preferences (Alford, Funk and Hibbing 2005) and political participation (Fowler, Dawes and Baker 2008). The remainder of this paper is structured as follows: in sections II and III, we describe the method and the experiments used in detail; in section IV, we report the results and in section V, we discuss our findings. Section VI concludes.

2 DATA COLLECTION

2.1 SUBJECT RECRUITMENT

The study was undertaken in collaboration with the Swedish Twin Registry at Karolinska Institutet.[2] The registry, which is the largest twin registry in the world, has been described in detail elsewhere (Lichtenstein et al. 2006). All of our invitees were same-sex twin pairs that had previously participated in the web-based survey STAGE, an acronym for 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 61 %. Its primary purpose was to study environmental and genetic influences on a number diseases (Lichtenstein et al. 2006), but it also contains self-reported data on marital, employment and fertility status as well as information on the frequency of twin contact. To allow for further examination of the effects of our methods of recruitment on the representativeness of our sample, we also merged the STAGE cohort to a specially requested dataset of socioeconomic and demographic variables compiled by Statistics Sweden.

In a first recruitment effort, during the summer and fall of 2006, a total of 658 twins (71 DZ and 258 MZ pairs) participated in the Swedish cities of Stockholm, Gothenburg, Uppsala, Malmö, Lund, Linköping, Norrköping, Helsingborg, Örebro, Västerås and Kristianstad. Due to the relatively small sample of DZ twins, a second round of data collection took place in February 2008. Both MZ and DZ twins were invited to participate, but DZ twins were pursued somewhat more vigorously, with personalized invitations and reminders sent to those who did not respond. This recruitment effort was successful in augmenting the sample size of DZ twins, and the complete dataset comprises 920 twins, 141 DZ pairs and 319 MZ pairs. A vast majority of subjects, approximately 80 %, are female. For the second data collection round, twins were recruited in the cities of Stockholm, Gothenburg, Uppsala, Malmö, Lund, Helsingborg, Örebro, Växjö, Västerås, Jönköping, Borlänge and Umeå. In all of the experimental sessions a condition for participation was that both twins in a pair be able to attend the same session. Moreover, invitations were only extended to twins who were both domiciled in the same city or its surrounding areas. Zygosity was resolved by questionnaire items which have been shown to have a reliability of somewhere between 95 and 98 % (Lichtenstein et al. 2006).

2.2 EXPERIMENTAL PROCEDURES

When subjects arrived to an experimental session they were seated apart and given general instructions orally. They were asked not to talk to one another during the experiment and to alert the experimenter if they had any questions (questions were rare and were answered in private). Subjects were also told about the strong norm against deception in experimental economics. After having filled out a form with information for the administration of payments, subjects were given instructions for the first experiment (the modified dictator game, see below). There were no time constraints, so when all participants finished making their decisions, the next set of instructions were handed out. Subjects participated in a total of five different experiments. The experiment phase was followed by a short questionnaire with survey questions, a personality test and a test of cognitive ability. On average, experimental sessions lasted a little more than an hour and average earnings were SEK 325 (exchange rate; $1 is about SEK 6).

2.3 GIVING

We used a modified dictator game to measure preferences for giving ('altruism').[3] In a standard dictator game (Forsythe et al. 1994) a subject decides how to split a sum of money between herself and another person (see Camerer [2003] for an overview of dictator game results). A variant of this approach first used by Eckel and Grossman (1996) is that the subject decides how to allocate a sum of money between herself and a charity. As donations to charity may be more strongly related to empathy and altruism when compared to donations in the standard dictator game, we implemented this approach. Fong (2007) has shown that empathy is a more important motivation for dictator game giving when recipients are perceived to be in great need, in their case welfare recipients). In the present study subjects decided how to allocate SEK 100 (about $15) between themselves and a charity called 'Stadsmissionen'. Stadsmissionen's work is predominantly focused on helping the homeless in Sweden. All subjects responded to the dictator game question and are included in the analysis below (319 MZ pairs and 141 DZ pairs).

2.4 RISK-TAKING

To measure risk aversion subjects were presented with six choices, each between a certain payoff and a 50/50 gamble for SEK 100 (about $15). The certain payoffs were set to SEK 20, 30, 40, 50, 60, or 80. After subjects had made their six choices, one of these was randomly chosen for payoff by rolling a die. The gamble was resolved with a coin toss in front of the participants. The measure of risk aversion determines seven intervals for the certainty equivalent of the gamble. A similar question has been used by Holt and Laury (2002). Nineteen subjects provided inconsistent responses (2 % of the total sample) and these were dropped (leaving 307 MZ pairs and 135 DZ pairs for the analysis).[4] We refer to this measure as risk aversion and it is our primary measure of risk preferences.

We supplement this first measure of risk preferences with two hypothetical questions designed to measure risk attitudes. The first question, which we denote risk investment, asks the subjects to assume that they have won SEK 1 million on a lottery and that they are then given the opportunity to invest some of this money in a risky asset with an equal probability of doubling the investment or losing half the investment. Subjects can then choose between six different levels of investments: SEK 0, 200,000, 400,000, 600,000, 800,000 or 1 million. This question is similar to the question with real monetary payoffs, but involves much larger (although hypothetical) stakes. The second question, risk assessment, measures general risk attitudes on a 0–10 scale, where 0 is complete unwillingness to take risks and 10 is complete willingness to take risks. This scale question measures general risk attitudes rather than monetary risk attitudes. Dohmen et al. (2005) showed that all of these three measures of risk attitudes are significantly related to each other, and established the behavioral validity of the two hypothetical questions with respect to real risk-taking.

3 TWIN METHODOLOGY

Comparing the behavior of identical and nonidentical twins is a form of quasi-controlled experiment. MZ and DZ twins differ in their genetic relatedness. If a trait is heritable, then it must be the case that the correlation in MZ twins is higher than the correlation in DZ twins. We start by examining the MZ and DZ correlations. Such an examination serves two purposes. A number of authors (Loehlin 1965; Goldberger 1977, 1979), have noted that moving from a crude comparison of correlations to a full-fledged variance decomposition requires making some strong independence and functional form assumptions. A first purpose is therefore to examine whether or not a significant difference in correlations exists. This serves as a diagnostic of whether the traits in question are under genetic influences. Second, as explained below, the workhorse models in behavior genetics do imply certain restrictions on the MZ and DZ correlations. Correlations that fall significantly outside the space of permissible correlations are therefore an indication of model misspecification and the raw correlations can be used to test for such misspecification. To explain why, it is necessary to introduce some basic concepts from behavior genetics (See chapter 3 in Neale and Maes [2004]). By phenotype, we simply mean the observed outcome variable. The location of a gene on a chromosome is known as a locus. Alleles are the alternative forms of a gene that may occupy the same locus on a chromosome. Finally, the genotype of an individual is the alleles he or she has at a locus. Suppose that the phenotype of twin $j \in \{1, 2\}$ in family i can be written as the sum of four independent influences,

$$\chi_{ij} = C_{ij} + E_{ij} + A_{ij} + D_{ij}, \tag{1}$$

where C_{ij} is the common environmental factor, E_{ij} is the individually-experienced unique environment factor, A_{ij} is an additive genetic factor and D_{ij} is a dominance factor. Common environmental influences are defined as those influences shared by both twins, for example the home environment, so that $C_{i1} = C_{i2}$. Unique environmental influences, by contrast, are defined as environmental experiences idiosyncratic to each twin.

Behavior geneticists distinguish between additive genetic effects and dominance effects. For an intuitive illustration of the difference, consider the simple case where there are two possible alleles, a_1 and a_2, so that each individual, getting one allele from each parent, has genotype (a_1, a_1), (a_1, a_2), or (a_2, a_2). Dominance is then present whenever the effect of having genotype (a_1, a_2) is not equal to the mean effect of genotypes (a_1, a_1) and (a_2, a_2). In other words, dominance can be thought of as an interaction effect.

Since the influences are assumed to be independent, the model predicts that the covariance in MZ twins is equal to,

$$COV_{MZ} = \sigma_A^2 + \sigma_D^2 + \sigma_C^2, \tag{2}$$

because identical twins share the same genes and were reared together. The phenotypic covariance between DZ twins is derived in Mather and Jinks (1977) as,

$$COV_{DZ} = \frac{1}{2}\sigma_A^2 + \frac{1}{4}\sigma_D^2 + \sigma_C^2. \tag{3}$$

The coefficients of genetic relatedness for DZ twins in equation (3) thus imply that DZ twins share half the additive genetic effects and a quarter of the dominance effects.

Notice that parameters of this model are not identified with only twin data, since we have one equation less than the number of parameters to be estimated. This ambiguity is typically resolved in twin research by assuming that all gene action is additive, so that $\sigma_D^2 = 0$. Behavior geneticists distinguish between broad heritability, defined as $\frac{\sigma_A^2+\sigma_D^2}{\sigma_A^2+\sigma_D^2+\sigma_C^2+\sigma_E^2}$ and narrow heritability, defined simply as $\frac{\sigma_A^2.}{\sigma_A^2+\sigma_D^2+\sigma_C^2+\sigma_E^2}$. The identifying restriction that σ_D^2 equals zero can be tested by examining if the ρ_{DZ} is at least half of ρ_{MZ}, and the greatest difference in correlation allowed by the model arises when $\sigma_C^2 = 0$ and $\sigma_A^2 = 0$, in which case ρ_{MZ} is four times greater than ρ_{DZ}.

In our empirical analysis, we start by comparing the correlations of MZ and DZ twins using the bootstrap. Letting N_{MZ} be the number of complete MZ pairs, we draw N_{MZ} pairs with replacement 1000 times and calculate both parametric and 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 the number of negative entries in the vector divided by 1000. The use of a one-sided test is theoretically justified in our case since the notion that the DZ correlation could be greater than the MZ correlation is not a particularly interesting alternative hypothesis. We also use the same bootstrap technique to test the hypothesis that the DZ correlation is at least half as large as the MZ correlation. The result of the latter exercise will inform our choice of identifying restrictions.

For our two main outcome variables, we estimate mixed-effects Bayesian ACE models[5]. We report results treating outcome variables as continuous as well as ordinal. Using the same notation as previously, the model is written as,

$$y^*_{ij} = \chi_{ij} \tag{4}$$

where χ_{ij} is the sum of genetic, shared environment and unshared environment random effects. For MZ twins the latent variable is the sum of three random effects:

$$\chi^{MZ}_{ij} = A_i + C_i + E_{ij}, \tag{5}$$

where A_i is the family genetic factor, C_i is the family shared environment factor, E_{ij} is the individually-experienced unshared environment factor. For DZ twins the latent variable is a function of four random effects variables:

$$\chi^{DZ}_{ij} = A_{1i} + A_{2ij} + C_i + E_{ij}, \tag{6}$$

where A_{1i} is the family genetic factor shared by both twins, A_{2ij} is the individually-inherited genetic factor that is unique to each twin, and C_i and E_{ij} are the same as for MZ twins. In the continuous models, we take the outcome variables in the experiment to be y^*_{ij}. In the ordered models, the outcome variables are instead modeled under the assumption that y^*_{ij} is not directly observed. Instead, the observed variable y_{ij} is assumed to be one of $k + 1$ ordered categories separated by k thresholds which are estimated as part of the model. The three risk measure naturally fall into categories, and hence these categories are used in the analysis. A visual inspection of Figure I shows that the distribution of dictator game responses is roughly trimodal, with peaks at the three focal points: donating the entire endowment, half the endowment, or keeping the entire endowment. Approximately 80 % of responses are in one of those three categories. Consequently we construct an ordinal variable where individuals who donate between 0 and 33 are coded as 0, individuals who donate between 33 and 66 are coded as 1, and individuals who donate more than 66 are coded as 2. We use the variances of the random effects to generate estimates of heritability, common environment, and unique environment. Since the underlying components are not constrained, the estimated proportions can range anywhere from 0 (the component has no effect on variance) to 1 (the component is solely responsible for all observed variance).

Replicating the methods used in this literature, we assume that our unobserved random effects are normally distributed and independent,

$$A \sim N\left(0, \sigma_A^2\right), \tag{7}$$

$$A_1 \sim N\left(0, \sigma_A^2/2\right), \tag{8}$$

$$A_2 \sim N\left(0, \sigma_A^2/2\right), \tag{9}$$

$$C \sim N\left(0, \sigma_C^2\right), \tag{10}$$

$$E \sim N\left(0, \sigma_E^2\right). \tag{11}$$

The variance of A_1, the family genetic effect for DZ twins, is fixed to be half the variance of A, the family genetic effect for MZ twins, reflecting the fact that MZ twins on average share twice as many genes as DZ twins. Moreover, DZ twins are also influenced by individually-specific genes A_2 that are drawn from the same distribution as the shared genes since on average half their genes are shared and half are not. These assumptions about the genetic variance help to distinguish shared genes from the shared environment variable C that is assumed to have the same variance for both MZ and DZ twin families, and the residual unique environment variable E from which a unique draw is made for each individual. The contribution of a variance component is simply estimated as $\frac{\sigma_i^2}{\sigma_E^2+\sigma_A^2+\sigma_C^2}$, where $i \in \{A, C, E\}$.[6]

We estimate three types of models in addition to the ACE model. An AE model accounts for only heritability and common environment, a CE model accounts for only common and unique environment, and an E model accounts for only unique environment. Procedurally, the difference between the ACE and these sub-models is that one or more variances are restricted to equal zero. Estimating submodels allows for testing whether the parameter restriction results in a significant deterioration in fit. For example, in the AE model the random effect for the common environment is not estimated. To compare the fit of ACE, AE, CE, and E models we used the deviance information criterion (DIC), a Bayesian method for model comparison analogous to the Akaike Information Criterion (AIC) in maximum likelihood estimation. Models with smaller DIC are considered to have the best out of sample predictive power (Gelman et al. 2004). The DIC is defined as the sum of deviance ($Dbar$), a measure of model fit, and the effective number of parameters (pD), which captures model complexity.[7]

In our Markov Chain Monte Carlo procedure we use vague, or flat, prior distributions to ensure they do not drive our results. For the thresholds, τ_i, we use a mean-zero normal distribution with variance $1,000,000$ and for the precision parameters associated with σ_A^2, σ_E^2 and σ_C^2 we use a Pareto distribution with shape parameter equal to 1 and scale parameter equal to 0.001 which is the equivalent of putting a uniform $(0, 1000)$ prior on the variances. A Pareto distribution has proven to work well for variance components in genetic models (Burton et al. 1999; Scurrah, Palmer and Burton 2000). In addition, we use convergence diagnostics to make sure that the stationary posterior distribution has been reached. To ensure that the models converged to their target posterior distribution, we began sampling from the joint posterior distribution after convergence was established using the Brooks and Gelman (1998) statistic (values of less than 1.1 on each parameter indicate convergence). For all of the models the "burn-in" period was $100,000$ iterations and the chains were thinned by 100.

4 RESULTS

In Table I we report some background statistics. On average, subjects donated 54 % of their endowment in the dictator game to the charity and the average certainty equivalent in the risky gamble was 52.[8] Results from the first hypothetical question reveal that subjects invest on average 31 % of their endowment. Finally, on a scale from 0 to 10, subjects report an average willingness to take risks of just above 5. Tests of equality for all four variables fail to reject the null hypothesis that the MZ and DZ means are equal at the five percent level. To give an impression of individual variation in responses, in Figure I we plot histograms of the distributions for risk aversion and giving, separately, for DZ and MZ twins. A visual inspection reveals that there is ample variation in responses, and fails to lend much support to the hypothesis that the frequency distributions vary by zygosity. Histograms and scatterplots for the survey based risk measures are provided in Figures A1 and A2 in the online appendix.

In Table II, we report parametric and non-parametric correlations for MZ and DZ twins. Pearson correlations do not differ appreciably from Spearman correlations. These correlations convey a lot of information, and since a purely environmental model cannot account for any differences between MZ and DZ correlations they serve as a preliminary diagnostic of whether the preferences in question are in part under genetic influence. For giving the Spearman correlation is 0.319 for MZ twins and 0.106 for DZ twins, consistent with a genetic effect. Similarly, for risk aversion the Spearman correlation is 0.222 for MZ twins and 0.025 for DZ twins, while for risk investment, the corresponding figures are 0.264 and 0.096. However, for risk assessment the separation is larger, with an MZ correlation of 0.367 and a DZ correlation of -0.034. As the sample size is smaller for DZ twins, these correlations are estimated with less precision, yielding wider confidence intervals. Yet, testing the equality of the correlations using the bootstrap, the one-sided p-value is less than two percent for giving, risk aversion and risk assessment. Though the MZ correlation is higher than the DZ correlations also for risk investment, the hypothetical investment question, the difference is not significant at 5 % (p=0.07). The robust separation of MZ and DZ correlations is illustrated in Figure II, where we plot the response of twin 1 against the response of twin 2, separately for MZ and DZ twins. Hence, the evidence is very compelling that genes do contribute to phenotypic variation in both giving and risk aversion.

We also used the same bootstrapping method to test the null hypothesis that the DZ correlation is at least half the MZ correlation, as implied by the ACE specification. For neither risk aversion (p=0.16), risk investment (p=0.36) nor giving (p=0.30) can we reject the null hypothesis. On the other hand, we can reject the null hypothesis for risk assessment (p=0.02), suggesting that the estimation of an ACE model is inappropriate. Notice that even though we cannot reject the hypothesis at conventional levels of significance in three out of four cases, it is still striking that the estimated DZ correlations are always less than half the MZ correlations.

In what follows, we restrict our attention to the results from our experiments with monetary incentives, and results for the supplemental risk measures are reported in Tables A3–A5 in the online appendix. Since we cannot reject the null hypothesis that the DZ correlation is at least half the MZ correlation for our two main experimental measures, we do not depart from the convention of estimating ACE models. In Tables III and IV we present the estimates of the variance components of the ACE-model and its nested submodels. Parameter estimates are similar, regardless of whether the outcome variable is treated as continuous or ordinal. The estimate of genetic influences on giving is 0.22 (0.28) in the most general version of the continuous (ordered) model. Corresponding estimates for risk aversion are 0.14 and 0.16, while the contribution of the common environment is closer to zero, both in our modified dictator game and for risk aversion.

It is interesting to contrast these results to those that have previously been reported for other outcome variables of interest to economists. For example, Björklund, Jäntti and Solon (2005) estimated heritability of earnings in Sweden using multiple sibling types, and obtained heritability estimates for income in the range 10 to 30 %, whereas Taubman's original estimates based on a sample of white US war veterans were slightly higher (Taubman 1976). The estimates for trust and trustworthiness reported in previous papers, though imprecise, are also in the neighborhood of 20% in both US and Swedish data (Cesarini et al. 2008). Generally, the estimated heritabilities for our experimentally elicited preferences are a little lower than the reported broad heritabilities for personality, which tend to be around 50 % (Plomin et al. 2001b), and lower still than the estimates of the heritability of IQ (Neisser et al. 1996). In making the comparison to psychological variables it is, however, important to bear in mind that the reliability of the measurement instruments used by psychometricians in IQ and personality research may be different than the reliability of behavior in economic experiments.

In light of these results, it is not surprising to find that both for giving and risk aversion the diagnostics of model fit repeatedly point to the AE model as the most appropriate. Setting C to equal zero is potentially a drastic step, but is consistent with the fairly low DZ correlations that we observe. When the AE submodel is estimated, the estimates of A for giving are 0.31 (0.39) in the continuous (ordered) models. The corresponding figure for risk aversion is 0.21 (0.25). We also report the results from CE and E models. CE models always have fit diagnostics worse than the AE and ACE models. Not surprisingly, the E model fits the data very poorly.

4.1 Equal Environment Assumption

Critics of the classical twin design cite a number of alleged failures of the equal environment assumption, including that MZ twins are more likely to interact, and that parents, on average, give MZ twins more similar treatment (Pam et al. 1996). Indeed, Björklund, Jäntti and Solon (2005) have shown, using a dataset with nine different sibling types, that estimates of the variance components in income do change substantially when the equal environment assumption is relaxed. In the context of research on personality and IQ, the evidence is, however, fairly convincing that any bias that arises from the equal environment assumption is not of first order. Most importantly, for measures of personality and cognitive ability, studies of MZ and DZ twins 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, this suggests that it is unlikely that the assumption is a major source of bias. Second, although it is true that MZ twins report a higher frequency of contact with one another than DZ twins, twin similarity has been shown to cause greater contact rather than vice versa (Posner et al. 1996). Other studies have failed to find a significant relationship between similarity and contact. For example, one large study found that the frequency of contact is not correlated with the similarity in social attitudes (Martin et al. 1986). Third, the claim that the greater similarity of MZ twins is due to more uniform parental influences rests on fairly weak empirical ground. Measures of the degree of similarity in parental treatment turn out to not be correlated with similarity in IQ or other personality measures (Bouchard et al. 1990). Also, in the relatively rare cases where parents miscategorize their twins as MZ instead of DZ (or the converse), differences in cognitive ability and personality persist (Bouchard and McGue, 2003). Finally, we note that our estimated Cs are very low, and it would appear that the Bayesian estimator, if anything, overstates the importance of shared environment compared to other standard estimators.[9]

4.2 Measurement Error

In the simplest case where the studied preference is observed with mean zero random error, we can think of the unique environment component as being comprised of two terms, $E_{ij} = E^*_{ij} + \epsilon_{ij}$, where ϵ_{ij} is a mean zero variable with variance σ^2_ϵ, and is i.i.d. across time. Under these assumptions, it is easy to show that the estimates of A and C need to be scaled up by a factor of $\frac{1}{1-\sigma^2_\epsilon}$. For example, under the conservative assumption of a retest correlation of 0.8, this would imply a σ_ϵ^2 of 0.2, and therefore the estimates of A and C would need to be scaled up by 25 %, i.e. to somewhere between 0.18 and 0.41 for A in our ACE models. There is surprisingly little evidence on test-retest stability in economic experiments. One recent paper (Brosig, Riechmann and Weimann 2007) examined the temporal stability of individual behavior in modified dictator and prisoner's dilemma games, and found that individual behavior is unstable across time in a given game. However, the authors used a concept of stability which is not easily mapped to an estimate of σ_ϵ^2. Other papers have estimated error rates from identical responses to items, typically finding reversal rates of the order of 10–20 % (Harless and Camerer 1994; Hey and Orme 1994).

4.3 Representativeness

Compared to most experimental work, our sample is an improvement in terms of representativeness since we draw our subjects from a population-based registry and not a pool of college students. Yet, it is important to establish the "selectivity" of our sample. In particular, three questions arise. First, are the MZ and DZ twins who agree to participate drawn from similar environments? Second, to what extent does our method of sampling lead to overrecruitment of subjects with certain characteristics? If any such characteristics are associated with heritability, then estimates of variance components will be biased. Third, in light of the fairly skewed ratio of MZ twins to DZ twins in our sample, are there any reasons to believe that this has affected our estimates?

A basic assumption of the ACE model is that MZ twins and DZ twins are drawn from the same environment. We have already demonstrated that in terms of experimental outcomes, the MZ and DZ distributions appear to be the same. To further investigate this hypothesis, we conducted a battery of tests for equality on background variables including gender, years of education, employment status, health, income and marital status. With the exception of age, we did not find any significant differences between the MZ and DZ samples. The results are reported in Table V.

Second, it is possible that the twins who participated are not representative of the population as a whole. Like most twin studies (Lykken, McGue and Tellegen 1986), our method of recruitment led to an oversampling of women and of MZ twins. Comparing our participants to the STAGE cohort as a whole on a number of background variables, we find few economically interesting differences. These results are also reported in the online appendix.

A comparison to the entire STAGE cohort is only an imperfect measure of representativeness, however, since STAGE respondents are also a self-selected group. We have therefore merged our experimental data to information on educational attainment, marriage status and income from Statistics Sweden, and can thus further examine how our sample compares to the population mean for the cohort born 1959 to 1985. The population marriage rate for women is 36 % and 29 % for men. This is slightly higher than what we observe in our experimental sample. For income, the population averages are close to those of our participants. On average men earn 247,000 SEK, while our male subjects earn 244,000 SEK. For women the corresponding figures are 181,000 and 197,000. Finally, we find that the average years of education in the cohort as a whole is 12.09 for men, and 12.49 for women, which is slightly more than one year less than the average for our experimental sample.

The upshot of this discussion is that our method of sampling leads to mild overrecruitment of subjects who are younger than average, less likely to be married and have fewer children on average. There is also modest overrecruitment of subjects with better than average educational attainment. Is this above average educational attainment of our subjects a source for concern? For instance, it has been suggested that the heritability of intelligence might be moderated by social stratum (Turkheimer et al. 2003), at least in children, and a similar argument might apply to the effect of educational attainment on our outcome variables. To investigate this, we modify the continuous version of our baseline model to allow for interaction between A and years of education.[10] The fit of the new model is slightly better for risk aversion and slightly worse for the other three variables, suggesting the interaction between A and education should not be included. For risk aversion heritability increased somewhat, to 0.21 (95% CI 0.02, 0.39), compared to the baseline model.[11]

Finally, there is a third, more subtle way, in which recruitment bias may be affecting our estimates. A plausible explanation for the overrecruitment of MZ twins is that since MZ twins are in more frequent contact with each other, it is easier for them to coordinate on a date and time. The concern here is that coordination costs, or willingness to participate more generally, might be associated with behavioral similarity. If so, this will inflate correlations, leading to an upward bias in the estimates of A and C. If this form of selection is more severe for MZ or DZ twins, it will also bias the estimates of the relative importance of common environmental and genetic influences. A reasonable proxy variable for costs of coordination is the frequency of contact between twins. Self-reported data on frequency of contact is available in STAGE.[12] When we compare twins who took part in our study to those who did not, there is a practically and statistically significant difference in the anticipated direction. MZ twins who participated in the study report a frequency of contact of 260 interactions per year, whereas those who did not participate report 234 interactions per year. The corresponding figure for DZ twins are 199 and 155. These differences are highly significant. In other words, frequency of contact is a robust predictor of participation. The crucial question, however, is whether frequency of contact predicts behavioral similarity. To test this, we regress the absolute value of the within-pair difference in giving and the three measures of risk on the average self-reported frequency of contact. Controlling for zygosity, the coefficient on frequency of contact is never significant. In other words, a reasonable proxy variable for "costs of coordination" does not seem to be related with behavioral similarity.

A second robustness test is to take variables that are available for the STAGE cohort in its entirety and ask if there are any systematic differences between subjectes who participated in our experiments and those who did not in terms of correlations. If correlations in health, income, years of education and the numerous other variables we investigate are consistently higher in the experimental sample, this would then suggest that these are a self-selected group with greater concordance in general. The results from this exercise are reported in

Table A2 of the online appendix of this paper. There is no tendency for the patterns of correlations to differ between the two groups.

4.4 Genetic Non-Additivity

The models we use – like most behavior genetic models – assume that genes influence a trait in an additive manner. That is to say, the genetic effect is simply the sum of all individual effects. This is by far the most common way to achieve identification. It has long been known that the twin model suffers from parameter indeterminacy when, for example, dominance effects are present because the number of parameters to be estimated exceeds the number independetly informative equations (Keller and Coventry 2005). The fact that our DZ correlations are less than half of the MZ correlations could be the result of sampling variation. But it could also be an indication that there is some non-additive genetic variation present. For one of our risk measures, risk assessment, we are in fact able to reject the hypothesis that the DZ correlation is at least half the MZ correlation. In Table A5 of the online appendix to this paper, we report the results of an ADE model, and show that this model fits the data better, as judged by the DIC criterion.

A more rigorous way to test for non-additivity would be to extend the dataset to include also sibling, parent-child, or even cousin data. Though our data does not contain such information, Coventry and Keller (2005) recently completed a major review of all published parameter estimates using the extended family design compared to classical twin design estimates derived from the same data. The authors report that the estimates of broad heritability in twin studies are fairly accurate. However, the classical twin design overestimates the importance of additive genetic variation and underestimates the importance of non-additive genetic variation. Evidence from studies of adoptees point in the same direction. In a recent metastudy by Loehlin (2005), the author reports average correlations of 0.13 for personality and 0.26 for attitudes in families with children reared by their biological parents. However, 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). Since only additive genetic variance is transmissible across generations (Fisher 1930), doubling the parent-child correlation produces an upper bound on the estimate of narrow heritability. The fact that this upper bound is lower than estimates derived from twin studies reinforces the point that there is probably non-additive variation in personality and attitudes. The low DZ correlations we observe suggest that a similar situation obtains for economic preferences.

We thus concur with the conclusion in Coventry and Keller (2005), namely that the estimates from the classical twin design should not be interpreted literally, but are nevertheless very useful because they produce reasonably accurate estimates of broad heritability, and hence of genes as a source of phenotypic variation.

5 DISCUSSION

In this paper, we have used standard behavior genetic techniques to decompose variation in preferences for giving and risk-taking into environmental and genetic components. We document a significant genetic effect on risk taking and giving, with genes explaining approximately 20 % of phenotypic variation in the best fitting models. The estimated effect of common environment, by contrast, is smaller. Though these results are clearly in line with the behavior genetic literature (Turkheimer 2000), the implication of these findings in the context of modern economics merit further comment.

In particular, it is important to exercise great care in interpreting the estimates of variance components. Contrary to what is sometimes supposed, they are estimates of the proportion of variance explained and thus do not shed any direct light on the determinants of average phenotype. 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 low, or zero, heritability. The same argument is true for common environment. A low estimated C could simply mean that there is little variation in how parents culturally transmit preferences or values to their children. This caveat is especially important to bear in mind when interpreting heritability estimates from a study population such as ours, where it seems plausible to assume that environmental variation between families is modest.

Like any other descriptive statistic, a heritability estimate is specific to the population for which it is estimated, and, though our findings are probably informative about heritability in other modern Western societies, we caution against further extrapolation. Variation in our study population is in all likelihood small relative to cross–country differences or historial environmental differences that could potentially generate greater variation in risk preferences and giving. The perhaps most striking and intuitively illustration of this point comes from the study of income, which is moderately heritable in Sweden as well as in the US (Björklund, Jäntti and Solon 2005; Taubman 1976). In recent centuries incomes have increased manifold, and even today an individual's country of origin is by far the most important determinant of that individual's income (Sala–i–Martin 2006). In other words, a heritability statistic says little about the malleability of a trait with respect to environmental interventions (Goldberger 1979).

Caution should also be exercised in interpreting our estimate of unique environment (E) since it is not possible to separately identify unique environment and measurement error without knowledge of test-retest correlations (Plomin and Daniels 1987; Plomin et al. 2001a). This is because if there is noise in the elicitation of preferences, such noise will be subsumed under the estimate of unique environmental effects.[13] Further, a number of important sources of unique environmental effects, such as accidents, are non-systematic in nature. The observation that the human genome could not possibly specify every synaptic connection in the brain and that random events could lead to different developmental outcomes, even in genetically identical individuals, falls into this category (Molenaar, Boomsma and Dolan 1993; Jensen 1997).

Economists have traditionally expressed agnosticism about the causal mechanisms behind individual differences in preferences. While choosing to overlook genetic explanations is often well motivated on the grounds of parsimony, especially in studies taking a historical or geographical perspective, our findings combined with the pre-existing behavior genetics literature uncover a unique and potentially important source of preference heterogeneity. Despite ample experimental evidence the origins of individual behavioral variation in economic games have thus far remained elusive, and many attempts to find theoretically appealing and empirically stable correlates to preferences elicited experimentally have yielded contradictory results (Camerer 2003). If preferences are indeed under moderate genetic influences any attempt to understand heterogeneity in preferences without taking this into account will be incomplete.

Recently, much interest has been directed toward finding biological or neurological correlates to experimental behavior. Of course, this does not necessarily imply neither causality nor a genetically mediated association. However, the fact that many of the biological variables with known associations to individual differences in strategies or preferences are strongly heritable does lend some support, if only circumstantial, to our findings. For instance, financial risk-taking has been shown to vary over the menstrual cycle in women (Bröder and Hohmann, 2003; Chen, Katuscak and Ozdenoren 2005), and correlates both with facial masculinity and circulating testosterone levels in men (Apicella et al. 2008). A number of imaging studies have also explored the neural correlates of both giving and financial risk-taking. One study found activation in the striatum both on receiving money and donating to charity (Moll et al. 2006). Another study found similar activation patterns and demonstrated enhanced activation when the charitable donation was voluntary (Harbaugh, Mayr and Burghart 2007). In the context of financial risk-taking, Kuhnen and Knutson (2005) demonstrayed that risk-seeking is associated with activation in the nucleus accumbens, whereas risk-aversion is associated with activation in the insula. In general, brain structure is under strong genetic influence, though there are substantial regional differences in heritability (Thompson et al. 2001; Toga and Thompson 2005). The same is true for hormone levels (Harris, Vernon and Boomsma 1998; Bartels et al. 2003).

6 CONCLUSION

In this paper, we have presented an empirical investigation into the relative contributions of individual differences in genes and environment to observed variation in economic preferences for risk and giving. Notwithstanding the fact that all twin siblings are of the same age and were raised together in the same family, the genetically identical MZ twins still exhibit much greater similarity in their preferences for risk and giving than do DZ twins. While our results do not allow us to be as assertive as Sir Francis Galton, they do suggest that humans are endowed with genetic variation in their proclivity to donate money to charity and to take risks. By now there is a plethora of studies exploring the sources of individual variation in economic experiments and games, yet up until recently considerations of genetic influences have remained relatively absent. Here we have argued that this failure to consider genes obscures an important source of preference heterogeneity. Ultimately, we hope that a better understanding of the underlying individual genetic heterogeneity[14] in economic preferences, and the adaptive pressures under which these preferences evolved will lead to a more comprehensive economic science that can bridge some of the unexplained gaps between empirical data and economic theory (Burnham 1997; Cosmides and Tooby 1994).

Finally, our findings suggest a number of directions for future research. In recent years we have witnessed rapid advancement in the field of molecular genetics, including the initial tentative steps toward uncovering the complex genetic architecture underlying variation in individual personality and preferences. In fact, we are aware of one paper which has already uncovered a polymorphism on the AVPR1a gene that is associated with generosity in the dictator game (Knafo et al. 2008). The identification of specific genes, or more likely combinations of genes, associated with particular traits holds promise for economic research. Most importantly, as noted by Benjamin et al. (2007), it will allow for the study of interactions between genotypes and policies to better predict the consequences of policy on individuals. A second direction for future research is to look beyond the laboratory and instead consider field proxies for the underlying preferences. There are well known issues associated with the generalizability of laboratory findings (Levitt and List 2007), and documenting similar genetic influences in the field therefore ought to be a priority. A third, and perhaps most natural, direction is to try to disentangle additive and non-additive genetic variation. We anticipate that studies employing the extended family design will shed more light on this issue. The fairly low DZ correlations we observe provide some tentative, but far from conclusive, evidence for non-additivity.

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Notes

[1]For an extensive collection of essays on the intergenerational transmission of economic opportunity, see the volume edited by Bowles, Gintis and Osborne Groves (2005).

[2]The study and subject recruitment was approved by the Ethics Committee for Medical Research in Stockholm.

[3]Independently, Bardsley (2007) and List (2007) have shown that augmenting the choice set of the dictator to allow him or her to take money from the partner dramatically reduces generosity. This suggests that people's behavior in the standard dictator game is sensitive to cues about social norms in experimental settings. Regardless of one's favored interpretation of giving in dictator games, we will provide evidence suggesting that such giving is heritable.

[4]An inconsistent response is one in which the certainty equivalent is not uniquely defined, i.e. an individual that chose SEK 20 rather than the gamble in the first question and then chooses the gamble rather than SEK 30 in the second question. Such behavior is a strong indication that the subject has either misunderstood the question, or has failed to take it seriously.

[5]Researchers have increasingly used Bayesian methods, implemented using Markov Chain Monte Carlo (MCMC) algorithms, to estimate the variance components in ACE models. The likelihood functions in genetic models often present computational challenges for maximum likelihood approaches because they contain high-dimension integrals that cannot be evaluated in closed form and thus must be evaluated numerically. For a detailed discussion of Bayesian ACE models, we refer to van den Berg, Beem, and Boomsma (2006).

[6]If we tried to estimate all three components of variance simultaneously in the ordered model, it would not be identified, so we fix the variance of the unshared environment σ_E^2 to be one.

[7]Letting θ be the parameter vector, y the data, p the likelihood function, and $f(y)$ a standardizing term which is a function of the data alone, the deviance is defined as,

$$D(\theta) = -2\ln\left(p\left(y\,|\theta\,\right)\right) + 2\ln f\left(y\right).$$

Then $Dbar$ is defined as,

$$Dbar = E_\theta\left(D\left(\theta\right)\right),$$

and pD is defined as,

$$pD = Dbar - D\left(\bar{\theta}\right),$$

where $\bar{\theta}$ is the expectation of θ. The deviance information criterion can then be calculated as,

$$DIC = pD + Dbar$$

For further details, see Spiegelhalter et al. (2002).

[8]To facilitate interpretation, in Table I we define the certainty equivalent as the midpoint between the lowest sure amount that the subject is willing to accept and the category immediately below. For example, a subject chooses the gambles at 20, 30 and 40 and then prefers 50 SEK with certainty, is assigned a certainty equivalent of 45.

[9]It is clear by inspection that a method of moment estimator would produce non-sensical negative estimates of common environment. Estimating continuous ACE models using maximum-likelihood in MPLUS (Muthén and Muthén 2006), and bootstrapping the standard errors, estimated Cs are always equal to zero, and the estimated heritabilities are 0.21 for risk aversion, 0.31 for giving, 0.29 for risk investment and 0.35 for risk assessment. All estimates of A are significant at the five percent level.

[10]This model is $\chi_{ij}^{MZ} = A_i + \beta * A_i * Education_{ij} + C_i + E_{ij}$ for MZ twins and $\chi_{ij}^{DZ} = A_{1i} + A_{2ij} + \beta * (A_{1i} + A_{2ij}) * Education_{ij} + C_i + E_{ij}$ for DZ twins.

[11]The DIC for the risk aversion, risk investment, risk assessment, and dictator game interaction models are 7813, 3881, 3698, and 4919 respectively. New baseline models were run to account for the fact that the interaction models were based on fewer observations due to missing values for the years of education variable. The baseline DICs are 7824, 3872, 3695, and 4915.

[12]We construct the frequency of contact variable as follows. Subjects who report at least one interaction (by e-mail, telephone or letter) per day are assigned a value of 365. Subjects who report less than one interaction per day are simply assigned a value equal to the number of interactions per year. Interestingly, frequency of contact also provides a falsification test of the basic twin model. Since this variable is the same for both twins in a pair, it cannot possibly be heritable. A higher MZ correlation than DZ correlation would then suggest that measurement errors are more correlated in MZ twins. Fortunately, this turns out not to be the case. In our experimental sample, the MZ correlation is 0.76 and the DZ correlation is 0.71. In STAGE as a whole, the correlations are 0.77 and 0.75.

[13]This result also has implications for the genome-wide association studies that are currently underway, examining genetic variation across the human genome and behavior in experimental games. Noise in the elicitation in, for instance, social preferences is likely to frustrate these efforts. Multiple measurement would be one way of dealing with the problem.

[14]Genetic variation can be maintained in equilibrium for a number of reasons. For a discussion of this difficult subject in the context of personality differences, see two recent papers by Dall, Houston and McNamara (2004) and Penke, Denissen and Miller (2007).

8 Tables and Figures

Table I.
Experimental Behavior

		MZ Twins	DZ Twins	p-value
Giving	Mean	53.60	54.43	0.77
	S.D.	37.27	37.94	
	n	638	282	
Risk Aversion	Mean	52.38	51.88	0.71
	S.D.	18.53	17.80	
	n	625	276	
Risk Investment	Mean	30.25	33.19	0.08
	S.D.	21.22	21.28	
	n	638	279	
Risk Assessment	Mean	4.98	5.25	0.07
	S.D.	1.98	1.96	
	n	636	279	

Notes. The p-value is for the test of the hypothesis that the mean of the MZ and DZ distributions are the same. Standard errors are adjusted to take non-independence into account (Liang and Zeger 1986).

Table II

Parametric and Non-parametric correlations for mz and dz twin pairs. 95% confidence intervals within parentheses.

		MZ twin pairs	DZ twin pairs	p-value of diff.
Giving	Spearman	0.319*** (0.211–0.426)	0.106 (-0.067 – 0.292)	0.015
	Pearson	0.317***(0.208–0.424)	0.099(-0.075 – 0.279)	0.013
	# pairs	319	141	
Risk Aversion	Spearman	0.222*** (0.118–0.341)	0.025 (-0.150 – 0.189)	0.020
	Pearson	0.222*** (0.099–0.342)	0.024 (-0.135 – 0.179)	0.024
	# pairs	307	135	
Risk Investment	Spearman	0.264*** (0.149–0.364)	0.096 (-0.077 – 0.277)	0.066
	Pearson	0.304*** (0.177–0.408)	0.110 (-0.079 – 0.315)	0.057
	# pairs	319	139	
Risk Assessment	Spearman	0.367***(0.266–0.468)	–0.034 (-0.217 – 0.148)	0.001
	Pearson	0.384*** (0.280–0.481)	–0.043 (-0.237 – 0.139)	0.001
	# pairs	317	139	

Notes. ***, **, *=significantly different from zero at 1%, 5%, and 10% level. All results are bootstrapped. P-values are one-sided.

Table III.
Results of the ACE model and its nested submodel for giving. 95% confidence intervals within parentheses.

		Model			
		ACE	AE	CE	E
	A	0.22 (0.05, 0.36)	0.31 (0.21, 0.40)	–	–
Continuous	C	0.09 (0.01, 0.23)	–	0.25 (0.16, 0.33)	–
	E	0.70 (0.60, 0.79)	0.69 (0.60, 0.79)	0.75 (0.67, 0.84)	1.00 (1.00–1.00)
DBar		4719	4706	4783	5043
pD		227.3	234.9	184.8	2.0
DIC		4946	4941	4968	5045
	A	0.28 (0.06, 0.46)	0.39 (0.27, 0.51)	–	–
Ordered	C	0.11 (0.01, 0.30)	–	0.32 (0.21, 0.43)	–
	E	0.61 (0.50, 0.73)	0.61 (0.49, 0.74)	0.68 (0.57, 0.79)	1.00 (1.00–1.00)
DBar		1693	1688	1761	2023
pD		236.0	238.7	189.8	2.0
DIC		1929	1927	1951	2025

Notes. A is the genetic contribution; C is the common environment contribution; E is the unique environment contribution.

DBar: Deviance.

pD: Effective number of parameters.

DIC: Bayesian Deviance Information Criterion.

Table IV.
Results of the ACE model and its nested submodel for Risk Aversion. 95% credible intervals within parentheses.

		Model			
		ACE	AE	CE	E
	A	0.14 (0.02, 0.27)	0.21 (0.11, 0.31)	–	–
Continuous	C	0.07 (0.00, 0.18)	–	0.17 (0.08, 0.26)	–
	E	0.80 (0.69, 0.89)	0.79 (0.70, 0.89)	0.83 (0.74, 0.93)	1.00 (1.00–1.00)
DBar		7713	7707	7752	7914
pD		160.8	163.9	130.6	2.0
DIC		7873	7871	7883	7916
	A	0.16 (0.01, 0.30)	0.25 (0.14, 0.36)	–	–
Ordered	C	0.09 (0.01, 0.22)	–	0.20 (0.10, 0.30)	–
	E	0.75 (0.65, 0.86)	0.75 (0.64, 0.86)	0.80 (0.70, 0.90)	1.00 (1.00–1.00)
DBar		2760	2752	2804	2985
pD		181.4	186.3	149.1	5.9
DIC		2941	2938	2953	2991

Notes. A is the genetic contribution; C is the common environment contribution; E is the unique environment contribution.

DBar: Deviance.

pD: Effective number of parameters.

DIC: Bayesian Deviance Information Criterion.

Table V.
MZ DZ Comparison for Background Variables.

	MZ Twins		DZ Twins			
	Mean	S.D.	Mean	S.D.	p-value	Data Source
Female	0.77	0.42	0.82	0.39	0.24	Multiple
Age	34.30	7.35	35.95	7.81	0.03	Multiple
Education	13.70	2.22	13.63	2.18	0.69	Stat. Sweden
Income	201973	152674	217548	119997	0.19	Stat. Sweden
Employed Full Time	0.54	0.50	0.60	0.49	0.23	STAGE
Unemployed	0.03	0.18	0.04	0.19	0.80	STAGE
Self-Employed	0.04	0.20	0.07	0.25	0.32	STAGE
On Sickleave	0.04	0.19	0.02	0.12	0.10	STAGE
Government Employee	0.40	0.49	0.45	0.50	0.26	STAGE
Cognitive Ability	0.03	0.99	–0.06	1.02	0.30	Exp. Session.
Emotional Stability	–0.04	1.00	0.10	0.99	0.09	Exp. Session.
Agreeableness	0.02	0.98	–0.04	1.04	0.55	Exp. Session.
Extraversion	-0.04	0.98	0.08	1.04	0.16	Exp. Session.
Conscientiousness	–0.02	1.01	0.04	0.98	0.55	Exp. Session.
Health	1.87	0.81	1.88	0.79	0.86	STAGE
Marital Status	0.25	0.43	0.29	0.46	0.26	Stat. Sweden
Number of Children	0.70	0.99	0.76	0.99	0.55	Stat. Sweden

Notes. Education refers to years of education. Income is the sum of wage income, taxable transfers and income from own company for the year 2005 (in SEK). Employment information was gathered when the subject responded to the STAGE questionnaire. Psychological measures were adjusted to have mean 0 and standard deviation 1 for the whole sample. Health is self-reported on a scale from 1 to 5. Marital status is a dummy variable taking the value 1 if the subject is married. Number of children is number of children under 18 living in the respondent's household in the year 2005. The p-value is for the test of the hypothesis that the mean of the MZ and DZ distributions are the same. We utilized adjusted Wald tests for equality taking into account non-independence within twin families (Liang and Zeger 1986).

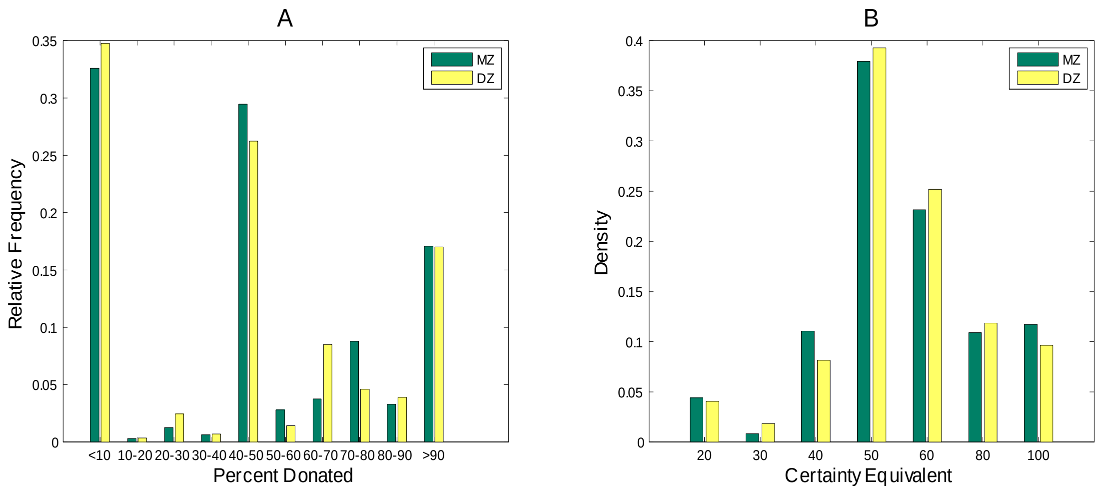

FIGURE I.

Panel A: The distribution of giving (percent donated), by zygosity.
Panel B: The distribution of risk aversion (certainty equivalent), by zygosity.

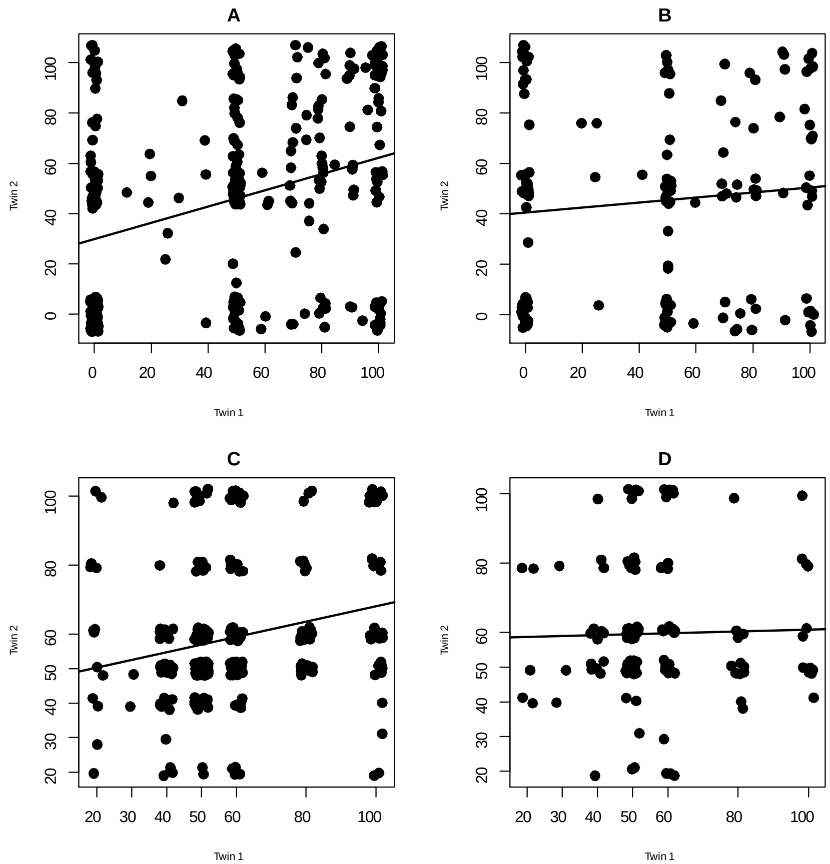

FIGURE II.Scatterplots jittered for expositional clarity.
Panel A. Scatterplot for the dictator game, percent donated, MZ twins.
Panel B. Scatterplot for the dictator game, percent donated, DZ twins.
Panel C. Scatterplot for risk aversion, certainty equivalent, MZ twins.
Panel D. Scatterplot for the risk aversion, certainty equivalent, DZ twins.