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Heritability of different measures of overconfidence

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Abstract

Incorrect estimation of own absolute and relative abilities is common and can have detrimental effects on a person's educational, social, employment, and financial outcomes. It is not yet fully understood from where interpersonal differences in overconfidence emerge. In this paper, we estimate the heritability of two types of overconfidence, overestimation, and overplacement, in a sample of 1120 twins. We find that the genetic heritability of overestimation (overplacement) is about 19% (17%) and that most of the interindividual variation in overconfidence is due to individual-specific environmental factors.

Keywords: Heritability; overconfidence; genetics; twin study

JEL Classification: D91; Z13

1. Introduction

Overconfidence (and underconfidence) is a miscalibrated belief about own absolute or relative performance. It is a pervasive individual trait, with significant implications, and has been explored across various domains, including economics, psychology, and behavioral sciences. Overconfidence may be viewed as beneficial, as it may enhance ambition, morale, and persistence, creating a reinforcing mechanism where inflated confidence increases the likelihood of success (Bénabou & Tirole, 2002). For example, in a controlled laboratory experiment, Reuben et al., (2012) found that men tend to be more overconfident than women, leading to male overrepresentation in leadership positions. Using a British Cohort study, Adamecz-Völgyi and Shure (2022), estimate that overconfidence explains about 5–11% of the gender gap in top job employment for midlife adults.

However, research on overconfidence is not only relevant to understanding gender gaps[1] and overconfidence is not always beneficial. Inflated overconfidence can have a range of serious negative outcomes, such as increased new business entry and failure (Camerer & Lovallo, 1999), stock market bubbles (Glaser & Weber, 2007), ideological extremism (Ortoleva & Snowberg, 2015), wars (Johnson, 2004), and excessive litigation (Neale & Bazerman, 1985). Overconfidence can have perverse effects including poor financial decision-making (Daniel et al., 1998), more myopic investments (Kaplan et al., 2022), and academic complacency (Vancouver et al., 2008). This begs the question, why are some people more overconfident than others?

We focus on the foundations of overconfidence by asking: 'To what extent are interpersonal differences in overconfidence explained by differences in genes and in environment?.' More accurately,

[1]For example, overconfidence can also vary over age (Menkhoff et al., 2013) and occupation (Ilieva et al., 2018).

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and consistent with related studies, we analyze a miscalibrated belief in which positive values denote overconfidence and negative values underconfidence. Only two twin studies estimate the genetic heritability of overconfidence. Cesarini et al., (2009) find that 16%–34% of interpersonal variation in the tendency to overestimate own relative performance (overplacement) is due to differences in genes among adult Swedish twins. Vogt et al. (2022) attribute 18%–28% of interpersonal differences in overestimation (measured as a residual between subjective belief in and objective performance) to genes in a sample of children aged 7–15 years in Texas. The remaining variation in overconfidence in both studies was mainly due to unique personal circumstances.

We extend earlier work by measuring different types of overconfidence in one sample. We consider both overestimation, the overconfidence in own absolute performance, and overplacement, the overconfidence in own relative performance. Overestimation and overplacement are conceptually (and empirically) distinct (Moore & Healy, 2008), with overestimation relying on imperfect information about own performance and overplacement additionally relying on imperfect beliefs about other's performance. We estimate the extent to which these distinct traits are explained by genes and the extent to which their correlation is explained by shared genetic influences. The potential interaction between genes and the common social environment means that the heritability of behavioral traits may vary across social contexts. Existing twin studies provide only two data points: for adult Swedes and for children in Texas. Here, we study a different social context (Australia) and check whether previous findings generalize.

2. Data

We analyze the Australian Twins Economic Preferences Survey (ATEPS) (Kettlewell & Tymula, 2021). 560 twin pairs (ages 18–66, mean 44.7) completed the online survey in 2020–21. The monozygotic pairs (identical) total 401, and 159 are dizygotic (fraternal).[2] 83% of the sample is female and 59% have a university degree. Further demographic details are in Tables A1–A2.

In one task, participants undertook a cognitive challenge in which they were incentivized to solve ten puzzles from the matrix reasoning item bank, developed by Chierchia et al. (2019). Participants had 30 seconds to select the correct image from four available options to complete a sequence (Figure A1 presents an example).[3] They completed a practice round before starting the task. Puzzles had different difficulty levels, with an expected average score of 6/10. Participants earned AU$2 per correct answer if this task was chosen for payment.

Upon completing the task, participants reported 'How many of the ten puzzles do you think you got right?' and 'Where do you think you will rank in the puzzle task compared to other twins in this study, out of 100?.' As in Cesarini et al. (2009), the raw differences between perceived and actual score and rank are our baseline measures of overconfidence (overestimation and overplacement).[4] However, raw differences can be a biased measure of overconfidence (see e.g., Cronbach & Furby, 1970; Edwards, 2001; Juslin et al., 2000). For example, we worry that very low/high scores constrain the ability to reveal under- and overconfidence due to floor and ceiling effects. Consequently, for our twin decomposition analysis, we again follow Cesarini et al. (2009) and estimate our models controlling for performance in the individual puzzle task.[5]

[2]Forty-two are mixed sex and so we control for sex in our analysis. For the 518 same-sex twin pairs, zygosity was determined using either self-reported genetic test results (184 pairs), different self-reported blood type (25 pairs), or using the peas-in-a-pod questionnaire (309 pairs), which has been shown to predict zygosity with more than 90% accuracy (Ooki et al., 1990).

[3]If a participant failed to answer within the 30 seconds, the failure was recorded as an incorrect answer. This was relatively rare – only 13% of people failed to submit one or more answer (less than 7% more than one).

[4]Percentile ranks are defined as the minimum integer value X such that the participant's score is greater than X% of participants. Participants with tied scores also receive the same rank.

[5]Specifically, we include the baseline scores as covariates in the SEM model that we estimate. This is empirically equivalent regressing actual score/rank on perceived score/rank and using the residuals as measures of overconfidence.

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Participants correctly answered 5.0 (s.d. = 1.9) puzzles on average. They are slightly underconfident about their score (μ= − 0.57 s.d. = 2.23) but overconfident about their rank (μ=13.42 s.d. = 39.96). The distributions of both types of overconfidence look normal (Figure A2) and overestimation and overplacement are slightly correlated (r=0.20).

3. Methodology

To decompose the variation in overconfidence into genetic and environmental effects, we exploit that monozygotic (MZ) twins are genetically identical, while dizygotic (DZ) twins share 50% of the genes on average (assuming no assortative mating). Greater discordance between DZ than MZ twins is then indicative of genetic heritability. Despite growing access to genomic data, twin studies relying on discordance remain the dominant approach to estimate heritability (Becker et al., 2021). This is in part because of practical advantages – relatively small samples of twins are required to estimate heritability whereas tens or hundreds of thousands of genomes may need to be mapped. Additionally, twin studies have other appealing features such as capturing rare genetic variations and not being affected by incomplete genetic mapping (Friedman et al., 2021; Wainschtein et al., 2022).[6]

Formally, denote the variance of a trait Y as $\sigma^2_Y = \sigma^2_a + \sigma^2_d + \sigma^2_c + \sigma^2_e$ where the independent components are additive genetic effects (σ^2_a), dominance genetic effects (where expression of genes depends on interactions at particular loci) (σ^2_d), environmental effects common to siblings (e.g., from shared parenting) (σ^2_c), and environmental effects that are unique (σ^2_e), which include noise. Identification comes from the assumption that the common family environment contributes to overall variance; the same for MZ and DZ twins. Because additive and dominance genetic effects cannot be separately identified, $\sigma^2_d = 0$ is frequently imposed. Under these assumptions, the correlation of Y between MZ twins (r_{MZ}) captures $(\sigma^2_a + \sigma^2_c)/\sigma^2_Y$ and between DZ twins (r_{DZ}) $(0.5\sigma^2_a + \sigma^2_c)/\sigma^2_Y$. These correlations can be manipulated to estimate narrow sense heritability: $h^2 = \sigma^2_a/\sigma^2_Y = 2 * (r_{MZ} - r_{DZ})$.

In practice, researchers assume normality for the variance components and use structural equation modeling (SEM) to estimate variance shares (Neale & Cardon, 1992). The SEM approach is appealing because it ensures variance shares sum to one, it can accommodate controls and can be extended to multivariate decompositions, here allowing to estimate the degree to which overlapping genes explain the correlation between different overconfidence types. We follow the SEM tradition and estimate the bivariate AC(D)E-Cholesky model, using the *umx* package for R (Bates et al., 2019). We report our estimates as shares of explained variance.

We estimate four versions of the model and compare their fit. Our baseline model imposes $\sigma^2_d = 0$ (ACE), most common in the literature. Next, we impose $\sigma^2_d = \sigma^2_c = 0$ (AE), then $\sigma^2_a = \sigma^2_d = 0$ (CE), and finally $\sigma^2_c = 0$ (ADE). As discussed earlier, we follow Cesarini et al. (2009) and estimate our models with and without controlling for individual puzzle task score (rank), since very low/high scores (ranks) constrain the ability to reveal under- and overconfidence. We use non-parametric bootstrap clustered at the twin pair level (999 replications) to estimate standard errors and confidence intervals.

4. Results

First, we compare pairwise correlations in overconfidence for MZ and DZ twins (Fig. 1).[7] For overestimation, $r_{MZ} = 0.11$ (s.e. = 0.049) and $r_{DZ} = -0.02$ (s.e. = 0.089). The stronger correlation for MZ twins hints at the importance of genes. The lack of correlation for DZ twins suggests dominance

[6]Nonetheless, the strong assumptions underlying twin studies continue to generate debate among scholars. Proponents of twin studies often point to evidence supporting key assumptions (like equal environments for MZ and DZ twins) or to violations not being economically important (see e.g., Barnes et al., 2014; Conley et al., 2013; Felson, 2014). For a more critical perspective see, for example, Burt and Simons (2014).

[7]Fig. 1 uses the raw differences in perceived and actual puzzle task score/rank as measures of overestimation/overplacement. For the same figure after residualizing overestimation (overplacement) on puzzle task score (rank) and sex, see Appendix Figure A3.

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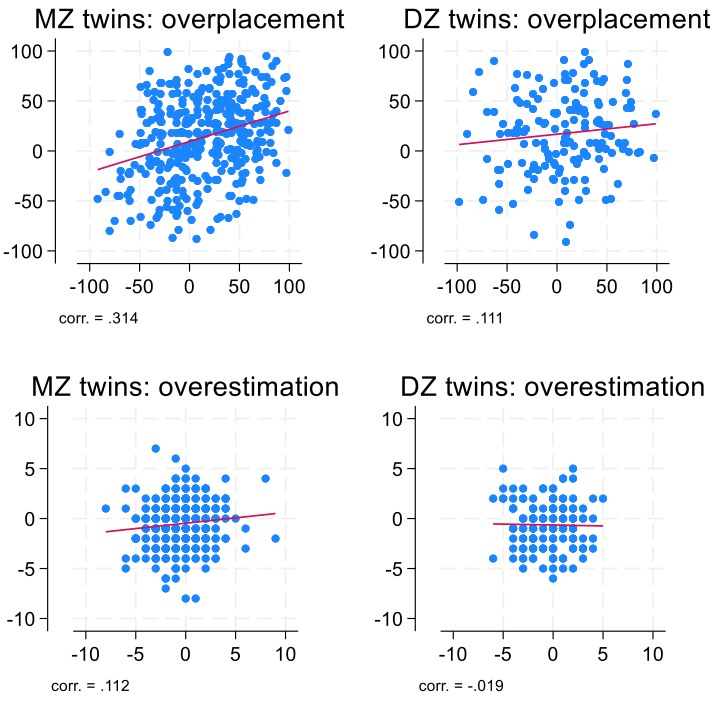


Fig. 1 Overconfidence correlations

genetic effects might be important (i.e., genes act multiplicatively); however, given fewer DZ twins this may reflect imprecision. Overplacement correlations are stronger, $r_{MZ} = 0.31$ (s.e. = 0.047) and $r_{DZ} = 0.11$ (s.e. = 0.080), pointing to genes accounting for part of the interpersonal differences.

Next, we formally estimate the variance shares using SEM (Table 1). The SEM path diagram (Fig. 2) shows the ACE model standardized factor loadings, including the covariances. When adjusting for the task score (Panel A), we estimate genetic heritability of 17% [CI = 0.11%, 27%] for overplacement and 19% [12%, 27%] for overestimation in the ACE specification (AE is virtually identical). AE model fits best and ADE fits slightly better than ACE. The implied heritability is similar across model specifications with little evidence for the importance of common family environment. There is strong negative correlation between genes that explain overplacement and overestimation (Fig. 2). Following Kuntsi et al. (2004), we calculate the genetic correlation coefficient = − 1.00 [−0.59, − 1] implying a near perfect relationship (for unadjusted overconfidence corr. = − 0.85 [−0.04, − 1]). This implies that genes that increase the tendency for overplacement decrease the tendency for overestimation.

Without controlling for the task score (Panel B), the genetic variance estimates for overplacement are larger (29% [23%, 36%] in the best-fitting AE model), which is also the case in Cesarini et al. (2009) (34% unadjusted, 16% adjusted). However, our estimates for overestimation become smaller (10% [3%, 18%]). In either case, conclusions are qualitatively similar – unique experiences explain most variation, but genes are a non-trivial factor.

5. Discussion

We provide estimates for the extent to which interpersonal differences in overconfidence are explained by genes versus environment in an Australian adult twins sample. Our unique contribution is that we study two types of overconfidence – overplacement and overestimation – in the same sample to uncover whether these distinct measures of overconfidence have different and related

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Table 1 AC(D)E-Cholesky decomposition estimates

	A.1: Overplacement (adjusted)			
	ACE	AE	CE	ADE
$\hat{a}^2$	0.17 (0.11, 0.27)	0.17 (0.10, 0.25)		0.00 (0.00, 0.00)
$\hat{d}^2$				0.19 (0.10, 0.27)
$\hat{c}^2$	0.00 (0.00, 0.03)		0.13 (0.07, 0.21)	
$\hat{e}^2$	0.83 (0.75, 0.90)	0.83 (0.75, 0.90)	0.87 (0.79, 0.93)	0.81 (0.74, 0.89)
$\hat{a}^2 + d^2$				0.19 (0.11, 0.26)
	A.2: Overestimation (adjusted)			
$\hat{a}^2$	0.19 (0.12, 0.27)	0.19 (0.13, 0.28)		0.00 (0.00, 0.00)
$\hat{d}^2$				0.21 (0.12, 0.30)
$\hat{c}^2$	0.01 (0.00, 0.00)		0.14 (0.09, 0.23)	
$\hat{e}^2$	0.81 (0.73, 0.88)	0.81 (0.72, 0.87)	0.86 (0.77, 0.91)	0.79 (0.71, 0.86)
$\hat{a}^2 + d^2$				0.21 (0.14, 0.29)
AIC	4525.661	4519.661	4528.436	4522.217
$\Delta\chi^2$ p-val	-	1.00	0.032	-
	B.1: Overplacement (unadjusted)			
$\hat{a}^2$	0.25 (0.13, 0.33)	0.29 (0.23, 0.36)		0.09 (0.00, 0.28)
$\hat{d}^2$				0.21 (0.05, 0.32)
$\hat{c}^2$	0.04 (0.00, 0.16)		0.24 (0.18, 0.31)	
$\hat{e}^2$	0.71 (0.64, 0.77)	0.71 (0.64, 0.77)	0.76 (0.69, 0.82)	0.70 (0.64, 0.77)
$\hat{a}^2 + d^2$				0.30 (0.23, 0.36)
	B.2. Overestimation (unadjusted)			
$\hat{a}^2$	0.05 (0.01, 0.12)	0.10 (0.03, 0.18)		0.04 (0.00, 0.56)
$\hat{d}^2$				0.07 (0.02, 0.14)
$\hat{c}^2$	0.04 (0.00, 0.11)		0.07 (0.01, 0.14)	
$\hat{e}^2$	0.71 (0.64, 0.77)	0.90 (0.82, 0.97)	0.93 (0.86, 0.99)	0.89 (0.82, 0.96)
$\hat{a}^2 + d^2$				0.11 (0.04, 0.18)
AIC	6231.450	6225.617	6238.001	6227.943
$\Delta\chi^2$ p-val	-	0.983	0.006	-

Notes: SEM estimates for the additive genetic effects ($\hat{a}^2$), dominance genetic effects ($\hat{d}^2$), common environment ($\hat{c}^2$), and unique environment ($\hat{e}^2$). Adjusted estimates control for the puzzle task score in the overestimation equation and the percentile rank in the overplacement equation; all estimates control for sex. $\Delta\chi^2$ p-val tests the improvement in log-likelihood relative to ACE. Nonparametric bootstrap 95%-level confidence intervals clustered at the twin pair level in parentheses.

heritability. Two previous twin studies estimated the heritability of overconfidence. Cesarini et al. (2009) estimated the heritability of overplacement in adult Swedes and Vogt et al. (2022) estimated the heritability of overestimation in Texan children.

We find that genetic variation can account for 17%–29% of variation in overplacement, an estimate that is in line with Cesarini et al. (2009). We estimate the heritability of overestimation at 10%–19%, less than 18–28% estimated for Texan children in Vogt et al. (2022). While it is true that Vogt et al. (2022) sample differs from ours, particularly in age, we cannot definitively say why our estimates are smaller than theirs.

Additionally, we find a negative genetic correlation between overplacement and overestimation suggesting that individuals who are genetically predisposed to exhibit more overplacement will show

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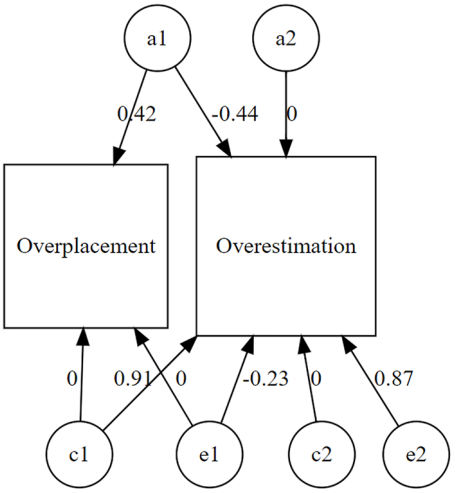


Fig. 2 SEM path diagram (standardized coefficients). Notes: Standardized factor loading for ACE model controlling for puzzle task score/rank

less overestimation. This estimate is fairly imprecise, and we caution against overinterpretation; nonetheless, the idea that the genes that drive overplacement might also nudge us away from overestimation is intriguing and could have practical implications. For example, researchers are increasingly using polygenetic scores to proxy for different traits. Our results imply that if a score is developed for overconfidence, researchers should be very careful in understanding what type of overconfidence the score reflects before applying it to their work, to avoid misinterpreting results. Moore and Healy (2008) defined three separate forms of overconfidence. In addition to overplacement, and overestimation studied in this paper, they also recognize overprecision which is an excessive certainty in the accuracy of one's knowledge. The implications of our finding are similar to Moore and Healy (2008) who, using a basic Bayesian belief updating model, predict a negative correlation between overplacement and overestimation and support this theoretical prediction with their own data and previous literature. They find that in difficult tasks people tend to overestimate their own absolute but underplace their own relative performance, and in easy tasks people underestimate and overplace. Moore and Healy urged researchers to not assume that all different types of overconfidence are the same and our results underscore this recommendation.

Our estimates suggest overconfidence is largely due to idiosyncratic circumstances. The relatively modest pressure from genetic forces could mean this trait is quite malleable (although it bears mentioning genes are not deterministic and merely increase the chances of certain behaviors). Given the impact that both over- and underconfidence have on various aspects of our lives, and considering their malleability, future research should focus on understanding how to achieve optimal levels across all types of overconfidence. We estimate that the variance due to genes is smaller for overestimation than for overplacement, suggesting that overestimation should be more easily influenced by non-genetic factors than overplacement.

Finally, we note that our study focused on a specific sample – Australian adults – and measured overconfidence with a single task. While our results align with previous findings for adults (Cesarini et al., 2009), further research utilizing a variety of tasks designed to measure different types of overconfidence is necessary.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/esa.2025.10016

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Competing interest. Nothing to declare.

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