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Interpreting estimates of heritability – A note on the twin decomposition

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ABSTRACT

While most outcomes may in part be genetically mediated, quantifying genetic heritability is a different matter. To explore data on twins and decompose the variation is a classical method to determine whether variation in outcomes, e.g. IQ or schooling, originate from genetic endowments or environmental factors. Despite some criticism, the model is still widely used. The critique is generally related to how estimates of heritability may encompass environmental mediation. This aspect is sometimes left implicit by authors even though its relevance for the interpretation is potentially profound. This short note is an appeal for clarity from authors when interpreting the magnitude of heritability estimates. It is demonstrated how disregarding existing theoretical contributions can easily lead to unnecessary misinterpretations and/or controversies. The key arguments are relevant also for estimates based on data of adopted children or from modern molecular genetics research.

1. Introduction

A longstanding question in social science is to what extent outcomes such as IQ, education and income vary due to genetic or environmental factors. A classical method to address this issue is to decompose the variation among twins and compare outcome correlations of monozygotic (*MZ*) twin pairs, who are genetically identical, with those of dizygotic (*DZ*) twin pairs who share on average 50% of the genetic makeup. Taubman (1976) and Behrman and Taubman (1976, 1989) reported genetic heritage was associated with some 40% of the variation in earnings and 50% of the years of schooling. The twins heritability model has remained popular in psychiatry, psychology and has been used also in sociology (Rodgers et al., 2008), political science (Alford et al., 2005) as well as economics. For instance, Cesarini et al. (2009a, 2009b, 2010) reported that heritability explains about 20% of individual variation in preferences, risk-taking, overconfidence and risk-aversion. In addition, Barnea et al. (2010) found a substantial element of investment decisions to be explained by a genetic factor. However, despite its popularity, the model has also been subject to criticism over the years, mainly related to that heritability estimates may comprise genetically induced environmental effects (Goldberger, 1979; Jencks, 1980; Joseph, 2001, 2002; Horwitz et al., 2003; Heckman, 2007; Manski, 2011).[1]

The purpose of this short note is to highlight theoretical contributions which have in common that they are crucial for the interpretation of heritability estimates but, unfortunately, are frequently not discussed by authors

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[1] Heckman (2007, p. 13251) writes that "[g]*enes and environment cannot be meaningfully parsed by traditional linear models that assign unique variances to each component*".

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presenting the twins heritability model.[2] The main point made is that much controversy could be avoided if authors combined the emphasis on the size of the genetic component with a more open discussion on that it may include gene environment interactions, passive, active and evocative gene environment correlations (Plomin et al., 1977; Jencks, 1980) as well as reciprocal causation between the outcome variable of interest and environments, generating individual and social multiplier effects (Dickens and Flynn, 2001). Their importance for the size of heritability estimates is essentially untestable, implying that any assessment ultimately depend on each readers' beliefs.[3] In the following, the twin model is formally derived and the sources of potential misunderstandings are explained. A more detailed account of the key arguments follow in Sections 3 and 4 before Section 5 exemplifies potential policy implications and Section 6 concludes.

2. The twin model decomposition

The twins heritability model typically decomposes the variation in outcomes (or phenotypes) into a hereditary part, denoted h^2, and a common environmental part, c^2. To formally derive these components, assume that an observed outcome Y is determined as the sum of two unobserved variables, genotype G and environment E[4]:

$$Y = G + E$$

These are symmetrically defined so that G is the expected value of Y for persons with a given genotype across the full population, and E is analogously the expected Y for a given environment. Taking the variance across the distribution of individuals, and dividing by σ_Y^2, gives the decomposition:

$$1 = h^2 + c^2 + 2rhc$$

where h^2 and c^2 are the ratios of genotypic and environmental variance in relation to σ_Y^2, and $h = \sigma_G/\sigma_Y$, $c = \sigma_E/\sigma_Y$. Pairing each individual with a genetically identical *MZ* twin sibling, whose outcome functions are $Y' = G' + E'$, the covariance across twin pairs is

$$\sigma_{YY'} = \sigma_{GG'} + \sigma_{EE'} + 2\sigma_{GE'}$$

Division by σ_Y^2 gives the correlation decomposition[5]

$$r_{YY'} = r_{GG'}h^2 + r_{EE'}c^2 + 2r_{GE'}hc$$

For *MZ* twins, $r_{GG'} = 1$. Denoting the left hand side correlation coefficient r_{MZ}, and assuming G and E uncorrelated so that the last term drops out, the right hand side consists of a hereditary part h^2 and a part related to the shared (common) environment c^2.

$$r_{MZ} = h^2 + r_{EE'}c^2$$

For *DZ* twins, one assumes they share exactly half of the genes. This is the case if there is no assortative mating and only additive genetic variance. The hereditary component of an outcome is then in expectation half of that observed for *MZ* twins, which yields[6]:

$$r_{DZ} = \frac{h^2}{2} + r_{EE'}c^2$$

The equal environment assumption (EEA) stipulates that the term $r_{EE'}$ is equal for *MZ* and *DZ* twins, implying that environments are as similar for *DZ* pairs as they are for *MZ* pairs. The hereditary part h^2 can then be empirically derived as

$$h^2 = 2(r_{MZ} - r_{DZ})$$

This is the simplest form of the twin model. Although studies often diverge from this basic framework, they generally rely on the additive functional form. The reason this may be controversial is that G and E are widely believed to be correlated. Empirically, *MZ* twins have been observed to experience more similar parental treatment and environments than *DZ* twins (Lytton, 1977; Scarr and Carter-Saltzman, 1979; Loh and Elliott, 1998; Borkenau et al., 2002). That would imply a non-linear specification and also violate the EEA. However, these are aspects which authors are (or should be) aware of, and the conventional interpretation of the h^2 estimate is instead that it includes a complex gene environment interplay involving G, E and Y as well as interactions between different gene types, G×G. For example, if genes influence the probability of smoking it may in turn increase the risk for lung cancer. In this case, the genetically induced environmental factor (tobacco smoke) will generate a higher correlation in lung cancer among *MZ* twins. One could argue whether this should be considered a genetic or environmental effect on lung cancer, but in the standard framework of the twin model it enhances the heritable variation in lung cancer risk. The amount of bias caused by the various forms of gene–environment interplay is in practice very difficult to assess.[7] The point made in this paper is that if authors are not explicit about this, readers may either interpret the h^2 estimate as a "genetic effect" (as authors sometimes refer to) or, conversely, may find h^2 based on such strong assumptions that it is uninformative.

[2] In this article, "the twins heritability model" and "the twin model" are used interchangeably to refer to the decomposition of the variance of *MZ* and *DZ* twins to understand the heritability of traits and behaviors. In economics, there are other purposes for using twin data which are not addressed in this paper, including as controls for unobserved endowments in family fixed effects models and that twin births may represent unanticipated shocks.

[3] It should be clear that this note does *not* contain any novel theoretical contribution but wishes to emphasize relevant aspects which by convention are often left out by authors.

[4] The outline here is based on Goldberger (1978).

[5] $r_{YY'} = \frac{\sigma_{YY'}}{\sigma_Y^2}$, $r_{GG'} = \frac{\sigma_{GG'}}{\sigma_G^2}$, $r_{EE'} = \frac{\sigma_{EE'}}{\sigma_E^2}$, $r_{GE'} = \frac{\sigma_{GE'}}{\sigma_G\sigma_{E'}}$

[6] To the extent that the two terms on the right hand side fail to explain r_{MZ} and r_{DZ}, there are also non-shared environmental factors, x^2. Economists would perhaps call it the residual component as $x^2 = 1 - r_{MZ}$.

[7] In Jencks (1980, p. 727): "*Since we have no working definition of 'environment' and no way of saying when two individuals have the same environment, we cannot hope to devise general procedures for estimating the bias.*"

3. Genetically induced environments

The presence of gene environment correlations (rGE) reflects that the probability of experiencing a specific *E* differs for individuals with different *G*. Plomin et al. (1977) distinguished between three rGE mechanisms through which genes may influence environments and indirectly cause various outcomes.

- *Passive genetic influence*: the genes of the parents influence the parents' behavior and thereby the environment of the child (e.g. intelligent parents raise their children in an intellectual environment).
- *Evocative genetic influence*: the behavior of the child evokes different responses from parents and others which are part of the child's environment. Borkenau et al. (2002) showed that treatment in childhood of *MZ* twins was significantly more similar than among *DZ* twins for nine items out of ten.[8]
- *Active genetic influence*: the genes of the child influence how the child experiences and generates his/her environment to compensate or reinforce inherited traits, e.g. due to their genetic similarity, *MZ* twins may generate their own environments more similarly than *DZ* twins (cfr. the tobacco lung cancer example above).[9]

In this framework, it follows that a hereditary component in years of schooling may contribute to an even larger h^2 for IQ measured later in life. Even though the environmental factor schooling may increase the later measured heritability of IQ, the definition of h^2 implies that both estimates reflect the initial *genetic* differences. If authors are unclear about this, the interpretation of the h^2 estimate is also unclear. Unfortunately, in the literature, genetically induced environments are rarely defined or discussed and it is therefore very common that the critique of the twin model claims that authors overestimate heritability, e.g. Heckman (1995), Lerner (2006), Charney (2008) and Nisbett (2009).[10]

There are other examples where critics simply do not accept that the environmental mediation is included in h^2 (Goldberger, 1979, Joseph, 2002).[11] Joseph criticizes the twins' heritability model by providing an example where an environmental factor, lead, is the cause of increased risk of a symptom, schizophrenia. If *MZ* twins spend more time together than *DZ* twins (through active and evocative rGE); they would be more likely to encounter the same environments, be more concordant for schizophrenia, and the twin method could "*be measuring nothing more than environmental effects*" (Joseph, 2002, p. 77).

In addition, the presence of gene–environment interactions (G×E) implies that heritability estimates may also be influenced by *E* factors which are shared by some *MZ* and *DZ* twin pairs, e.g. in the shape of schooling institutions. This happens if the sensitivity to a specific *E* differs for individuals with different *G* (Rutter et al., 2001; Moffitt et al., 2006; Lundborg and Stenberg, 2010). In the framework of the twin model, if the influence of *E* on *Y* depends on *G*, it will be more similar for *MZ* twins as they are genetically identical. Molecular genetics research provides many examples of G×E hypotheses. Caspi et al. (2002) reported evidence that a specific gene (termed MAOA) could moderate the association between childhood maltreatment and antisocial behavior in adulthood, such as criminal activity, and Caspi et al. (2003) found depressions were linked to G×E involving earlier stressful life events. However, evidence of G×E has typically been difficult to replicate, and they should primarily be seen as hypothetical examples of G×E mechanisms. Moffitt et al. (2006, p. 6–7) argues convincingly, on an evolutionary basis, why it is plausible that G×E are very common, though they may increase the estimate of c^2 as well as of h^2 if environments are genetically mediated.

4. Heritability – reciprocal causation and multiplier effects

Empirically, the size of the reported heritability estimates is difficult to reconcile with the so called Flynn effect (Flynn, 1994, 2000). The Flynn effect refers to that IQ scores among 18 year old Dutch men increased 1952–1982 by 1.33 standard deviations, or 20 IQ points (SD 15) on a test derived from Raven's Progressive Matrices, and that similar developments apply to a number of countries. Since the presented estimates of h^2 for IQ have regularly been above 60% (e.g. Neisser et al., 1996), the recorded increase in IQ would require environmental improvements of more than 2.5 standard deviations or, as put by Dickens and Flynn (2001, p. 348), that the average Dutch man in 1982 lived in an environment "*well into the highest percentile of the 1952 Dutch distribution*".

The contribution of Dickens and Flynn (2001) is to reconcile the rise in IQ over time with the size of the estimates of h^2. They demonstrate how small exogenous environmental changes may have large effects on both total environment and IQ. This is because reciprocal causation between *E* and *Y* may generate multiplier effects. Formally, expressing variables as standard deviations from their means, they assume:

$$Y_i = hG_i + cE_i$$

with G_i and E_i uncorrelated, the square of the correlation coefficients h and c are the fractions of variance in Y_i explained by G_i and E_i respectively. They then add an

[8] The items included if they were dressed alike, were given similar hair styles, had the same leisure activities arranged for them, were referred to as "The Twins", received the same toys as presents.

[9] Jencks (1980) defined active and evocative rGE as endogenous environments, but did not include passive rGE since only the portion of the environment caused by the child's genotype is considered in his definition.

[10] From his critique of *The Bell Curve* (Herrnstein and Murray, 1994), Heckman (1995, p. 1103): "*. . .the authors assume that AFQT is a measure of immutable native intelligence. In fact, AFQT is an achievement test that can be manipulated by educational interventions.*" From Nisbett (2009, p. 27): "*. . .the correlation between identical twins overestimates heritability* [. . .] *because the environmental experiences of identical twins who are reared separately in quite different environments are highly similar since they look so much alike or have other characteristics in common that tend to elicit the same sorts of behavior from other people.*"

[11] Goldberger (1979, p. 341); "*this line of argument* [rGE and G×E included in h^2] *will not do*, for it *violates the basic definition of genotype as the expected phenotype of persons with a given genetic constitution, the expectation being taken over the full distribution of available environments. It revises the definition by taking the expectation over the distribution of environments with which that genetic constitution is currently associated.*"

individual multiplier, or reciprocal causation, between Y_i and E_i by allowing for changes over time:

$$Y_{it} = hG_i + cE_{it-1}$$

$$E_{it} = \alpha_1 Y_{it} + (u + e_i)$$

Within parentheses is the sum of exogenous environmental influences; a random variable with mean u. The individual multiplier may be generated by an increase in some exogenous environmental improvement (u) which affects everyone or by a random event which affects the individual's environment (e_i). An increase in this factor will increase Y_{it} via the coefficient c and the new level of Y_{it} will improve the environment in the next period via α_1. Thus, if the level of IQ today tends to be correlated with the quality of today's environment, this will lead to still better environments which in turn will enhance IQ and so on. The process is both repetitive and cumulative and may produce an upward or downward spiral of IQ that could become very large.[12]

A social multiplier is added by expanding the function determining E_{it} to depend on the average $\bar{Y}_t$ in the population:

$$E_{it} = \alpha_1 Y_{it} + \alpha_2 \bar{Y}_t + (u_t + e_{it})$$

In terms of IQ, it is now influenced by an average of all the environmental effects of all of society's members. It implies a *double* upward or downward spiral because if the IQ of an individual increases, it will by definition also increase the average IQ in the society. This improves everyone's environment, and causes IQ levels to increase further through the improvement of interactions between pupils in school, between pupils and teachers, between teachers and teachers, between parents and children and so forth. The Flynn effect no longer needs to be explained by a large improvement in exogenous environmental factors. Instead, a collective increase in the quality of interactions, which is small at the individual level, may have large effects on the population average in relation to the standard deviations.[13] An example from their simulations, assuming $h = .2$ and $\alpha_2 = .5$, implies that overall improvements in E_{it} by .01 standard deviations each year (via u) for 30 years would roughly match the observed IQ gain among Dutch youth between 1952 and 1982.

In sum, the multiplier effects contribute further to suggest that estimates of h^2 are partly generated by environmental factors. Nevertheless, the convention in the behavioral genetics literature is that this aspect is regularly left out or is insufficiently explained.

5. Implications

While it is clear that most outcomes are in part genetically mediated, quantifying the genetic heritability is a different matter. The key argument made in this note is that authors in the behavioral genetics literature can avoid misleading interpretations of heritability estimates, both on behalf of the researchers themselves and by their critics, by explicitly defining "genetically induced environments" as active and evocative rGEs (Section 3) as well as reciprocal causation and multiplier functions (Section 4), referring to the theoretical accounts given already by Plomin et al. (1977), Jencks (1980) and Dickens and Flynn (2001).

One may wonder if this point is important, since no obvious policy implications follow from a large hereditary component. As famously exemplified by Goldberger (1979), genes leading to poor eyesight may almost perfectly be compensated with eyeglasses. That example is intuitive but also very specific. To compensate a lack of genetic endowments (e.g. for cognitive ability and health) may not always work so smoothly. As a different example, one may consider educational policy where a government must choose a pedagogic strategy and also choose how much resources should be allocated to compensate low achievers. Both these decisions could well be influenced by whether those who set the political agenda believe 60% of the variation in IQ is predominantly determined by genetic endowments (e.g. Neisser et al., 1996), or whether they believe the 60% reflects only small genetic differences whose correlations with environments blow up the heritability estimate. To the extent that this affects the interpretation made by readers of these articles, it may in turn influence policy decisions.[14] In sum, although it may in essence be true that heritability estimates are "fundamentally uninformative" (Manski, 2011), it may not be unimportant for policy in practice.

6. Discussion

Since the early 2000s, molecular genetics researchers have been able to directly observe individuals' genetic variants and their associations with specific outcomes.[15] In relation to the points made in this paper, many of these studies have reported very low genetic associations.[16] However, this may just reflect problems with statistical power and it is possible that they eventually will corroborate the heritability estimates based on twins or adopted children. By extracting information from genes with some but small explanatory power, Yang et al. (2010) reported heritability estimates of 45% for the variation in

[12] If one allows for reciprocal causation by inserting the function for E_{it} and assuming $Y_{it} = Y_{it-1}$ in the long run, the square root of heritability h^2 is reduced as $h < h/(1 - \alpha_1 c)$ in the likely case that c and α_1 are above zero.

[13] As permanent environmental changes, Dickens and Flynn suggest improvements in education, that radio and television may have enhanced cognitive learning, that extended leisure has promoted reading, puzzle solving and/or that smaller families may have increased quality time with children.

[14] To be concrete, a stronger emphasis on environmental factors may lead to a focus on documentation (as in the Reggio Emilia pedagogy). The focus on the environment in the first place (via documentation) makes it possible to find out in retrospect if a low achieving child has been met with the same attitude as other (high ability) children? Is there some other interest which takes away the child's focus from developing this particular ability? Is there a lack of structure? Rather than seeking to bring out some innate ability, these questions may communicate a different attitude toward the potentials of that child. This may or may not constitute an important difference.

[15] Beauchamp et al. (2011) contains an excellent discussion on how findings from molecular genetics may be of relevance for researchers in economics and other social sciences.

[16] The hereditary component reported for IQ scores was less than 3% (Hirschhorn, 2009, Goldstein, 2009) and similar for height (Butcher et al., 2008, Meaburn et al., 2008), though this increased to 10% when studying a sample of 183,000 individuals (Allen et al., 2010).

height and Davies et al. (2011) explained 40% of the variation in IQ. This can be compared with that quantitative genetics frequently have shown heritability estimates in IQ scores of .60 or higher. Visscher et al. (2006) explored that the observed genetic correlation between 3375 sibling pairs (non-twins) varied between 38 and 62 percent, and estimated that 80 percent of the variation in body height was heritable. This comes very close to estimates from behavioral genetic studies.

It is important to note that the (old) theoretical arguments brought forward in this paper on active/evocative rGEs (Section 3) and reciprocal causation (Section 4) are relevant also for other heritability estimates from studies of adoption data or molecular genetics. All estimates include, to an unknown extent, the mechanisms pointed out as well as gene–gene-interactions. While one may argue that heritability estimates are based on strong assumptions, there is nothing unusual about that in the academic literature. Nevertheless, it is reasonable to expect that we should be open about these underlying assumptions to avoid generating misinterpretations of the evidence.

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