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NATURE OR NURTURE?
A Note on the Misinterpreted Twin Decomposition

by

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Abstract: The classical twin model has often been used to determine whether variation in outcomes such IQ, schooling and other behavioral traits, originate from genetic endowments or environmental factors. Despite some heavy criticism from prominent scholars, the model has recently reappeared in highly ranked economics journals to perhaps spark off a revival of the method. This article seeks to specify the assumptions which generate the apparently profound divide in viewpoints. A general problem is that most authors do not properly discuss the underlying assumptions of the twin model. It has partly led to a disarray of thoughts, concrete examples are provided, since the interpretation of the results and the risk for misleading interpretations are not spelled out. Therefore, perhaps surprisingly, a brief account of the theories behind the main assumptions of the twin model provides a useful contribution.

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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. The classical twin model addresses this issue by comparing outcome correlations of monozygotic (MZ) twin pairs, who are genetically identical, with correlations of dizygotic (DZ) twin pairs who share on average 50 per cent of the genetic makeup. Under certain assumptions, the variation may be decomposed into a genetic and an environmental component. In the economics literature, Taubman (1976) and Behrman and Taubman (1976, 1989) reported genetic heritage was associated with some 40 percent of the variation in earnings and 50 percent of the years of schooling. While the twin model has remained popular in psychiatry, psychology and sociology, some recent articles in prestigious economics journals signal a revival of the methodology also in economics. Cesarini et al. (2009a) reported about 20 per cent of individual variation in preferences and risk-taking is explained by heritability and Cesarini *et al.* (2009b, 2010) reported similar magnitudes for overconfidence and risk-aversion. In stark contrast to the acceptance implied by these publications, the model has been subject to criticism over the years from several prominent researchers, and has even been described as meaningless (e.g. Goldberger 1979, Jencks 1980, Joseph 2001, 2002, Horwitz et al. 2003, Heckman 2007).[2]

The purpose of this article is to point out the twin model's major issues of controversy, foremost regarding how results of the twin model are interpreted.[3] In particular, the implications of the equal environments assumption (EEA) are clarified, stipulating that environmental differences within twin pairs should be equal regardless of zygosity. The EEA may appear straightforward but it is surrounded by misunderstandings and misconceptions, from both

[2] Heckman (2007, p13251) writes that "[g]*enes and environment cannot be meaningfully parsed by traditional linear models that assign unique variances to each component*".

[3] While the topic may partly be seen as a matter of curiosity, there is undoubtedly also a provocative element related to some alleged implications for policy and/or identity. The sensitivity of the issue was not least demonstrated by the controversy surrounding *The Bell Curve* (Herrnstein and Murray 1994) and, albeit to a much lesser extent, *The Blank Slate* (Pinker 2002).

users of the model and its critics (examples given in Section 2 and Section 4). A detailed account therefore seems highly motivated. Since MZ twins tend to experience more similar environments than DZ twins, the twin model must assume this is genetically induced. A problem then is that there are subtle arguments which need to be addressed in detail. Unfortunately, this is rarely done and, instead of discussions about the credibility of the relevant assumptions, it has bred a disarray of thoughts in the twin model literature. A contribution of this paper is to discuss the assumptions implied by the EEA which, if violated, potentially undermine the heritability estimator.

The paper is organized as follows: Section 2 contains a formal account of the twin model decomposition, where its underlying assumptions are presented. The definition of genetically induced environments closely follows Plomin et al. (1977). In Section3, the additive functional form is addressed, and the contribution of Dickens and Flynn (2001) is used to reconcile empirical findings on nationwide increases in IQ with twin model estimates. Section 4 goes through empirical research to discuss the credibility of the EEA regarding pre- and post-natal environments. Section 5 presents a brief account of the evidence based on twins reared apart, which is often referred to as support for the twin model estimates. Section 6 concludes with a discussion.

2 The classical twin model decomposition

To formally demonstrate the idea of the classical twin model, assume first the correlation coefficient of an outcome among MZ twins, r_{MZ}, to consist of a hereditary part h^2 and a shared environment c^2. Adding a similar assumption for DZ twins, who on average share half of the genes, we can write:

$$r_{MZ} = h^2 + c^2 \qquad [1]$$

$$r_{DZ} = h^2 / 2 + c^2$$

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, e^2. Economists would perhaps call it the residual component as $e^2 = 1 - r_{MZ}$.

Crucially, the shared environmental factors, c^2, are assumed to influence correlations of MZ and DZ twins in the same manner. This is the equal environment assumption (EEA). With c^2 assumed equal regardless of zygosity, the term h^2 can be expressed as

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

With a numerical value on h^2 inserted in [1], c^2 can be derived as

$$c^2 = r_{MZ} - h^2$$

This is the simplest form of the twin model. Although studies often diverge from this basic framework, it illustrates the role of the EEA and that estimates of h^2 are based on an additive functional form. Thus, the estimate of h^2 encompasses direct genetic effects (G), interactions between different gene types (GxG) and gene-environment interaction effects (GxE). Moreover, h^2 also captures the effects of gene environment *correlations* (rGE), meaning that it includes the effects of genetically induced environments.[4] This is important to note. The EEA may otherwise seem highly implausible since MZ twins are often presumed to experience more similar environments than DZ twins.

[4] GxE reflect that the sensitivity to a specific E differs for different G, whereas rGE reflect that the probability of a experiencing a specific E differs for different G.

Now, given that the EEA allows for effects of environments which *originate* from genetic differences, it is useful to be explicit about what kind of gene-environment interplay one could have in mind. Plomin *et al.* (1977) distinguished between three 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 (10 items) was significantly more similar than among DZ twins.

- *Active genetic influence*: the genes of the child influence how the child experiences and generates the 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). [5]

In this particular framework, it follows that a heritability estimate based on a gap $r_{MZ} - r_{DZ} > 0$ in earnings can *not* be questioned by that there is a gap in $r_{MZ} - r_{DZ} > 0$ also in the years of schooling. The definition of h^2 instead implies that initial genetic differences explain both gaps in correlations and, consequently, *that* is the underlying assumption which is meaningful to criticize. However, it is very common in the literature that the critique of the twin model is off target on this point, as the EEA allows for GxE and rGE (e.g. Heckman 1995, Lerner

[5] 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.

2006, Charney 2008).[6] These controversies appear unnecessary and largely caused by unclearly stated assumptions. Unfortunately, in the quantitative genetics literature overall, it is striking how unconcerned authors are to clarify the theoretical mechanisms which underlie the interpretation and the understanding of the h^2 estimate. At best, it is briefly stated that h^2 encompasses environmental effects which are genetically induced. It is rarely discussed, as in Plomin et al. (1977), what kind of interplay this could imply.

There are other examples where critics simply do not accept these assumptions (Goldberger 1979, Joseph 2002).[7] Joseph criticizes the EEA by providing an example where an environmental factor, lead, is the cause of increased risk of a symptom, schizophrenia. Even if MZ twins for strictly genetic reasons spend more time together (through active and evocative rGE), they would be more concordant for schizophrenia. The twin model views this as an effect of heritage even though it is caused by an environmental factor (Joseph 2002, p77). The controversy is then about terminology. this may in fact be one of the major sources of controversy surrounding heritability estimates. Again, this highlights the value of clearly stated assumptions in quantitative genetics research.

Importantly (and hinted at in Joseph's example), while rGE is allowed within the EEA, the rGE must not be related to the perceived level of (dis)similarity of the co-twin sibling. If the physical twin (dis)similarity affects the individual twin behavior, e.g. the willingness to mimic the behavior of the twin sibling, it would make zygosity an environmental factor which violates the EEA. Twin studies would in such a case compare correlations between two groups

[6] From Heckman's critique of the Bell Curve Heckman (1995, p1103): "*…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*."

[7] Goldberger (1979, p341); "*this line of argument* [rGE and GxE 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*."

where not only the genetic similarity varies, but environmental similarities vary partly as well. We return to this issue in Section 4.

3 Non additive effects

As pointed out, gene environment interaction effects are attributed to h^2. The genetic component is thereby overestimated whereas the environmental component is underestimated (Rutter 2000, Rutter *et al.* 2001). The so called Flynn effect (Flynn 1994, 2000) indicates that the presence of non-additive effects is problematic for the twin model. The IQ scores among 18 year old Dutch men increased 1952- 1982 by 1.33 standard deviations (similar developments apply to a number of countries). Since the presented estimates of h^2 for IQ have regularly been above 60 percent (e.g. Neisser et al. 1996), the recorded increase in IQ would require absurd environmental improvements, more than 2.5 standard deviations. Dickens and Flynn (2001) seeks to model mechanisms of interplay between genes and environments to reconcile the rise in IQ over time with the large estimates of h^2. They propose a reciprocal causation between genes and environments which produces gene-environment correlations. Foremost, they highlight two mechanisms which produce different multiplier effects which allow for large environmental impacts which are hidden ("masked") in the estimates of h^2.

First, reciprocal causation may act as a multiplier. If a high 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. Hence, "*the fact that past environmental influences have affected today's ability makes today's environment a sort of weighted average of all environments experienced in the past*"(p351). This makes it possible that a small genetic advantage may evolve into a large effect via the interplay with environments. This reciprocal causation is one of two different multiplier effects in the model.

Second, the *social* multiplier. This differs from the reciprocal causation in that it is not related to individuals' active rGE. Instead, it is related to overall changes in the society. That is, 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. This collective increase in the quality of interactions may be small, but the slightly improved interactions constitute a consistent factor (rather than a temporary one), which may have a large effects on the population average. Exemplifying with the means and standard deviations from flipping of a coin 100 times, they demonstrate how small consistent factors may increase mean values enormously in relation to the standard deviations (in their example, when giving heads and tails values 1.1 and 2.1 instead of 1 and 2).

The mechanisms suggested would explain why effects of environments tend to be hidden in twin model estimates, as the rGE makes influential (permanent) environmental factors just reinforce the heritability estimate.[8] Of course, when one compares IQ between generations, the environmental influence is not hidden by rGE. To explain the improved IQ, the model implies that one should look for permanent (but possibly small) 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.

4 Equal environments assumption

[8] This would also explain why c^2 and e^2 often vanish with the age of the twin samples, see Dickens and Flynn (2001) for a detailed discussion and further references.

In this section, an account is given of empirical studies which deal with some specific implications of the EEA. The discussion encompasses pre-natal differences between MZ and DZ twin couples, that MZ twins may be treated more similarly due to parents imposing an identity on them as MZ twins, and that twins may have mutual effects on one another which differ between MZ and DZ twins. This last point turns out to have potentially important effects on how we interpret the hereditary component of the twin model.[9]

A Pre-natal environments

The nutrition and the overall development of the embryo in utero largely depend on the human placenta. Regarding this aspect, pre-natal environmental conditions differ between MZ and DZ twins. The chorion, which is the sac which surrounds the embryo in utero, is shared by about two-thirds of MZ twins. These also share the same placenta. Thus, only about one-third of the MZ twins are dichorionic and have separate placenta, whereas this always applies for DZ twins.

An interesting test is to single out dichorionic MZ twins and compare their correlations with monochorionic MZ twins, and with DZ twins. Jacobs *et al.* (2001) studied 451 Belgian same-sexed twin pairs aged 9-11 where the chorion type had been determined. Their results on total IQ scores support the EEA as correlations for mono- and dichorionic MZ twins were almost identical (.83 - .82). A slightly different picture was reported in partial subtests regarding verbal tests in arithmetic (.66 - .49) and vocabulary (.77 - .70). While these figures indicate that h^2 might be overestimated, the correlation among dichorionic MZ twins was always significantly higher than DZ twins whose recorded correlations were .44, .34 and .46 respectively.

[9] When tested empirically, the EEA has not been rejected (Kendler et al. 1993, Hettema et al. 1995, LaBuda et al. 1997, Kendler and Prescott 1999, Kendler et al. 2000, Xian 2000, Borkenau et al. 2002) but as acknowledged by Bouchard and McGue (2003, p26), there are so many minor environmental factors which intervene that these may well be extremely difficult to capture.

The effect of chorion type has been examined on a number of issues. No differences have been reported on neonatal temperament, psychiatric syndromes, pro-social behavior, blood-pressure, BMI, height and/or weight (Riese 1999, Wichers *et al.* 2002, Hur, 2007 and Fagard *et al.* 2003, Hur and Shin 2008, Loos *et al.* 2001), but significant differences on dermatoglyphic patterns, brain morphology and tooth size (Reed *et al.* 1997, Reed *et al.* 2002, Burris and Harris 2002, Race *et al.* 2006). In sum, chorion type may tend to exaggerate $r_{MZ} - r_{DZ}$ differences, but is unlikely to be of any fundamental importance.

B Post-natal environments

It may be argued that growing up with an identical twin sibling or a fraternal twin sibling constitutes two different environments. There are two concerns. First, parents may exaggerate MZ twin similarity by dressing them the same way and impose an identity of similarity between them which is not originating from genetic endowments. Studies on twins whose zygosity has been mistaken have supported the EEA. Treatment of twins has been found to be related to actual zygosity rather than perceived, implying that parents respond according to differences/similarities between twins rather than create them (Lytton 1977, Loh and Elliott 1998).[10]

Second, the EEA implies that "*members of a twin pair are not having any mutual effect on one another, i.e.* [in terms of] *sibling cooperation/rivalry*" (Gillespie *et al.* 2003, p384). To see this more clearly, it may be useful to consider that the purpose of the twin model is to check how correlations vary between groups due to their respective genetic compositions (MZ or DZ), when environmental factors influence correlations in the same manner in both groups (i.e. regardless of zygosity). If MZ twins are treated more similarly due to *evocative* genetic

[10] As discussed in Section 2, if parent's more similar treatment of MZ twins is evoked by their genetic similarity, it is not a problem for the EEA.

influences, it increases their correlations but it is in line with the EEA. This may transfer into more similar environments (e.g. peers) also through *active* genetic influence, but it still does not violate the EEA if it is assumed to follow strictly from the fact that each MZ twin, as an individual, generates more similar environments.

In contrast, if the fact that the co-twin is identical rather than similar affects the attitudes *within* twin couples, towards each other, zygosity becomes an environmental factor. The same applies if the above mentioned similarity of treatment also affects the mutual influence on one another within twin couples (e.g. the amount of envy). Again, the zygosity of the co-twin has an impact on the environment, and the EEA is violated as the environmental factor *zygosity* influences how correlations of twin couples emerge. A greater rivalry between DZ twins has been reported in several studies (Koch 1966, Eaves 1976, Carey 1986, Smith 2007). In this case, the EEA is valid only if the rivalry is unrelated to the (dis)similarity of the co-twins and, in addition, the rivalry must not affect the similarity of parental treatment of the twins.[11] These assumptions seem highly unlikely to hold.

Related arguments can be found in Horwitz et al. (2003), on non-genetic reasons for more similar peers among MZ twins, and Joseph (2002) who explicitly addressed the higher level of closeness of MZ twins. Joseph refers to Kringlen (1967) who documented the environments of 75 MZ and 42 same-sex DZ pairs, where one or both had been diagnosed with schizophrenia. Over 90 per cent of the MZ twins experienced "identity confusion" and 65 per cent had "extremely strong" level of closeness, whereas these fractions among DZ twins were only 10 per cent and 19 per cent respectively. If the emotional attachment, or the identity confusion, stems from that the sibling looks like a copy of oneself, the EEA is violated. The EEA only holds if the attachment emerges from active and/or evocative genetic influences.

[11] In short, if the (dis)similarity of treatment is related to the level of rivalry, the EEA is violated.

Let us for the sake of argument assume that the greater rivalry between DZ twins is caused by reasons unrelated to the genes of the individual twin. One may then wonder if the impact would be serious or innocuous for the estimates of h^2. First, rivalry is likely to be a permanent factor. This fact alone may be sufficient to yield non-trivial differences in how they experience the "same" home environment, because these differences may be amplified over time. Studies have shown that small differences in child behavior may evoke large differences in parental harshness, warmth and/or monitoring (Rowe 1981, 1983, Dunn and Plomin 1986, Reiss 2008 and references therein).[12] If we assume the rivalry is a small environmental effect at each point in time, but permanent, it is interesting to consider an example provided in Dickens and Flynn (2001), where a small *genetic* advantage for playing basketball turns into a large advantage thanks to the environmental factor that the father enjoys playing basketball. This is a minor environmental factor if the father plays with his son once every year, but an important factor if it happens several times a week on a permanent basis. Their argument shows how a small genetic advantage may develop into a large advantage through environmental influence. Correspondingly, twin rivalry/cooperation may have large effects on DZ correlations due to its persistence and ensuing environmental interaction effects (ExE) and correlations (rEE). This would potentially make estimates of h^2 strongly upward biased. However, the argument is by and large untestable and as pointed out by Joseph (2002), the twin literature tends to put the burden of proof for demonstrating the invalidity of the EEA in the hands of the critics of the twin model.

Cesarini et al. (2009a, p828) bring forward three arguments to support the validity of the EEA. Their first argument regards the evidence of twins reared apart, which is discussed in Section 5. Second, they argue that the EEA is valid despite the fact that MZ twins may have

[12] Different treatments may not necessarily imply different experiences, but Asbury *et al.* (2008) reported that also MZ twins differ significantly in their experiences of class-room environments, to a large extent explained by their respective achievements.

more frequent contacts. This is in line with that a higher frequency of contact may be explained by that it is related to active and/or evocative rGE, which would not violate the EEA (see Section 2). However, they appear to believe that the higher frequency in itself is the problem, and therefore claim that the higher frequency of contact between MZ twins is caused by their higher degree of similarity.[13] If that argument holds, the EEA *is* violated precisely for the reasons discussed above, as it implies that the similarity of MZ twins affects their behavior towards one another (similarity becomes an environmental factor). As for their third argument, parents' more uniform treatment of MZ twins is claimed to not have influenced their correlation levels. Again, their argument does not make sense as the more uniform parental treatment of MZ twins would be expected, within the confines of the EEA, by way of active and evocative rGE. Unfortunately, it is difficult to discard this confusion around the twin model in general, and the EEA in particular, as isolated mistakes by an individual researcher, since the paper was written by five co-authors and reviewed by six anonymous referees according to the acknowledgment. In addition, a similar reasoning is found in Cesarini et al. (2010). The disarray of thoughts is underlined by the key role of the EEA for a valid interpretation of h^2 as reflecting heritability. As with the off-target critique discussed in Section 2, it appears largely to be a consequence of authors' insufficient discussion on what "genetically induced environments" imply in terms of gene-environment interplay.

5 Twins reared apart

Given the debatable properties of the EEA, a very convincing support of the twin model estimates would be if results could be reproduced with twins reared apart. More specifically, given random assignment to foster families, if the difference $r_{MZ} - r_{DZ}$ also holds for twins reared

[13] From Cesarini et al. (2009a, p828). "*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).*"

apart, $r_{MZA} - r_{DZA}$, the estimates would not have to rely on the EEA. In fact, it is an often inferred argument that such studies do yield results similar to those of conventional twin studies. For example, Cesarini et al. (2009a, p828) write: "*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).*" However, the shortcomings in the available data are quite severe, and the evidence is far from as fitting as the above quote might imply.

Overall, studies of twins reared apart are based on small samples where twins were in fact partly reared together. Joseph (2001 p79) points out that samples of twins reared apart "*are biased in favor of similarity, since the twins had to have known of each other's existence in order to respond to the researchers*". One must also consider that they may have been reared by relatives, in different branches of the same family, and/or that they may have stayed in the same regions and/or kept in contact. Both in the US and Sweden, authorities responsible for the placement into rearing families were instructed to match biological and rearing families on their characteristics (Scarr and Weinberg 1994, Appendix of Holmlund *et al.* 2008). With non-random placement, the observed difference in correlations should obviously be closer to what is observed for twins reared together.

An influential study on twins reared apart is the Minnesota study of twins reared apart, MISTRA. Bouchard *et al.* (1990) reported correlations for MZ twins reared together and reared apart. For IQ scores (full scale), it was .88 if reared together (n = 40 twin pairs) and .69 (n = 48) for MZ twins reared apart, a difference which was slightly larger in verbal skills (.88 - .64). In their article, they focus on other traits as they argue in defense of the EEA: "*For almost every behavioral trait so far investigated, from reaction time* [.80 - .81] *to religiosity* [.49 - .51], *an important fraction of the variation among people turns out to be associated*

with genetic variation. This fact need no longer be subject to debate; rather, it is time instead to consider its implications" (Bouchard *et al.* 1990, p227). In a later study, Bouchard (1998) include "*preliminary*" correlation coefficients also for DZ twins reared apart; .47. Although difficult to establish with precision, it would imply a point estimate of h^2 which is .44, i.e. lower than many conventional twin studies but still in support of a substantial hereditary component.[14] McGue (2008), in a brief survey of behavioral genetics research, brings out the evidence from Bouchard et al. (1990) as the most relevant, even though it is only based on MZ twins reared apart.

In a study on Swedish twins, Lichtenstein *et al.* (1992) reported correlation coefficients for all four relevant groups, MZ and DZ twins reared together and reared apart (aged 26-60). For highest attained education, the $r_{MZT} - r_{DZT}$ difference reported was .64 - .47 (n = 45 and 37). For male twins reared apart, the $r_{MZA} - r_{DZA}$ difference was .42 - .11 (n = 21 and 45). This indicates a maintained or even increased difference in correlation for male twins reared apart. However, for female twins, the estimate of h^2 would drop from .36 to .22 if twins were reared apart.[15] The twin couples included in Lichtenstein *et al.* (1992) were conditioned to have been separated at the age of ten or earlier. Björklund *et al*. (2005) used the same condition when analyzing register data of earnings correlations for Swedish twins. The $r_{MZT} - r_{DZT}$ difference for males was then .36 - .17 (n = 2052 and 3269) whereas for twins reared apart .07 - .16 (n = 45 and 41). This is completely at odds with the hypothesis of the EEA. For females, the results also thoroughly reject the EEA as twins reared together show .31 - .12 (n = 2395 and 3474) whereas the reported difference for twins reared apart is -.05 - .17 (n = 41 and 64).

[14] $h^2 = 2*(.69 - .47) = .44$.
[15] The $r_{MZT} - r_{DZT}$ difference was .70 - .52, (n = 39 and 53) and for twins reared apart .56 - .45, (n = 19 and 79).

6 Discussion

Modern molecular genetics have made it possible to study the entire human genome, i.e. the complete set of genetic markers of an individual. Since 2005, so called genome wide association studies (GWAS) has allowed researchers to directly observe individuals' genetic variants and their associations with specific outcomes (Pearson and Manolio 2008 for an introduction to GWAS). Generally speaking, the direct genetic effects reported has so far been tiny (Hirschorn 2009, Goldstein 2009), less than 3 percent of the variation in height (Weedon *et al.* 2008), and even less of the variation in IQ scores (Butcher *et al.* 2008, Meaburn *et al.* 2008). This may be because the traits are influenced by gene types in combination, and that these will be identified in future research. Considering that quantitative genetics frequently have shown heritability estimates in IQ scores of .60, or higher. The two strands of literature still appear far away from reconciling the evidence.

This paper has shown and discussed the assumptions necessary to believe that genes have a multiplying effect. One may argue that the twin model is based on strong assumptions, but there is nothing unusual about that in the academic literature. Nevertheless, it is reasonable to ask from authors to be open about the underlying implications of the twin model assumptions, and thereby demonstrate the risks for generating misleading interpretations.

The upper bound of heritability, reported in quantitative genetics, would be reduced if one believes GxE interactions are substantial. Some researchers believe that they are, basing their belief on an increasing body of evidence of GxE in molecular genetics and that epigenetic research has established that environments influence gene functions independently of the DNA (see Lundborg and Stenberg 2010 and references therein for an overview of GxE and

epigenetic research). Perhaps more importantly, this research also challenges the traditional view of separating between effects of genes and environment.

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