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Heritability and public policy reconsidered, again

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Abstract: I offer a way out of the Taubman-Goldberger controversy on the public policy (ir)relevance of heritability studies by arguing for a quasi-experimentally controlled comparison of the estimates that these studies provide. If the environments individuals are exposed to are under such control, changes in the genetic and the common environment (family) variance components underlying inter-individual differences can be ex-post informative regarding the evolvement of sources of inequalities in a population. Using administrative data from the Netherlands, I empirically illustrate this reappraisal of heritability studies by estimating two different gene-environment interactions in test scores from a high-stakes national educational achievement test.

Keywords: Education, Equality of opportunity, Inequality, Heritability, Public policy

JEL codes: I24, I38, J18

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Heritability studies aim to quantify the extent to which inter-individual differences in a trait or observable characteristic can be explained by genetic differences in a certain population (Jencks 1980; Visscher, Hill and Wray 2008). For instance, heritability estimates of human height are typically above 80% in developed countries, with differences in environmental circumstances such as living standards explaining the remaining part of the variation (Polderman et al 2015; Silventoinen et al 2003). A steadily increasing number of studies shows that economic preferences (Benjamin et al. 2012; Cesarini et al. 2009a; 2009b; Wallace et al. 2007) and socio-economic status indicators are also partly heritable (Benjamin et al. 2012; Bingley, Cappellari and Tatsiramos 2023; Branigan, McCallum and Freese 2013; Hyytinen et al. 2019; Polderman et al. 2015; Silventoinen et al. 2020; Van der Loos et al. 2013).

The main reason provided for estimating the heritability of preferences and outcomes in economics is to quantify descriptively the relative importance of genetic and environmental sources of socio-economic inequalities, especially for their roles in explaining the proliferation of these inequalities through generations (Harden 2021; Mogstad and Torsvik 2023; Taubman 1981). Nevertheless, debates about the policy relevance and information value of heritability estimates have been fierce and heated in economics from the onset. Early estimates of the heritability of educational attainment and income (Behrman and Taubman 1976; Taubman 1976a; 1976b) were argued to "serve no worthwhile purpose" (Goldberger 1979, 346) and set aside with the suggestion that "the entire effort is misguided" (Goldberger 1978, 960) and that "no scientific purpose is served by the flood of heritability estimates" (Kamin and Goldberger 2002, 83). Nevertheless, in sympathy with Goldberger's position, thirty years after the Taubman-Goldberger debate (Goldberger 1979; Taubman 1981) Manski sighed: "The work goes on…", "…but I do not know why" (Manski 2011, 89).

Goldberger's critique is that heritability estimates do not carry information regarding the possible impact of public policy on outcomes, and he vividly illustrates this argument with his now famous eyeglasses example (Goldberger 1979, Manski 2011): Even if myopia would be fully genetically determined, policy makers still have the possibility to intervene by distributing glasses and by doing so remediate the problem of bad eyesight. Therefore, "Heritability analysis is just not a guide for policy, not a short-cut around the detailed cost-benefit analysis required for each specific policy proposal" (Goldberger 1979, 346). The eyeglasses example illustrates that heritability estimates are not informative regarding whether a policy intervention can change outcomes at the individual level, and that these estimates are only informative about the sources of inequalities in the environment from which the data were sampled (Hyytinen et al. 2019).

Mogstad and Torsvik (2023) recently pointed out that this limitation is not unique for heritability studies: “It is a general concern in empirical analysis that a parameter estimated on data from one population or in one environment may not generalize to other populations or to other environments” (Mogstad and Torsvik 2023, 351). In addition, information regarding the origins of a problem is also often not directly relevant to solving it. That genes influence myopia does not directly suggest eyeglasses as a remedy; the best way to avoid getting wet when it rains would be to stay home or bring an umbrella rather than trying to change the rainy weather (*cf.* Mogstad and Torsvik 2023, 351). Still, special caution seems needed (Harden 2021; Meyer et al. 2023), because “...although it may in essence be true that heritability estimates are “fundamentally uninformative” (Manski 2011), it may not be unimportant for policy in practice.” (Stenberg 2013, 204). Heritability estimates may have a bearing on the development of policy depending on how they are interpreted: “...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, or whether they believe the 60% reflects only small genetic differences whose correlations with environments blow up the heritability estimate.” (Stenberg 2013, 204). Heritability estimates cannot be used to discriminate between these two explanations, and this is exactly why Goldberger discouraged the conduct of heritability studies: “...heritability estimates serve no worthwhile purpose. One might view the calculations as a harmless exercise, rather akin to other quaint econometric activities like finding the bias to order $1/T^4$ of the k-class estimator in a structural equation with two right-hand side endogenous variables. Alternatively, one might recognize that the estimates are being put to just one use (however unintended and however unjustified), namely to discourage active socioeconomic policy by “demonstrating scientifically” that current inequalities are the inevitable dictates of nature. If so, the calculations are not harmless, and it may be a good idea to abandon the entire enterprise of estimating genetic variance components of socioeconomic achievement” (Goldberger 1979, 346).

Relatedly, although virtually all estimated parameters in empirical research are subject to misinterpretation and misuse (*cf.* Mogstad and Torsvik 2023), Goldberger’s proposal to abandon the conduct of heritability analyses is particularly worth considering given the controversial history of social and behavioral genetics (Bliss 2018). For example, heritability estimates of IQ have been misused to justify prejudice or to discriminate groups (Jensen 1969; Herrnstein 1971; Herrnstein and Murray 1994), and outdated and flawed speculations in the literature regarding a partial genetic cause of racial IQ gaps as well as decontextualized general findings on genetic influences are used today by White supremacists (Panofsky, Dasgupta and Iturriaga 2021). Such group comparisons fail to adequately acknowledge the profoundly different environments society has created for individuals of different minority groups (Harden 2021; Meyer et al. 2013). I wholeheartedly share the concern of potential misuse of heritability estimates, and in the present reappraisal of heritability studies for their policy relevance I caution that its practical relevance is constrained by the exogenous nature of the environmental condition being used in the comparison of heritability estimates. Therefore, I specifically stress that this study and the statistical approach put forward in it cannot be used to motivate the comparison of heritability estimates across ethnic groups.

Heritability studies not only estimate heritability, but also estimate the share of the variance in the analyzed outcome that can be attributed to environmental influences that are shared by siblings but which vary between families. Interestingly, irrespective of whether heritability estimates are deemed relevant for policy or not[1], the policy relevance of the estimate for this common or shared (family) environment component seems beyond discussion (Wolfram and Morris 2023). For example, Nielsen and Roos (2015) note “it reflects the potential effect on educational attainment of raising the quality of the most disadvantaged family environments to the level of the most advantaged one; it thus represents an upper bound on improvement in the trait achievable by policy intervention within the existing range of environmental variation” (539). This notion, although not receiving much attention in the economics literature[2], was arguably also the most interesting twist in the Taubman-Goldberger debate and where they eventually reached a common ground: While Taubman initially used to be concerned with the joint contribution of genes and the family environment to the variance in an outcome (“The total of family environmental and genetic effects will be labeled the family effect”; Taubman 1976b, 858), the debate with Goldberger made him reach the conclusion that “The decomposition is important for policy purposes, not in what it tells us about heritability but in what it tells us about the variation in (common) environment, which represents variation in opportunities” (Taubman 1981). Parents transmit to their children not only genes but also capital and social networks (Becker and Tomes 1979; Corak 2013) and equality of opportunity

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[1] That genes are an important source of inequalities, and for instance matter for intergenerational transmissions (*cf.* Becker and Tomes, 1979; 1986), is not a discussion point in the Taubman-Goldberger debate.

[2] It is a rather prominent view in the sociology literature, see for instance also Engzell and Tropf (2019) and Knigge, Maas, Stienstra, De Zeeuw and Boomsma (2022). In the economics literature, this view is hardly put forward. For instance, surprisingly, it is not discussed in the review by Manski (2011); its mentioning in a footnote only by Hyytinen et al. (2013) seems to prove the point since this footnote is no longer present in the eventual journal publication (Hyytinen et al 2019) of this working paper.

is more limited in a population where the family environment determines to a larger extent socio-economic success (Roemer 1998). Indeed, Goldberger (1979) also notes that “we have policies that merely equalize opportunity: they reduce the common environment variance”. While the size of the common environment component is not informative about how easy or cost-effective it would be to eliminate inequalities by equalizing opportunities, it bears repetition in the economics literature that heritability studies can deliver an indicator for the presence of inequality of opportunity. However, as Goldberger (1979) already noticed, it is equally true that inequality reductions can also be achieved through different means than intervening in the family environment; For instance, “resources might be redistributed to those whose backgrounds put them at a disadvantage in the labour market” (Goldberger 1979, 345).

Thus, merely using the estimate of the common environment as a static indicator of inequality of opportunity would still be subject to Goldberger’s critique that this estimate about “what is” does not tell anything about “what could be” (*cf.* Hyytinen 2019). In this study, I develop the literature by proposing that a controlled comparison of heritability estimates and of the common (family) environment variance component can be informative regarding the ex-post evaluation of policy effects: While it can be considered relevant to understand for a given population how much of the variance in a trait is explained by genetic differences and common environmental differences (Taubman 1981; Harden 2021), exogenous sources of variation can be used to estimate the extent to which the contribution of genes and the common environment to inter-individual differences changes due to (policy-shaped) environmental conditions. For instance, stretching the eyeglasses example (Goldberger 1979; Manski 2011), estimating the sources of variation in effective sight before and after the extension of basic health insurance to cover reimbursement for eyeglasses could potentially show that the impact of familial resources (i.e., the common environment component) has decreased.

I thus offer a way out of the Taubman-Goldberger controversy, by plotting their respective positions in a more general framework in which a policy-induced environmental change can be modelled. It does justice to the agreement reached in the Taubman-Goldberger debate (Taubman 1981; Goldberger 1979) concerning the distinction between genes and the family environment, as well as to Goldbergers’s and Manski’s (2011) position that the size of these components does net tell us anything about the possible impact of policy measures on an outcome. However, the change in these components across exogenous environments can be informative regarding the evolvement of sources of inequality in a population. To assess this change, I introduce the gene-environment (G×E) interaction model for twin studies (Purcell 2002), an extension of the classical twin study (Knopik et al. 2018), in the economics literature.

Admittedly, this is not the introduction of an ex-ante "cost-benefit analysis required for each specific policy proposal" (Golberger 1979), but it does entail the introduction of a tool to evaluate whether the contribution of factors beyond an individual's control (genes and the family background) to inter-individual differences has changed. Although this model captures only one specific measure of inequality of opportunity (*cf.* Ramos and Van de Gaer 2016), with equality of opportunity currently being a central concern in society I believe this assessment to be relevant and timely.

I start by providing simulations based on the model developed by Purcell (2002) for G×E analysis to illustrate that policy-informative heritability studies need to consider more than just the standardized variance components (i.e., the *proportions* of variance explained by additive genetic effects A (or h^2), common environment effects C (or c^2) and unique environment effects E (or e^2)) only. These relative proportions must be interpreted in conjunction with an assessment of the change in the unstandardized variance components because environments can influence the variance in the outcome variable significantly.[3] Vice versa, solely assessing the unstandardized variance components does not suffice as a change in importance can only be assessed in a relative fashion (i.e., by the standardized variance components). To empirically illustrate this methodological insight further, I draw on large-scale administrative data from the Netherlands to estimate the heritability of test scores by year of test (*cf.* Heath et al. 1985) and for pupils born just before and after the cut-off of October 1 used to assign them in school classes. This policy-set rule causes exogenous variation in relative age within a school class, with longer-lasting effects on educational outcomes, non-cognitive outcomes, and earnings, amongst others (Bedard and Dhuey 2006; Black, Devereux and Salvanes 2011; Cornelissen and Dustmann 2019; Oosterbeek, Ter Meulen and Van der Klaauw 2021; Van Aalst and Van Tubergen 2021).

In sum, I propose that heritability studies can be used to assess the origins of socio-economic inequalities ("what is") as well as to assess changes in the (relative) importance of such origins ("what could be") by exploiting exogenous sources of variation. That is, I revisit the Taubman-Goldberger debate by reappraising heritability studies in the economics literature for their ability to deliver an indicator for the presence and the development of inequality of opportunity and as a tool to ex-post (i.e., after the change in the environment having occurred) assess the impact of specific policy conditions on how sources of variation contribute to inter-

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[3] For this reason, stratification of a sample by environments into subsamples generally prohibits a proper comparison of (standardized) variance components obtained within these subsamples.

individual differences. In doing so, I amend the already rich toolbox of econometric methods for policy evaluations (Abadie and Cattaneo 2018) with Purcell's (2002) gene-environment interaction model. Empirically, I show that inequalities in test scores from a Dutch high-stakes national test resulting from factors beyond an individual's control (i.e., genes and the family environment) have decreased over the years 2006-2021. However, these factors are more important in explaining test scores for relatively younger pupils in a school class.

I. Methods

The intergenerational mobility literature often uses the Becker-Tomes (Becker and Tomes 1979; 1986) model of human capital transmission to estimate to what extent differences in observed outcomes across individuals can be attributed to particular parental characteristics. The overall family background, however, can play a role in shaping outcomes of children through various genetic and environmental influences. The relative contribution of such factors can be evaluated by exploiting different levels of environmental and genetic relatedness between family members (Kilpi-Jakonen et al. 2024). For instance, Solon (1999) posits a basic model where outcome Y_{ij} of individual j in family i is decomposed into a shared family component a_i and an individual-specific component b_{ij}:

$$Y_{ij} = a_i + b_{ij}. \tag{1}$$

In a sample of full siblings, since a_i and b_{ij} are assumed to be independent, this implies that the variance in outcome Y (σ_Y^2) can be decomposed into a shared family variance component σ_a^2, and a sibling specific variance component driven by idiosyncratic factors σ_b^2. Therefore, the correlation in outcomes between sibling j and j' provides the proportion of the variance that is due to shared factors:

$$Corr(Y_{ij}, Y_{ij'}) = \frac{Cov(Y_{ij}, Y_{ij'})}{\sigma_Y^2} = \frac{\sigma_a^2}{\sigma_a^2 + \sigma_b^2}\,. \tag{2}$$

Despite the important advances and broad range of empirical estimates in the intergenerational mobility literature, recent work still emphasizes the need to further unpack the black box of family background (Björklund and Jäntti 2020; Cholli and Durlauf 2022; Mogstad and Torsvik 2023). Following the conventional notation in the behavioural genetics literature (Knopik et al 2018), Equation 3 indeed decomposes the shared family component (a_i) and individual-specific component (b_{ij}) in Equation 1 into additive genetic influences (A), common (shared) environmental influences (C), and unique (non-shared) environmental influences (E). Here, the common environment captures all family, community, social or neighbourhood factors that are shared by a particular type of relative, and that are independent of the genetic factors. The unique environmental component is effectively the residual term capturing everything not explained by the genetic and common environmental factors:

$$Y = aA + cC + eE. \tag{3}$$

Under the assumption that the genetic and environmental influences are independent, the variance σ_Y^2 of outcome Y equals:

$$\sigma_Y^2 = a^2\sigma_A^2 + c^2\sigma_C^2 + e^2\sigma_E^2. \tag{4}$$

The fraction of the variance in outcome Y that can be attributed to the variance in additive genetics effects, $\frac{a^2}{a^2+c^2+e^2}$, equals the heritability h^2 of outcome Y. By comparing covariances between relatives with varying degrees of genetic relatedness, the parameters in the model can be estimated. The covariance in Y between relative 1 and 2 equals:

$$\sigma_{Y_1Y_2} = a^2\sigma_{A_1A_2} + c^2\sigma_{C_1C_2}. \tag{5}$$

There is no unique environmental variance component in Equation 5 because the covariance between pairs of individuals for this component is assumed to be zero. Further assumptions about the genetic ($\sigma_{A_1A_2}$) and environmental ($\sigma_{C_1C_2}$) covariances between related individuals such as siblings, adoptive siblings, parent-child pairs, or other combinations are needed to estimate a^2 and c^2. Still, the most popular application of the model is the classical twin study in which genetically identical monozygotic (MZ) twins (for which $\sigma_{A_1A_2} = 1$) are compared to dizygotic (DZ) twins who are as genetically equal as regular siblings. Under random mating of parents, an assumption that can be easily relaxed in the model (Knopik et al. 2018), $\sigma_{A_1A_2} = 0.5$ for DZ twins. In addition, the classical twin study relies on the equal environments assumption, meaning that both twin types are assumed to be influenced by the common environment to the same extent. This assumption, implying $\sigma_{C_1C_2} = 1$ for both MZ and DZ twins, has been criticized but appears to hold sufficiently broadly for educational outcomes to validate the model (Derks, Doland and Boomsma 2006; Evans and Martin 2000; Felson 2014; Mönkediek 2021).[4] The equal environments assumption makes that, in the classical twin model, difference in outcomes covariances between MZ and DZ twins can only result from

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[4] By complementing the classical twin model with information on the twins' spouses and children, Bingley, Cappellari and Tatsiramos (2023) test and reject the equal environments assumption for educational attainment in a Danish twin sample. They find that MZ twins share their environments far more than DZ twins, and the differential correlation in educational attainment between the two twin types loads onto the genetic variance component suggesting this component to be overestimated in the classical twin model. This is an intriguing result, although this extension of the classical twin model could only be identified by imposing additional moment restrictions from between generations, i.e., covariances between parents and children and between twins (or co-parents) and their niblings. In my current reappraisal of heritability studies, though, the size of the estimated variance components is of lesser importance (*cf.* Goldberger's critique) than the change in variance components.

genetic differences. Substituting for the assumptions regarding genetic and environmental similarity of MZ and DZ twins into Equation 5 allows for a straightforward estimation of the variance components A and C (and by implication of E). For this reason, the classical twin study is often called the ACE model (Knopik et al. 2018). This model is visualized in Figure 1a.

A large number of twin studies has shown that genes significantly contribute to variation in important indicators of social status such as educational attainment, occupational status, and income (Polderman et al. 2015). Nevertheless, researchers increasingly acknowledge that the assumption that genetics and environmental factors operate additively is too strong (Plomin 1977; Turkheimer 2000; Rutter 2006) as complex interplays between genetic and environmental factors cannot be disregarded (Biroli et al. 2022; Heckman 2007; Hunter 2005). The ACE-M twin model, as developed by Purcell (2002), allows for moderation of the genetic, common environmental, and unique environmental effect by an observed environmental factor M:

$$Y = (a + a'M)A + (c + c'M)C + (e + e'M)E. \tag{6}$$

This model, shown in Figure 1b, simplifies to the classical twin model in case a', c', and e' are all equal to 0. The linear moderation in the model may subsequentially have bearing on the *relative* proportions of the variance components A, C, and E. For instance, the proportion of the variance explained by additive genetic effects (heritability h^2) varies as a function of M:

$$h^2 = \frac{(a + a'M)^2}{(a + a'M)^2 + (c + c'M)^2 + (e + e'M)^2}. \tag{7}$$

A famous application of the ACE-M model concerns the analysis of how parental socio-economic status moderates the heritability of IQ (Turkheimer et al. 2003), with the results suggesting that the relative impact of genes on IQ is stronger in richer families while the relative impact of the common environment is stronger in poorer families. Nevertheless, the results obtained using the ACE-M are prone to bias if moderator M itself is heritable (Van der Sluis, Posthuma and Dolan 2011): Endogenous sorting into environmental categories preempts the comparison of variance components across categories. For this reason, in my revisiting of the Taubman-Goldberger debate I stress the exogeneity of M.

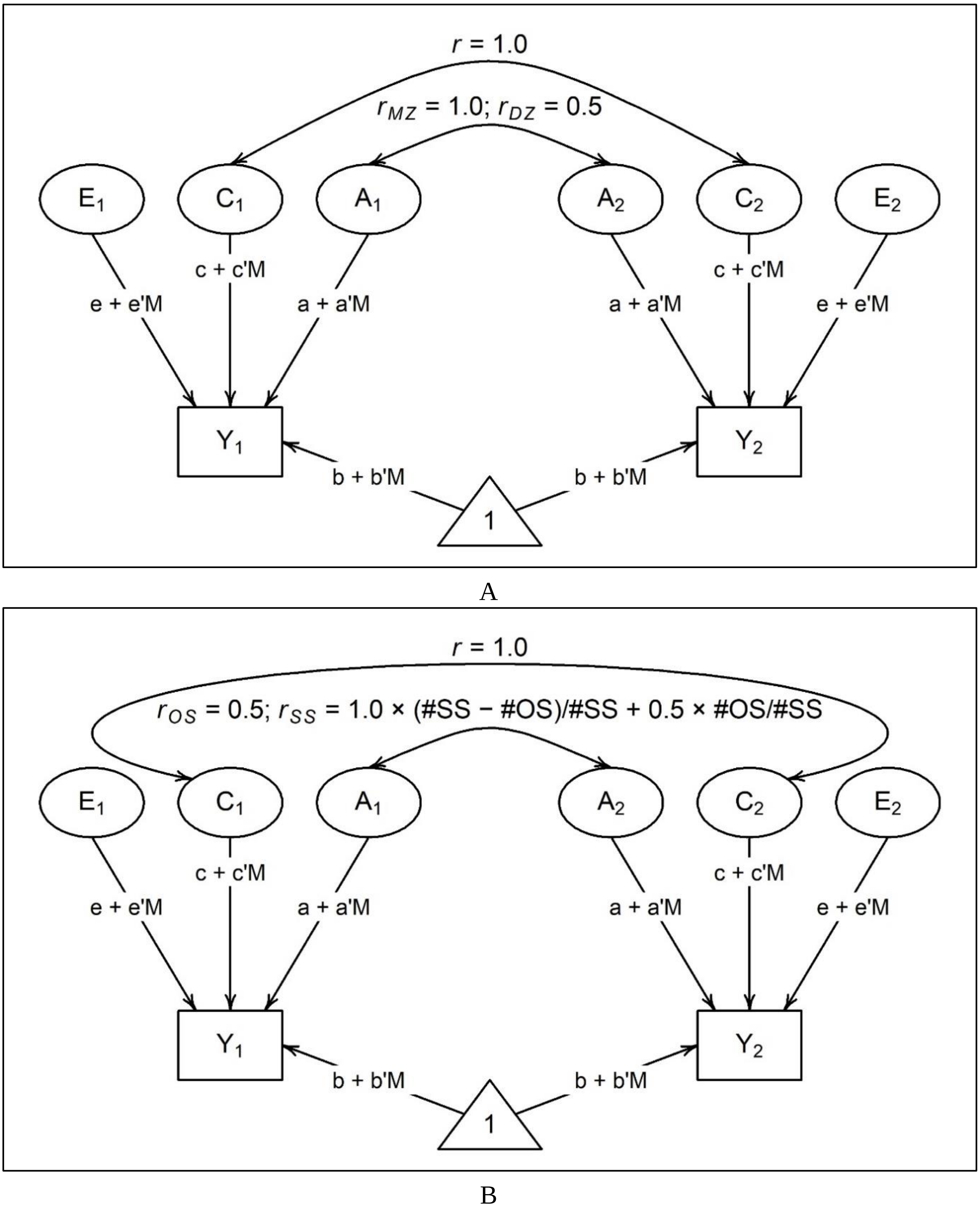


Figure 1. The ACE–M twin model for gene-environment interaction analysis (Purcell 2002). Observed outcomes Y_1 and Y_2 for twin 1 and twin 2, respectively, are explained by (1) a constant term capturing the means, (*A*) additive genetic effects, (*C*) common environmental effects, and (*E*) unique environmental effects. In Panel A, the structural model assumes a genetic correlation $r_{MZ} = 1$ for monozygotic (MZ) twins and $r_{DZ} = 0.5$ for dizygotic (DZ) twins; the correlation between common environmental effects equals $r = 1$ for both types of twins. In Panel B, the structural model assumes a genetic correlation $r_{OS} = 0.5$ for opposite-sex (OS) twins and $r_{DZ} = 1.0\times(\#SS–\#OS)/\#SS + 0.5\times\#OS/\#SS$ for same-sex (SS) twins; the correlation between common environmental effects equals $r = 1$ for both types of twins.

Another significant challenge to properly implement the ACE-M model is that it requires large samples to be statistically well-powered, because most twin registries are relatively small (Hur et al. 2019). An adaption of the ACE-M model, however, facilitates the use of large-scale administrative data for it. Based on parental information and birth dates, it is relatively straightforward to identify twins within administrative data. National registries typically do not contain information on the zygosity of twins, but opposite-sex (OS) twins are all dizygotic. By relying on the assumption that same-sex (SS) and OS twins are equally alike among DZ twins, the average genetic correlation among SS twins can be approximated by 1.0×(#SS–#OS)/#SS + 0.5×#OS/#SS (Atav, Rietveld and Van Kippersluis 2023; Calvin et al. 2012; Erola et al. 2022; Figlio et al. 2017; Pokropek and Sikora, 2015; Scarr-Salapatek 1971). That is, SS twins can be either monozygotic or dizygotic but by assuming a 1:1 sex ratio the average genetic relatedness within the set of SS twins can be approximated. At the expense of some statistical power, the comparison between OS and SS twins (Figure 1b) gives the same results as the comparison between MZ and DZ twins (Figure 1a). Still, the comparison between OS and SS twins requires the assumption of equal heritability across sexes, and thus a stronger equal environments assumption, i.e., SS twins and OS twins are influenced by the common (family) environment to the same extent. Moreover, with the sex ratio needed for the determination of genetic relatedness, analyses cannot be stratified by sex.

The parameters (paths) in the ACE-M model can be estimated using maximum likelihood as implemented in the “umxGxE” function *R* package “umx” (Bates, Neale and Maes 2019). In line with the model visualized in Figure 1b, I adjusted this function for the comparison between OS and SS twins. An advantage of the maximum likelihood approach is that nested models in which one of more paths in the model are dropped (e.g., all moderating paths) can be readily compared in terms of fit with the data. By looking at the significance of the change in fit, the most parsimonious model given the fit between model and data can be determined.

II. Results

This section consists of two parts. First, I use simulations to present various forms of G×E interplay that can be detected using the ACE-M model. Thereafter, I use data from Statistics Netherlands to estimate G×E interplays in test scores from a Dutch national educational achievement test.

A. *Simulations*

The ACE-M model (Purcell 2002) allows for non-equal variances at different levels of the moderating variable, which is important because the absolute magnitude of genetic effects and common environment effects may change and not only their proportional contribution to inter-individual differences. With the environmental variable also allowing to moderate the effects of the common and unique environment, it is also possible that variance components change as function of the moderator while the variance remains equal at different levels of the moderating variable. Therefore, to properly assess G×E interplay, one cannot suffice by only looking at the change in standardized components because from these it is not clear which (i.e., the genetic, common environmental, or unique environmental) effect is moderated. However, one can also not suffice by looking at the raw variance components only, because a change in importance can only be assessed in a relative fashion. Therefore, both raw and standardized variance components need to be evaluated in conjunction. The statistical validity of the ACE-M has been thoroughly studied (Purcell, 2002), but it is worth showcasing four main scenarios. Together, these scenarios illustrate why both raw and standardized variance components need to be evaluated in the model. Simulations have been conducted using the "umx_make_TwinData" and "umxGxE" functions of the *R* package "umx" (Bates, Neale and Maes 2019), and are based on 100,000 MZ and DZ twin pairs in each environment.

In the first scenario moderator *M* does not impact the contribution of *A*, *C*, and *E* to inter-individual differences (Figure 2, Panel A). As a result, the relative proportions of *A*, *C*, and *E* (standardized moderation effects) do not depend on *M*. In the second scenario, moderator *M* impacts the raw moderation effects in the same proportional fashion (Figure 2, Panel B). As a result, despite changes in the raw variance components, the standardized moderation effects (i.e., the relative proportions of *A*, *C*, and *E*) do not depend on M. Together, the first and second scenario showcase that both standardized and unstandardized variance components need to be assessed when analyzing interactions in twin models. In the third scenario (Figure 2, Panel C), *M* does not moderate the impact of *A* but does so for *C* and *E*. With the contribution of *A* being constant over *M*, this scenario illustrates that a proper assessment of interactions in twin models comprises all three variance components simultaneously. This is further illustrated in the fourth scenario (Figure 2, Panel D). Here, *M* impact both the raw and standardized variance components of all three sources of variation in the model.

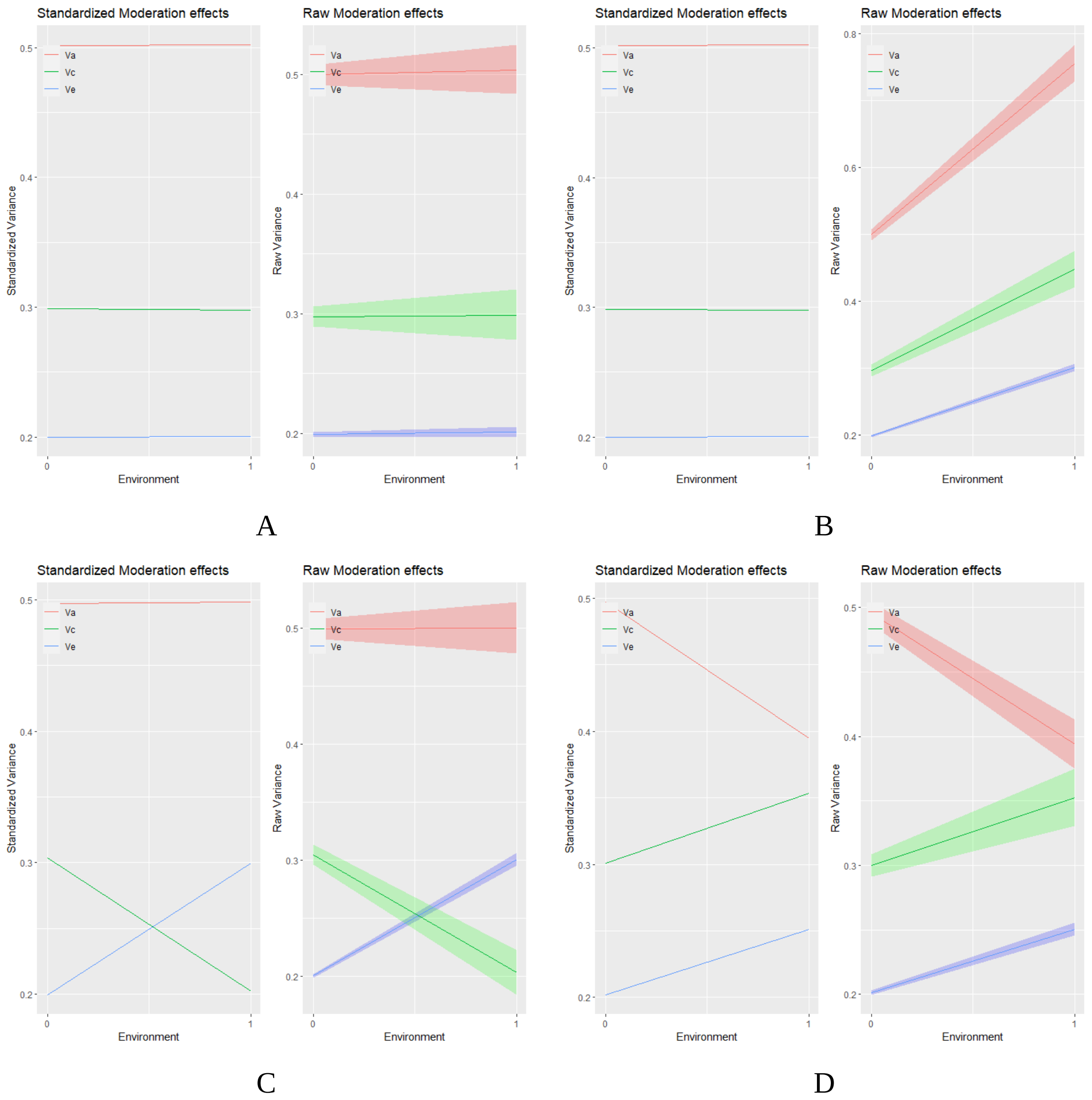
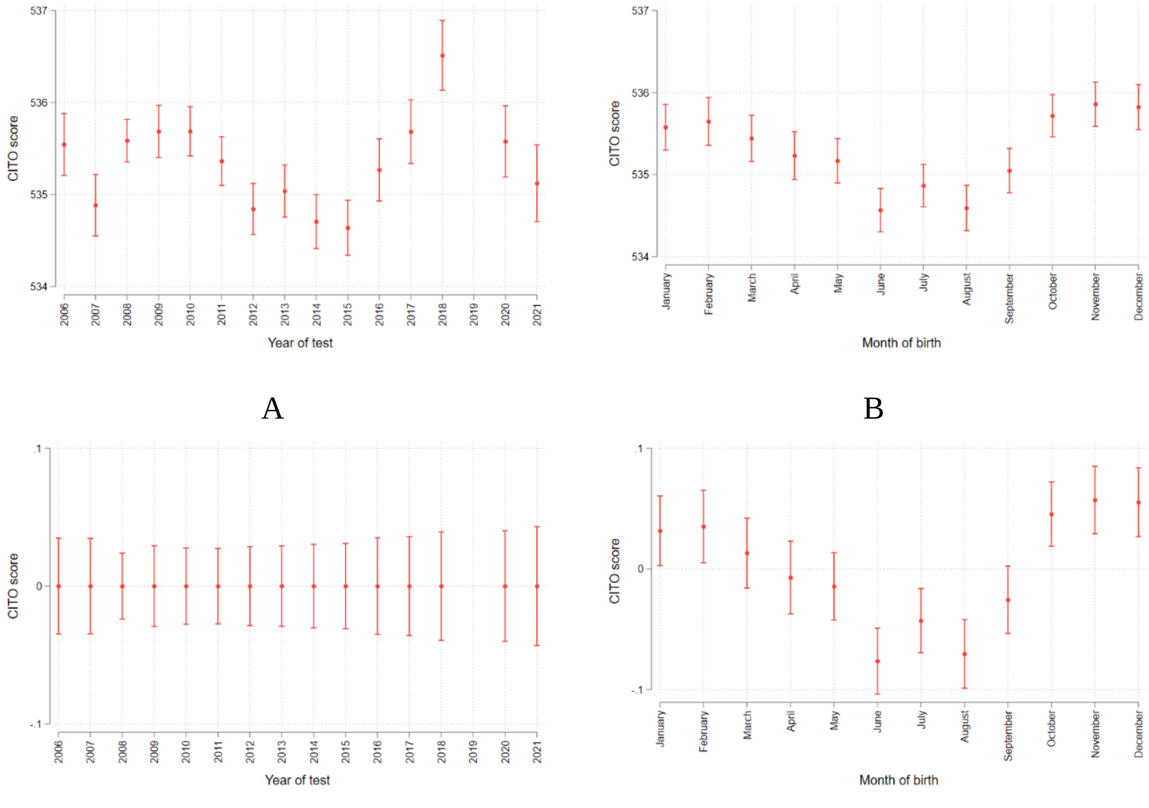


Figure 2. Results of simulations based on the ACE-M twin model for gene-environment interaction analysis (four scenarios). Moderator M (the environment on the x-axis) takes value 0 or 1 depending on the twin pair being affected by an exogenous environmental change or not. Error bars represent 95% confidence intervals.

B. *Empirical results*

In this part, I draw on administrative data on the universe of the Dutch population from Statistics Netherlands (CBS). In these data, I determine twins as individuals sharing the same two parents and being born in the same year and the same month. The outcome variable is the score on a nation-wide standardized educational performance test (CITO) around the age of 12 during the last year of primary education. This test is administered on three consecutive days in January or February. In combination with teacher advice, the result of this test is used to determine tracking placement in secondary school in the Netherlands. The CITO-elementary test consists of multiple-choice items assessing four different intellectual skills: Language, Mathematics, Information Processing, and World Orientation. Total performance is a standardized score between 501 and 550 (Van Boxtel, Engelen and De Wijs 2010). Raw test scores are available for the years 2006-2021. In total, I have complete data on 28,862 twin pairs (57,724 individuals), from which 10,409 are OS and 18,453 are SS twin pairs. Figure 3 shows descriptive statistics by year of test and month of birth, both before and after standardization by sex and year of test to mean 0 and standard deviation 1.[5] It can be seen (Figure 3, Panel A) that the mean of the raw CITO scores varies over the years, although not with a clear pattern. Data from the year 2019 are missing, because CITO tests were cancelled in that school year due to COVID-19 restrictions. Regarding month of birth (Figure 3, Panel B), CITO scores tend to decrease approximately linearly from October-September. The mean difference in raw CITO scores between these two months is 0.669 (p = 0.0005). Because of the variation over years and the comparison of OS and SS twins, I use the test scores standardized by sex and year of test in the heritability analyses.



A

B

C

D

Figure 3. Mean CITO scores (raw scores in Panels A and B and standardized scores by sex and year of test in Panels C and D). Error bars represent 95% confidence intervals.

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[5] It is possible, but not common, in Dutch primary schools to skip or re-take a class, depending on ability and teacher advice. For this reason, year of test is almost perfectly correlated with year of birth (r = 0.990).

The comparison of the standardized CITO scores between OS and SS twins results into standardized variance component estimates of 0.84 (*A*), 0.02 (*C*), and 0.14 (*E*). The estimate of the additive genetic components appears high, but is in line with earlier findings for CITO scores as found in the sample of the Dutch Twin Registry using the classical twin study design. That is, Bartels, Van Beijsterveldt and Boomsma (2009) find that 78–84% of the variance in CITO scores is explained by additive genetic effects while the remaining variance is accounted for by nonshared environmental effects. Similarly, De Vries et al. (2021) find estimates of 0.80 (*A*), 0.00 (*C*), and 0.20 (*E*). De Zeeuw et al. (2016), as I do, find some evidence for an impact of the shared family environment: 0.74 (*A*), 0.08 (*C*), and 0.18 (*E*). In addition, they find that there is no evidence for gender differences in the underlying etiology of the CITO scores, an important assumption in my comparison of OS and SS twins. Finally, in a much smaller and likely more selected sample, Bartels, Rietveld, Van Baal and Boomsma (2002) find estimates of 0.57 (*A*), 0.27 (*C*), and 0.16 (*E*). Also in this study, no evidence of sex-differences in the heritability of CITO scores is found.

Next, I investigate moderation by year of test to assess the presence of a linear trend in the development of inequality and sources of inequality over time. Panel A of Figure 4 shows that while the unstandardized variance component *A* increases from 0.81 to 0.86 over the course of the years 2006-2021, this increase is hardly visible in its standardized variance component (0.829 to 0.835) due to the overall increase in the outcome variance from 0.98 to 1.03 over the analyzed period. For *C* and *E*, changes are visible both in the unstandardized and standardized variance components. Importantly, a loglikelihood-ratio test reveals that dropping the moderating paths from the model results in a significant drop in model fit ($\chi^2 = 13.38$, $p = 0.004$). Thus, the inclusion of the moderating paths increases the fit between the data and the model. From the standardized variance components, I conclude that inequalities due to factors beyond an individual’s control (i.e., *A* and *C*) have decreased from 0.87 to 0.85. The common environment particularly has become less important in explaining the variance in CITO test scores (0.043 vs. 0.015). However, I note that further loglikelihood-ratio tests suggest that the most parsimonious model fitting the data equally good as the full model is a model without main effect and interaction effect for *C* ($\chi^2 = 1.63$, $p = 0.444$). The change in fit is, however, small.[6]

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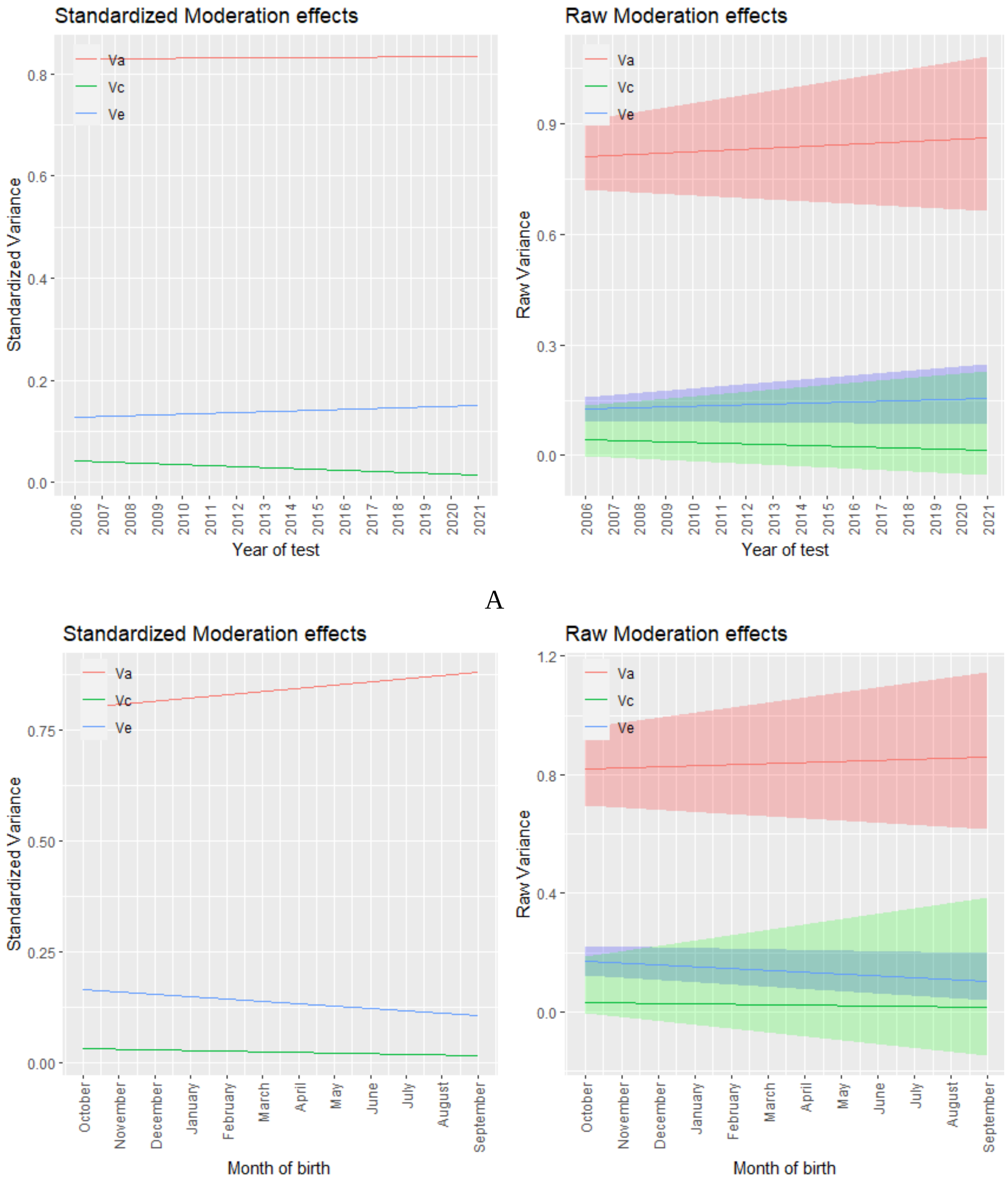
[6] Moreover, it should be considered that the loglikelihood-ratio test depends on the size of the analysis sample.



A

B

Figure 4. Results of the ACE-M model comparing CITO test scores (standardized by sex and year of test) of OS and SS twins. Panel A: Moderation by year of test; Panel B: Moderation by month of birth. Error bars represent 95% confidence intervals.

Panel B of Figure 4 shows the ACE-M model estimates for moderation by month of birth. Because of the October 1 cut-off used to assign pupils in school classes, the month of birth variable is defined as October = 1, November = 2, … , September = 12. The slopes in the unstandardized subfigure line up well with the slopes in the standardized subfigure. Again, I find using a loglikelihood-ratio test that dropping the moderating paths from the model results in a significant drop in model fit ($\chi^2 = 25.82$, $p < 0.001$). The inclusion of the moderating paths thus increases the fit between the data and the model. The standardized variance components suggest that factors beyond an individual's control (i.e., *A* and *C*) contributing to CITO test scores differences are more important (0.83 vs. 0.89) for relatively younger pupils in a school class. The standardized variance of the additive genetic factor differs particularly (0.80 vs. 0.88). Further loglikelihood-ratio tests suggest that the most parsimonious model fitting the data equally good as the full model is a model without main effect and interaction effect for *C* ($\chi^2 = 0.89$, $p = 0.640$). Again, the change in fit is negligible. In total, the results suggest that factors beyond the pupil's control (genes and the family environment) are more important in explaining test scores for relatively younger pupils in a school class, questioning equality of opportunity.

III. Conclusion

My revisiting of the Taubman-Goldberger controversy and reappraisal of heritability studies is a response to Manski's sigh why heritability studies are still being conducted (Manski 2011). Heritability studies can offer a description of the relative importance of genetic and environmental sources of socio-economic inequalities, but with Goldberger (1979) and Manski (2011) I agree that plain heritability estimates do not carry information value for public policy. The eyeglasses example keeps proving this point. However, going beyond the mere estimation of heritability, I argue for a quasi-controlled comparison of heritability estimates in the context of a G×E interplay model. By drawing on the ACE-M model for G×E interplay as developed Purcell (2002), I show that differences attributable to factors beyond an individual's control (genes, and especially the family environment) can carry information value regarding the evolvement of inequality of opportunity. In my synthesis, I do justice to the agreement reached in the Taubman-Goldberger debate (Taubman 1981; Goldberger 1979) concerning the distinction between genes and the family environment. Moreover, fully in line with Goldbergers's and Manski's position, I abstain from "claims of "more important" or "less important"" (of genes vs. the environment) that, indeed, serve "no worthwhile purpose" (Manski 2011, 92). However, I do argue that the estimates heritability studies provide can carry information in their comparison with each other across exogenously determined (policy-shaped) environments. In doing so, changes in the (standardized) variance components can be informative regarding the evolvement of sources of inequalities in a population and the proliferation of inequalities through generations (Atav, Rietveld and Van Kippersluis 2023; Mogstad and Torsvik 2023).

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