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Variation in the Heritability of Educational Attainment: An International Meta-Analysis

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Abstract

To assess heterogeneity in the influence of genetic variation on educational attainment across environmental contexts, the authors present a meta-analysis of heritability estimates in 15 samples and 34 subgroups differing by nationality, sex, and birth cohort. They find that heritability, shared environment, and unshared environment each explain a substantial percentage of the variance in attainment across all countries, with between-sample heterogeneity in all three variance components. Although they observe only meager differences in the total family effect by cohort or sex, they observe large cohort and sex differences in the composition of the family effect, consistent with a history of higher heritability of educational attainment for males and for individuals born in the latter half of the 20th century. Heritability also varies significantly by nation, with the direction of variation specific by sample. They find a markedly larger impact of shared environment on attainment than has been found for other social outcomes, with the percent of variation in attainment attributable to shared environment exceeding the percent attributable to heritability in one-third of the studies in our sample. Their findings demonstrate the heritability of educational attainment to be environmentally contingent, affirm the widespread and enduring role of shared environment in determining ultimate socioeconomic attainment, and emphasize the importance of considering behavioral genetics techniques in models of social mobility.

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Variation in Heritability of Educational Attainment: An International Meta-Analysis

Although it is now taken for granted that many physiological outcomes are influenced by genetic differences, a number of studies of heritability over the last three decades have considered behaviors and attitudes once thought to be primarily social, ranging from income to likelihood of divorce (Behrman, Taubman et al. 1977; McGue and Lykken 1992). In sociology, the limited research in this area has focused mainly on identifying social variables that alter the effect of genetics on social outcomes, contradicting notions of a 'nature-versus-nurture' dichotomy in which genes and environment represent distinct causal explanations (e.g. Boardman, Saint Onge et al. 2008; Guo and Stearns 2002; Rowe et al. 1999). In this work, authors have suggested that estimates of the percentage of variation in social outcomes explained by genetic and environmental differences are likely to be context-specific, varying systematically across different social conditions, policy environments, or subgroups of the population (Jencks 1980; Boardman 2009). For scholars of social inequality, these findings are particularly meaningful, as they suggest that models of variation in social outcomes that take into account only social predictors are missing an integral part of the story (Eckland 1967; Freese 2008).

Education was an early domain of interest among researchers assessing the heritability of social outcomes, primarily using test score data to contribute to long-standing debates on the genetic basis of intelligence or achievement (e.g. Bouchard and McGue 1981; Devlin, Daniels et al. 1997; Guo and Stearns 2002; Nielsen 2006).[1] Studies of the heritability of years of education attained, on the other hand, are relatively few in number (Heath et al. 1985; Behrman and Taubman 1989; Silventoinen, Krueger et al. 2004), despite attainment being a common operationalization of

[1]Also see Feldman and Lewontin (1975) and Goldberger (1978) for two early critiques of the use of heritability estimates of intelligence and other outcomes.

educational performance across many fields and a long-standing outcome of interest in research on social mobility. While test scores are a socially contingent outcome, attainment is arguably even more so, under the presumption that the number of years of education one completes reflects not just cumulative achievement, but a vast range of social factors likely to influence school experiences and continuation decisions through pathways other than academic performance. In this study, we address this omission with a meta-analysis of the largest set of heritability estimates of educational attainment yet assembled, using published and unpublished data ranging across ten countries and with birth years spanning more than a century.

Prior studies of the heritability of educational outcomes using micro-level data have identified a number of variables by which the estimated influence of genetic variation on performance varies, most commonly indicators of socioeconomic status (Guo and Stearns 2002; Rowe et al. 1999) or egalitarian policy initiatives (Heath et al. 1985). With few exceptions, these studies have been based on comparisons between subgroups in a single sample drawn from within a single country. Although still informative, this approach potentially complicates generalization beyond the environmental contexts of the original studies, as the social settings from which the individual samples were drawn will themselves encompass a vast array of relevant factors such as governmental structure, religious or racial heterogeneity, and levels of inequality and mobility on many social dimensions. A meta-analysis trades explanatory depth for historic or geographic breadth, allowing us to explore variation in average heritability of attainment across a broad range of samples as opposed to between subgroups in a single sample. While micro-level data is useful for capturing within-population variation in heritability by sample-specific characteristics such as individuals' relative poverty level, here we are uniquely able to evaluate the effect of larger-scale social categories such as nationality, and to assess the significance of sex and birth cohort across a vastly larger and more diverse respondent population than would be otherwise attainable.

Beyond identifying new social dimensions by which the heritability of attainment varies, the ability to compare estimates of heritability across sex, nation, and birth cohort makes this analysis particularly relevant in a social mobility context (e.g. Bjorklund, Jännti and Solon 2003; Diewald 2010). In the total family effect frequently calculated in studies of social mobility, the influence of genetic variation and family-level social influence are captured as a combined estimate (Hauser and Mossel 1985; Hauser and Wong 1989; Sieben and DeGraaf 2003). Although identifying differences in the family effect on attainment across varying social groups (Kuo and Hauser 1995; Kuo and Hauser 1997) and birth cohorts (Dronkers 1993; De Graaf and Huinink 1992; Smith and Cheung 1986; Hauser and Featherman 1974) has long been a topic of interest in the social mobility literature, few attempts have been made to identify group differences in the effect of family environment net of the effect of genetic relatedness. By comparing separate estimates of the impact of heritability and familial environment as calculated in our sample, we are able to observe not only nation, sex, and cohort differences in the total family effect on educational attainment, but also to detect differences in how that effect is decomposed into genetic and environmental components. Our findings emphasize the importance of calculating genetic effects separately from social family effects when estimating models of social mobility, as changes in the importance of home environment relative to genes appear to reflect group differences in mobility trends even while the sum total family effect remains stable.

Genetic and Environmental Influences on Educational Performance

The most conventional approach to assessing the percentage of variation in an outcome accounted for by the effects of genetics is the twin study, in which the difference in within-pair correlations between identical (monozygotic, MZ) and fraternal (dizygotic, DZ) twins are used to generate an estimate of heritability (Winerman 2004). This approach is predicated on the

assumption that higher correlations among MZ versus DZ twins can be attributed to MZ twins being genetically (nearly) identical, whereas DZ twins share only approximately half their genes by descent. The term *heritability* refers to the percentage of outcome variation accounted for by additive genetic variation,[2] while remaining variation in the outcome is attributed to two categories of environmental influence: the *shared environment*, those factors experienced similarly by twin pairs, and the *unshared environment*, those factors that individual twins experience uniquely.

In the case of educational outcomes, heritability estimates have been hypothesized to be lower for those individuals who experience greater constraints on educational opportunity, such as being raised in poverty, and higher for individuals not exposed to such constraints (Guo and Stearns 2002; Turkheimer et al. 2003). Although in past studies of the heritability of educational outcomes, higher heritability estimates have been frequently associated with higher attainment and achievement due to the particular aspects of environment considered, environmental constraints may also depress heritability by compelling individuals to attain at higher levels than they otherwise would.[3] One example of such a constraint would be the legal requirement to remain in school until a minimum age, whereby individuals otherwise inclined to attain fewer years of education may continue in school due to legal strictures.

The 'shared' or 'common' environment component reflects the proportion of variance in educational outcomes explained by all non-genetic sources of twin similarity in a phenotype,

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[2] Our heritability estimate captures narrow-sense heritability, or additive genetic effects only; for more on this, see Plomin, DeFries, McClearn and McGuffin (2001: 382). Although we are thus omitting dominant and epistatic variance from our estimate, research suggests that additive variance accounts for the majority, often close to 100%, of the total genetic variance for complex traits (Hill, Goddard, and Visscher 2008).

[3] Guo and Stearns (2002) and Rowe et al (1999) found that being raised by high-SES parents is associated with higher heritability of achievement, while Heath et al. (1985) found that liberal social and educational policies increased heritability of attainment. That heritability and educational outcomes were positively correlated in these instances does not suggest that heritability and educational outcomes will be always positively correlated.

including characteristics that twins experience similarly due to having been reared together. As we illustrate in table 1, across a wide range of social outcomes the percent of variation explained by this component has been consistently small, and nearly always smaller than the percent of variation explained by genes. The finding is so ubiquitous that Turkheimer (2004) coined it the 'second law of behavioral genetics': that "the environmental effect of being raised in the same family is substantially smaller than the genetic effect and is often close to zero" (161; also see Turkheimer 2000). This assumption poses a challenge for sociologists, as the effects of many social forces are ostensibly captured in the common environment estimate: characteristics of the family such as parental education, occupation, wealth, income, parenting style, and sibship size; within-population variation in race and ethnicity; characteristics of the neighborhood such as poverty level, access to schools and other institutional resources, crime, and so on.[4]

<TABLE 1 ABOUT HERE>

Variation not explained by genetic variation or by family environment is attributed to the effect of 'unshared' environment, which refers to individual-specific factors that influence twins to differ on an outcome of interest. With estimates of shared environment so consistently low in prior studies (table 1), unshared environment is typically expected to explain the vast majority of outcome variation not accounted for by genetic differences (Turkheimer 2004; Turkheimer 2000). An illness or accident that affects only one twin would be captured here, as would differences in birth weight, assigned teachers or peers. Although the sources of variance captured in the estimate of unshared environment are expected to be individual-specific and random, population differences

[4] Furthermore, since most policy initiatives are aimed at altering social factors experienced commonly by twins being reared together (Jencks 1980), some have suggested interpreting estimates of common environment as an upper bound on the extent to which policy manipulation can ameliorate inequality on a given social outcome (Rowe 1994). Others have suggested that this relationship is unclear, since some genetic disparities (e.g., poor eyesight) may be more easily addressed by policy than many social disparities (Goldberger 1978; Bjorklund, Jannti and Solon 2003; Jencks and Tach 2006; Manski 2011).

may reflect broad social trends that lead family members in a given society to vary more or less from one another on average. In a society where the philosophy regarding individuation of same-aged siblings leads to twins being deliberately placed in different classrooms, for example, one might expect a higher estimated influence of unshared environment than in societies in which twins are deliberately kept together in school.

In interpreting the shared and unshared environment components, an important distinction is made between the *objective* environment, including elements of the environment that can be catalogued objectively by an observer, and the *effective* environment, those elements of the environment that have some effect on one or both members of a twin pair (Turkheimer and Waldron 2000; Goldsmith 1993). An objective environmental condition experienced similarly by twins may still affect the two individuals differently, while objectively dissimilar environmental conditions may nonetheless produce identical effects. Turkheimer and Waldron (2000) pose the example of divorce: although twins reared together would be both objectively exposed to the divorce of their parents, it need not affect the two individuals in the same way. Our estimates are thus capturing *effective* shared and unshared environment, as we observe only actual outcomes, and not the objective conditions that shaped those outcomes.

The Present Study

In this study we consider all three variance components, assessing the extent to which the percentages of outcome variation in educational attainment attributable to genetic differences, shared environment, and unshared environment vary across samples differing by sex, birth cohort, and nation. We begin by estimating the degree of between-study heterogeneity in the percent of variance explained by each component among our full set of samples, which we expect to be high given the varying environments from which our samples are drawn. Where we find significant

between-study heterogeneity, we introduce our measures of sex, cohort, and nationality in order to assess the percent of variation accounted for by each of these sample characteristics.

Our expectation that sex and birth cohort will affect heritability of educational attainment is based on observed historical attainment trends. Although gender inequality in attainment declined over much of the twentieth century, men consistently achieved higher average rates of attainment in the majority of the nations in our sample, suggesting that men and women were subjected to different levels of social constraints with respect to educational continuation.[5][6] The attainment differential alone does not indicate which gender was subjected to greater constraints—whether men were pushed to progress in school farther than they otherwise might, or females were held back from progressing as far in school. While both may have been true to some extent, Heath et al.'s (1985) findings of sex differences in heritability between Norwegian twin cohorts educated before and after more liberal social and educational policies were introduced post-Second World War suggests that in Norway, it was men who experienced lower levels of environmental constraint. Although the policy change affected students of both sexes, for men this liberalization was associated with a decrease in the percent of variation explained by environmental factors and an increase in the percent of variation explained by heritability. For women the estimate of heritability remained relatively stable, and particularly in later cohorts, well below the heritability estimate for men.

[5] See Breen et al. (2009) for twentieth-century attainment trends in Italy, Great Britain, Sweden and Germany; Núñes (2001) on Spain; Alexander and Eckland (1974) and Everett et al. (2007) on the U.S.; and Booth and Kee (2009) on Australia.

[6] Gender differences will vary by the definition of attainment used. For example, although women in the U.S. had higher high school graduation rates than men through much of the twentieth century (Heckman and LaFontaine 2007), over the same period men continued on to post-high school education, completed 4-year colleges, and attained a higher mean number of years of education than women (Alexander and Eckland 1974; Everett et al. 2007; U.S. Census Bureau 2012). Since our outcome is ultimate attainment, it is the mean attainment rates that most interests us here.

If this pattern of greater social constraints on achievement for women and individuals born earlier in the twentieth century can be extrapolated across the countries and years from which our samples were drawn, we expect to see significant differences by sex and birth cohort in the influence of genetic variation on attainment, with the effect being larger for men and for later-cohort respondents. Sex and cohort differences in the percentage of variance in educational attainment explained by genetic versus shared environmental factors would make these estimates a useful tool for assessing social mobility, suggesting that an increase in the importance of common environment relative to genes can reflect a decrease in the opportunity for mobility even if the total family effect remains stable.

Net of sex and cohort, we expect that the vast number of differences between nations will also affect the level of social constraints that individuals experience with respect to educational attainment. Systems of government, extent of religious and ethnic diversity, and structure of public education are but a few examples of such differences, all of which are implicitly held constant in a single-nation study of variation in heritability. We anticipate that these differences will influence both magnitude and variation of our heritability estimates, leading to significant heterogeneity across the ten countries in our full sample. The geographic diversity of our samples leaves open the possibility that an observed effect by nation might reflect broader regional factors for which nation may serve as a proxy; we test for this possibility among a subgroup of only those samples drawn from Scandinavian countries, assessing whether heterogeneity of heritability estimates is significantly reduced within a set of nationally distinct but geographically proximate countries with a similar type of welfare state (Esping-Andersen 1990).

DATA AND METHODS

Despite the small number of published studies explicitly focused on the heritability of

educational attainment (Heath et al. 1985; Silventoinen, Krueger et al. 2004), twin correlations for level of education attained are often available in studies that consider education as a relevant factor affecting other outcomes of interest, primarily returns to schooling (Taubman 1976; Ashenfelter and Kruger 1994; Behrman, Taubman et al. 1977; Isacsson 1999; Bingley, Christensen et al. 2005; Miller, Mulvey et al 2006). Even when not available in published form, data on educational attainment is frequently collected by twin studies. We were able to locate fifteen twin samples in which correlations of educational attainment were either published or obtainable through direct correspondence with the researchers managing the data. Attainment was assessed as an ordinal scale, with values defined either by degree or by the number of years of education equivalent to a given degree. The number of categories differed by study and the relevant degrees differed by nation, reflecting differences in educational systems. The TwinsUK study (Moayyeri et al. 2012) stands as an exception since there was no categorical measure of attainment, but rather a measure of the age at which education was terminated.[7] Many of the samples are subdivided by sex or by age cohort, amounting to a total of thirty-four subgroups; we define subgroups by sex, birth cohort, and nationality. The data obtained are described in table 2, with sex, sample type, source, and birth years noted.

<TABLE 2 ABOUT HERE>

To conduct our search, we began with electronic databases, using search terms such as 'twin study,' 'twin registry,' 'educational attainment,' 'educational level,' and 'heritability.' We reviewed

[7] While differences in heritability by nation may also capture measurement differences across studies, structural variation between national education systems complicates efforts to distinguish scaling choices made by study investigators from actual between-country variation in the number of socially meaningful education categories. In all studies in our sample, both factors are likely relevant in determining the scales used. As such, we chose not to standardize across scales, since by collapsing categories one loses more information from societies that make a greater number of educational distinctions than from societies that make fewer distinctions. For one robustness check of the effect of measurement variation on the heterogeneity of heritability of attainment within a single country, see footnote 13.

the references of the relevant studies found, and pursued any additional studies that might have contained relevant data. We also conducted a similar search in the reverse direction: we sought out lists of twin registries internationally, and reviewed the citation lists on available registry websites to determine whether any studies had been published with the relevant educational statistics. Finally we expanded our list to include computations from yet unpublished sources, some of which we computed ourselves, and some of which we obtained through direct correspondence with study investigators.

We considered a sample to be "population-based" if respondents were either randomly selected from a defined population (e.g. MIDUS; AddHealth), or if all twins from a population census were automatically impaneled (e.g. the Italian Twin Registry; the Minnesota Twin Registry). The remaining samples consisted of individuals from within a particular geographic region who were solicited for survey participation on the basis of twinship (e.g. the SRI and TwinUK samples). We found one study based on a convenience sample of twins attending a twin convention (Ashenfelter and Krueger 1994); while the heritability estimate in this study did not differ substantively from others in our sample, the study was outside our exclusion criteria due to concerns regarding potential bias from the sampling method.

Although relevant twin registries exist in a number of additional non-English-speaking countries (e.g. Korea, Sri Lanka, China, Japan), we were not able to obtain data or estimates from these sources. As a result, our analysis is limited in international scope, spanning only the United States, Western Europe, and Australia. The published studies we found were also entirely from developed nations, although this is perhaps unsurprising, as the tally presented in Sung, Cho et al. (2006) indicates that only 2.7% of the papers published using twin study designs since 1950 have used data from low- or middle-income countries.

For each sample, we coded an indicator variable for sex and birth cohort, with our cohort

cutoff set at 1950. Male twins and twins born in the early cohort serve as the reference categories in all models. While nearly all of our samples are single-sex, the Swedish sample is mixed-sex[8]; likewise, while twenty-two of our samples are drawn from a relatively narrow range of birth years and can be classified reasonably into either pre-or post-1950 birth cohorts, the remaining samples were drawn from too wide a span of birth years to be readily classified. The variables for sex and cohort range between zero and one, with the values zero and one denoting homogeneous samples. A mixed sample is coded as the proportion of twin pairs in group one. The expectation maximization (EM) algorithm (Dempster, Laird, and Rubin 1977) is used to calculate maximum likelihood estimates when the proportion is missing for some samples.[9] As sex data is missing only for the Swedish sample, we expect that our results for sex should not be strongly affected, although the larger percentage of samples for which birth cohort was missing is important to note when interpreting findings. All computations were done in the statistical software package R (http://cran.r-project.org/) using functions written for this analysis.

Assessing Heritability: The Twin Study

Twin study methodology has evolved significantly over the past few decades, moving from simpler models to more complex versions that combine elements of twin, family, and population-based designs (Hatemi, Hibbing, et al. 2010; Neale and Maes 2004). While there are many methods of assessing heritability, here our estimates of heritability are calculated using the simple,

[8] We include the UK samples as mixed-sex estimates due to an insufficient number of men to run sex-by-cohort correlations separately, but the precise ratio of males to females in these estimates is known.

[9] The EM algorithm is a method for maximum likelihood estimation in the presence of latent variables. This approach does acknowledge the uncertainty in the value of missing cohort and sex data, which is reflected in the estimated sampling variance. As a robustness check, we also ran our models using list-wise deletion to handle missing data, and while significance drops off (as would be expected from the decrease in sample size), the magnitude of the coefficients on both sex and cohort remains unchanged from the model in which all samples were retained.

conventional equation $2(r_{mz} - r_{dz})$, where r_{mz}, r_{dz} are the intra-pair correlations of educational attainment for MZ and DZ twins respectively (Plomin, DeFries, McClearn and McGuffin 2001: 382). The simple estimator typically closely approximates estimates derived through more complex means and permits us to maximize cases from our meta-analysis by being calculable simply from twin correlations rather than requiring microdata.

As noted, twin studies partition variance in an outcome into that which is resolved by additive genetic variance (i.e., heritability), by the environment shared by members of a twin pair, and by the unshared environment. The first two of these are assumed to induce similarity between MZ twins while only the last induces divergence, and so the unshared environment component (e^2) is calculated as:

$$e^2 = 1 - r_{mz} .$$

The within-pair correlations on a given outcome—in our case, educational attainment—can therefore be expressed in terms of genetic (h^2) and shared environmental (c^2) components.

The correlation for MZ twins reflects full similarity of both genotype and shared environment,

$$r_{MZ} = h^2 + c^2 ,$$

whereas the correlation for same-sex DZ twins reflects half genetic similarity, but still full similarity of shared environment,

$$r_{DZ} = ½\, h^2 + c^2 .$$

Solving for h^2 and c^2, we find that our heritability estimate is equivalent to twice the difference between the MZ and DZ correlations,

$$h^2 = 2(r_{mz} - r_{dz}) ,$$

while our estimate of shared environment is equivalent to the remainder of the MZ correlation not accounted for by heritability,

$$c^2 = 2\, r_{dz} - r_{mz} = r_{mz} - h^2 .$$

For a given sample, the heritability, shared environment, and unshared environment components sum to one. Correlations, sample sizes, and the resulting estimates of h^2, c^2, and e^2 for our full set of studies are presented in table 3.

<TABLE 3 ABOUT HERE>

We do not attempt here to employ more complicated strategies for estimating heritability, including those that distinguish additive and non-additive sources of heritability or those that attempt to differentiate the main effect of genetic relatedness from the interaction between genetics and features of the environment that vary within populations. Although it is preferable to differentiate these effects in studies using micro-level data, in a meta-analysis one is largely constrained to the information available in published sources. As noted, by limiting our data requirements to only quantities derivative from r_{mz} and r_{dz} we are able to include a sufficient number of samples so as to make the comparative aims of this analysis feasible.

Meta-analysis

Meta-analysis is a statistical technique in which results from two or more studies of the same concept are mathematically combined to produce a single overall estimate, generally referred to as the "effect size" (Cooper, Hedges, and Valentine 1994). Although in its simplest form, a meta-analysis may simply be a weighted average, more advanced models are commonly used to take into account characteristics of studies that affect reliability of the effect sizes generated. Even in a set of studies testing the same hypothesis in which one believes that there is a single "true" underlying effect size, one may still expect some variation in the results due to study-specific differences.

Alternatively, one may suppose the possibility that there is no "true" effect size, but rather, that the actual effect itself may differ across studies, even in the absence of study-specific error. In

our case, we expect that environmental variation between our samples leads to differential exposure to factors such as national educational policies and cultural values, which in turn may alter the extent to which genes and environment explain variation in attainment. We thus consider a two-level hierarchical linear model with random effects to predict the true values of the h^2, c^2, and e^2 variance components in each study, as environmental differences may lead to sample-specific random variation (Cooper, Hedges, and Valentine 1994).

At the micro level, we assume that for study i, each observed variance component T_i is an estimate of a true study-specific variance component θ_i plus estimation error ε_i.[10] As the sample sizes in all of the studies in our data are reasonably large, we assume that T_i is normally distributed around θ_i with variance ν_i:

$$T_i = \theta_i + \varepsilon_i, \quad \varepsilon_i \sim N(0,\nu_i) \; . \tag{1}$$

At the macro level, we expect that study-specific differences may lead to between-study variation in θ_i, and so we model the true value of the variance components as

$$\theta_i = \beta_0 + \beta_1 N_{i1} + \beta_2 S_{i2} + \beta_3 C_{i3} + u_i, \quad u_i \sim N(0,\sigma_\theta^2) \tag{2}$$

where β_0 is the intercept; N_i is the nationality of respondents in study i; S_i is the sex of respondents in study i; C_i is the birth cohort of respondents in study i; and u_i is the random effect of study i, reflecting the deviation of the true value of the variance component from the value predicted by the study characteristics. Random effect u_i is normally distributed with mean zero and variance σ_θ^2, the between-studies variance. Combining the two models,

[10] For more on random effects models in meta-analysis, see Chapter 16 in Cooper, Hedges, and Valentine (1994), from which our models were adapted.

$$T_i = \beta_0 + \beta_1 N_{i1} + \beta_2 S_{i2} + \beta_3 C_{i3} + u_i + \varepsilon_i, \qquad u_i + \varepsilon_i \sim N(0, \nu_i^*) \ , \qquad (3)$$

where the total variance observed in T_i is $\nu_i^* = \sigma_\theta^2 + \nu_i$.

This model implies two sources of variation in true effect sizes: first, studies may differ by observed characteristics (nationality, sex, and birth cohort); second, studies may differ by a study-specific random effect *u*. If neither of these sources contributes significant variation, the true effect size for all studies will be equal to the intercept β_0 plus any sampling error ε. In this case the between-studies variance σ_θ^2 is zero, and all variation is attributed to sampling error ε. To generate the true effect size β_0 for all studies *k* under this assumption of homogeneity, we weight the results from each study *i* by the reciprocal of the sampling variances,

$$w_i = 1/\nu_i \ ,$$

so that studies with more precise estimates of the variance components will be given more weight than relatively imprecise estimates.

Since each T_i is only an unbiased estimate of β_0 when effect sizes are homogeneous, for each model presented in tables 4 and 5 we first evaluate homogeneity in true effect sizes by calculating the *Q* test statistic to detect presence or absence of heterogeneity between samples. As per Cochran (1954), *Q* is computed as the sum of squares of deviations from the overall estimate of effect size, with each study weighted by its inverse variance. The null hypothesis of homogeneity among effect sizes (H_0: $\sigma_\theta^2 = 0$) follows a chi-square distribution with *k*-1 degrees of freedom, in which *k* denotes the total number of studies. While the *Q*-test is standard in the literature, it informs only about the presence or absence of heterogeneity, rather than about the magnitude of true heterogeneity between

studies (Huedo-Medina, Sánchez-Meca et al. 2006). We thus also report the estimate of the magnitude of the between-study variance, σ_θ^2, and the I^2 indicator of the proportion of the total variability in effect sizes that results from heterogeneity between studies rather than from random sampling error (Higgins and Thompson 2002; Higgins, Thompson et al. 2003). The I^2 indicator has the additional benefit of being easy to interpret: a value of 0% (I^2=0) would indicate that all variability among estimates is due to sampling error, whereas a value of 100% (I^2=100) would indicate that all variability is due to heterogeneity between studies (Higgins and Thompson 2002). The confidence interval around the I^2 indicator is calculated using the formula suggested by Higgins and Thompson (2002). In the instances where we are able to reject the null of homogeneity, we proceed to evaluate possible sources of heterogeneity (model 3), recalculating our weights as

$$w_i = 1/v_i^*$$

to reflect the contribution of between-study variability to overall variability in the estimates.

To calculate v_i, we assume that since the estimates of r_{MZ} and r_{DZ} are calculated from two separate groups of respondents, they are statistically independent. The variances of the estimators h^2 and c^2 are therefore

$$Var(h^2) = Var(2(r_{MZ} - r_{DZ})) = 4(Var(r_{MZ}) + Var(r_{DZ}))$$

and

$$Var(c^2) = Var(2r_{DZ} - r_{MZ}) = 4Var(r_{DZ}) + Var(r_{MZ}) \quad .$$

The variance of the estimated correlation coefficients can be accurately approximated by

$$Var(r) \approx \frac{(1 - r^2)^2}{n - 1}$$

where n is the sample size. The variances of the h^2 and c^2 estimators are then

$$Var(h^2) \approx 4\left(\frac{(1-r_{MZ}^2)^2}{n_{MZ}-1}+\frac{(1-r_{DZ}^2)^2}{n_{DZ}-1}\right)$$

and

$$Var(c^2) \approx 4\left(\frac{(1-r_{DZ}^2)^2}{n_{DZ}-1}\right)+\frac{(1-r_{MZ}^2)^2}{n_{MZ}-1} \ .$$

Since $e^2 = 1 - r_{MZ}$, the variance of e^2 is equal to the variance of r_{MZ}. The variance of e^2 will therefore be smaller than the variances of h^2 and c^2, as can be seen in the forest plots presented in figures 1 through 3.

<FIGURE 1, FIGURE 2, FIGURE 3 ABOUT HERE>

Our weighting system has the drawback that the estimates of h^2, c^2, and e^2 are functions of r_{MZ} and r_{DZ}, with the variances being monotonically decreasing functions of r_{MZ} and r_{DZ}. This could potentially bias estimates by overweighting some effect sizes while underweighting others. For a meta-analysis involving simple estimates of correlation, this problem is usually addressed by using a variance stabilizing transformation; however, the transformations used for correlations are not suitable for linear functions of correlations. The potential for bias was investigated using simulations based on the empirical distribution of r_{MZ} and r_{DZ} values, and based on the results of these simulations, we concluded that any bias that exists in the estimates is small enough to be considered inconsequential.

RESULTS

For each of the twin correlations and variance component estimates presented in tables 4 and 5, our analysis proceeds in two steps. In step one, we generate an estimate of the grand mean (β_0), and calculate Q to test for sample homogeneity. If we are unable to reject the null of homogeneity, suggesting that the true values do not vary across populations, we interpret the grand mean as our

best estimate of the true value. If we are able to reject the null of homogeneity we proceed to step two, in which we seek to explain the observed heterogeneity by introducing covariates (nation, sex, and birth cohort) and study-specific random effects. Because $e^2 = 1 - r_{MZ}$, patterns of significance are identical across e^2 and r_{MZ} in all models.

In table 4, we present the results of our meta-analysis of the MZ and DZ correlations and the estimates of h^2, c^2, and e^2 across our full set of samples. In all models, heritability, shared environment, and unshared environment each explain a percentage of the variance in attainment that is significantly greater than zero. As hypothesized, the contribution of each component varies by sample: we are able to reject the null of homogeneity in the grand mean across both sets of correlations and all three variance components, with a large percentage of the variation attributable to between-study variance (I^2 values range from 77% for h^2 to 97% for r_{MZ} and e^2). Our estimates of the magnitude of between-study variance (σ_θ^2) are also relatively large—for h^2 and c^2, the between-studies standard deviation is 0.11, equivalent to about one-fourth of the grand mean.

Upon introduction of random effects and covariates, we see that estimates of e^2 (and r_{MZ}) vary significantly by nation, sex, and birth cohort, with unshared environment explaining four percentage points more of the variation in attainment among men than women, and four percentage points less of the variation among twins born post-1950 than among those born earlier. These significant group differences imply equivalent shifts in the sum of the remaining two variance components, which together constitute the total family effect. Microdata studies evaluating the influence of family on educational attainment across a range of Western nations have found family effects to either decline or remain relatively stable over the past century;[11] our non-zero but

[11] A decline in the effect of family factors on educational attainment was found in populations drawn from the United States (Kuo and Hauser 1997), the Netherlands (Dronkers 1993), and West Germany (De Graaf and Huinink 1992); no cohort change in the effect of family factors on

substantively meager decline in total family effect between cohorts in our international sample is consistent with these findings. Estimates of r_{DZ} also vary significantly by nation, sex, and birth cohort, with DZ correlations seven percentage points higher for women than for men, and eight percentage points higher for those born in the later cohort than for those born earlier.

<TABLE 4 ABOUT HERE>

Despite the small shift in the total family effect by sex and cohort, we see a far larger difference by sex and cohort in the composition of the family effect, with variation in genes explaining more of the variation in educational attainment for men and those born later in the century, and variation in shared environment explaining more of the variation for women and for respondents born earlier. Shared environment explains ten percentage points more of the variance in attainment for women than for men, and twelve percentage points less of the variance in attainment for those born after 1950 than for those born earlier. Conversely, genetic variation explains six percentage points less of the variance in attainment for women than men, and eight percentage points more of the variance in attainment for those born after 1950 than for those born earlier.[12] These differences are in the direction one would expect if women and respondents in the earlier cohort were exposed to greater social constraints with respect to educational attainment than men and respondents born later in the century.

As hypothesized, nation also appears to affect the extent to which genetic versus environmental differences account for variation in educational attainment. With the United States as our reference category, we observe significant differences ($p<0.05$) in at least one of the variance

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attainment was observed in samples drawn from Australia (Borgers et al. 1995), Hungary (Toka and Dronkers 1996), or in an additional sample from the United States (Sieben and DeGraaf 2003).

[12] To address the possibility that sex may be serving as a proxy for unmeasured characteristics—for example, veteran status, as our veteran samples consist of men only—we ran all models presented in table 3 on a subgroup consisting of only the 14 samples for which we have data on both males and females separately. We find no notable differences in the significance or direction of the coefficient on sex across any of the five models.

components for five out of nine countries: Finland, Italy, Norway, Spain, and the United Kingdom. The direction of the shifts between variance components fluctuates by country. Our finding of high levels of heterogeneity across all models in our subgroup of Scandinavian samples (table 5) suggests that nation is indeed the salient factor driving the heterogeneity observed in table 4, rather than nation serving as a proxy for broader regional characteristics.[13] While neither birth cohort nor sex are significant among the Scandinavian samples, the coefficients on sex and cohort for all three variance components are again in the direction one would expect if women and respondents in the earlier birth cohort were exposed to greater social constraints, similar to the full-sample results in table 4.[14]

<TABLE 5 ABOUT HERE>

Although we observed heterogeneity in the grand means for all three variance components in our full-sample analysis (table 4), the relative magnitudes of our grand mean estimates of heritability and common environment constitute an unanticipated yet notable finding. Counter to the well-established expectation that the estimate of common environment will be far below the estimate of heritability and often close to zero (Turkheimer 2004), here we observe grand means of heritability and common environment that are both large and substantively quite similar (h^2= 0.400, while c^2=0.361). As our inability to reject the null of homogeneity suggests that these estimates do vary by sample, the descriptive statistics presented in table 3 lend additional insight here: in 11 of our 34 samples, our estimate of common environment actually exceeds our estimate of heritability.

[13] Although Heath et al. (1985) found significant heterogeneity between the Norwegian samples, Norwegians represent but a small percentage of the full Scandinavian sample (20% of Scandinavian MZ twins and 14% of Scandinavian DZ twins).

[14] As one robustness check regarding the impact of differences in measurement scales on heterogeneity in our variance components, we also ran our models on a subgroup consisting of only our 10 U.S. samples, in which coding scales ranged from six attainment categories (Minnesota) to 13 attainment categories (AddHealth). After controlling for sex and birth cohort, we observed no residual heterogeneity in either MZ or DZ correlations or any of our three variance components, suggesting that variation in the number of coding categories alone was not sufficient to generate the extent of heterogeneity we observed by nation in our full sample.

Far from having a near-zero effect, in only five of our samples does the estimate of common environment fall below 20%, and in two it approaches 70%.

DISCUSSION

As Hauser and Wong (1989) note, early efforts to separately estimate the genetic and environmental components of variation in social outcomes were often framed as attempts to "resolve the old debate about nature versus nurture as sources of social inequality" (151). Our results reflect a far more complicated relationship between nature and nurture than a simple dichotomy, suggesting that variables such as nation, sex, and birth cohort influence the extent to which genetic and environmental factors explain variation in educational attainment. While heritability, shared environment, and unshared environment each explain a significant percentage of the variation in attainment across our samples, we find that the relative contributions of the three components vary between studies. National differences explain a portion of this observed heterogeneity, while we do not observe a similar effect for regional differences more broadly. This finding is important to consider when attempting to generalize results from single-nation microdata studies of heritability to other nations, given the numerous unique environmental characteristics implicit in any national policy environment.

With respect to variation in the influence of genetic differences on educational attainment by sex and birth cohort, we find our coefficients to be consistent with twentieth-century attainment trends. For men and individuals born in the latter half of the twentieth century, more of the variance in attainment can be explained by genetic variation, whereas shared environment explains a greater percentage of the variance in attainment for women and those born in the earlier half of the century. In a mobility analysis where genetic and family social factors are assessed cumulatively, these shifts would be masked by the relatively small sex and cohort differences in the total family effect. We

thus suggest calculating separate estimates of heritability and common environment as a method of distinguishing the impact of family characteristics that are transmitted strictly socially from those family characteristics that appear to follow from the greater genetic similarity within biological families (e.g. Bjorklund, Jännti and Solon 2003; Jencks and Tach 2006; Nielsen 2008; Diewald 2010). Shifts in the balance between these two types of family effects may reflect shifts in patterns of mobility even when the sum of the two remains stable.

Although we observed significant heterogeneity among all three variance components in our full-sample analysis (table 4), the sheer magnitude of the common environment estimates we observe, consistently across many samples, may surprise those who are used to seeing very low common environment estimates from twin study models. While such clear evidence of non-genetic, within-family influence on educational attainment may be less surprising to sociologists, the unusual robustness of this result relative to other outcomes studied in behavioral genetics cannot be understated. Table 1 suggests that children from the same household are likely to differ more in their sexual orientation, athletic participation, personality traits such as perfectionism and extroversion, alcohol consumption, smoking habits, and even on their IQ, than they do in their educational attainment. Indeed, for educational attainment, not only is our grand mean estimate of common environment of similar magnitude to our grand mean estimate of heritability, but in 32% of the studies in our sample, the estimated effect of common environment actually exceeds that of genetics (table 3).

We suggest that this divergence from prior literature in the effect of shared environment relative to genes may be in part due to differences between the outcome of educational attainment and the types of outcomes for which heritability estimates have been most commonly calculated. First, unlike the attitudes, beliefs, personality traits, and habitual behaviors listed in table 1, educational attainment has no intrinsic volatility. Attainment is determined with a clear cutoff, as

either one has completed a year of education or one has not, and once attained, education can never be subsequently unattained. This greatly reduces concern about test-retest variation, as any characteristic that fluctuates within an individual can be also expected to fluctuate between twins, inflating the estimate of unshared environment. Second, even if ultimate attainment is often reached after a child has left the home, most educational attainment takes place during the years in which twins are likely to be living in the same household in the same neighborhood, going to the same schools, and so on. The divergent life trajectories that twins may experience after leaving the home can be expected to be less relevant for attainment than for later outcomes, even more traditionally sociological outcomes such as income (Taubman 1976: 867). Future behavior genetic models of social outcomes may want to consider how these aspects of the outcome itself— intrinsic volatility and extent of common environment during the time the outcome occurred— may be expected to influence results, particularly when comparing estimates between outcomes that may differ in these respects.

Along with the benefits of meta-analysis, we recognize also its shortcomings. While we have attempted to address these issues, coding variation between studies, imperfect information regarding sex mix and cohort composition, and an inability to engage more advanced modeling techniques when calculating heritability estimates are among the tradeoffs of obtaining a sample as large and diverse as the one we present here. Further analysis by subgroup would be a valuable extension of this research, and should be possible in the foreseeable future given the number of international twin studies in which respondents are simply too young at present for ultimate educational attainment to be a reasonable outcome.

In addition, advances in the availability of molecular genetic data may very soon allow significantly more fine-grained analyses than what we present here. To date, social science studies of gene-environment interaction using molecular genetic data have largely focused on small

numbers of specific genetic variants, whose contribution to the overall population heritability of an outcome is unknown but almost certainly very small (Duncan and Keller 2011). If social outcomes are influenced by very large numbers of genes, as now seems clear (Beauchamp et al. 2011), studies of any individual gene considered in isolation may have limited value for understanding macroscopic social processes such as educational attainment. Far more promising are approaches based on genome-wide data, which can be used to establish associations between a given outcome and a range of genetic variants. Genome-wide approaches can also be used to estimate the heritability of outcomes in samples of individuals who are not known to be related, thereby addressing any concerns about particular assumptions of twin models or potential atypicalities of twins (Benjamin et al. 2012). A consortium seeking to do this for educational attainment across multiple data sources is already underway (see http://www.ssgac.org/); a recent GWAS analysis of an Australian sample (Martin et al. 2011) found only one polymorphism to be associated with educational attainment at a statistically significant level, emphasizing the likelihood that attainment is highly polygenic.

Table 1: Selected studies of the Heritability of Social Outcomes, by Percent Heritability and Percent Common Environment

	HIGH COMMON ENVIRONMENT (>33%)	**LOW COMMON ENVIRONMENT** (≤33%)
HIGH HERITABILITY (>33%)	- Smoking initiation in men (Li 2003)	- Alcoholism (Heath et al. 1997) - Altruism and Aggression (Rushton et al. 1986) - Age at first intercourse for respondents born ≥1952 (Dunne et al. 1997) - Attitudes on school prayer, property tax, moral majority, capitalism, astrology, draft laws, pacifism, unions, republicans, socialism, foreign aid, X-rated movies, immigration, women's liberation (Alford, Funk, and Hibbing 2005) - Attitudes on the death penalty, open-door immigration, doing athletic activities, voluntary euthanasia, exercising, organized religion, reading books, roller coasters (Olson et al. 2001) - College plans (Nielsen 2006) - Depression in women (Kendler et al. 2006) - *Electoral participation* (Fowler, Baker, and Dawes 2008) [1] - Extraversion (Pedersen et al. 1988) - Impulsivity (Pedersen et al. 1988) - Income (Taubman 1978) - Inhibition (Robinson et al. 1992) - IQ (e.g. Rowe et al. 1999) [2] - Loneliness (Boomsma et al. 2005) - Low birth weight (Clausson, Lichtenstein, and Cnattingius 2000) - Perceived Self-Confidence (McGuire et al. 1994) - Perfectionism (Tozzi et al. 2004) - Sexual orientation in women (Bailey et al. 1993) - Smoking initiation in women (Li 2003) - Smoking persistence (Li 2003) - Social cognitive skills (Scourfield et al. 1999) - Sensation seeking (Stoel, De Geus, and Boomsma 2006)
LOW HERITABILITY (≤33%)	- Age at first intercourse for men born <1952 (Dunne et al. 1997) - Attitudes on abortion and on living together (Alford, Funk, and Hibbing 2005) - Preference for loud music (Olson et al. 2001)	- Attitudes on the death penalty, censorship, military drill, gay rights, segregation, busing, nuclear power, democrats, divorce, modern art, federal housing, liberals (Alford, Funk, and Hibbing 2005) - Preference for sweets, organized sports, looking one's best at all times, legalizing racial discrimination, public speaking, wearing clothes that draw attention, being the leader of groups (Olson et al. 2001) - Depression in men (Kendler et al. 2006) - Neuroticism (Pedersen et al. 1988) - Stress coping styles (Kato and Pedersen 2005)

[1] In a sample from Los Angeles, estimates of common environment and heritability were both >33%; in a nationally representative sample, the common environment estimate was substantially lower than 33%.

[2] The heritability estimates for both IQ and achievement have been found to vary significantly by population subgroup (e.g. Guo and Stearns 2002; Rowe et al. 1999). Full population estimates have generally found heritability to be high and common environment to be low, with some exceptions (e.g. Thompson, Detterman, and Plomin 1993).

Table 2: List of Studies

Nationality	Sex	Birth Years	Source	Sample Title	Population-Based
Australia (1)	Male & Female	1893-1950	Baker et al. (1996)	Australian Twin Register	No
Australia (2)	Male & Female	1951-1965	Baker et al. (1996)	Australian Twin Register	No
Australia (3)	Male & Female	1964-1971	Miller, Mulvey, and Martin (1997)	Australian Twin Register	No
Denmark	Male only	1954-2004	Bingley, Christensen et al. (2005)	Danish Twins Registry	No
Finland	Male & Female	1936-1955	Silventoinen, Krueger et al. (2004)	Finnish Twin Cohort Study	Yes
Germany	Male & Female	1912-1985	Obtained from Investigator	Bielefeld Twin Study	No
Italy	Male & Female	1913-1977s	Obtained from Investigator	Italian Twin Registry	Yes
Norway (1)	Male & Female	1915-1939	Heath, Berg et al. (1985)	Norwegian Twin Panel	No
Norway (2)	Male & Female	1940-1949	Heath, Berg et al. (1985)	Norwegian Twin Panel	No
Norway (3)	Male & Female	1950-1960	Heath, Berg et al. (1985)	Norwegian Twin Panel	No
Spain	Male & Female	1940-1965	Obtained from Investigator	Murcia Twin Registry	No
Sweden	Mixed	1926-1958	Isacsson (1999)	Swedish Twin Registry	No
United States					
AddHealth	Male & Female	1976-1984	Calculated from Sample	National Longitudinal Study Of Adolescent Health	Yes
MIDUS	Male & Female	1921-1969	Calculated from Sample	Midlife in the United States Study	Yes
Vietnam Veterans	Male only	1939-1957	Taubman (1976)	Vietnam Era Twin (VET) Registry (NAS-NRC)	No
Minnesota	Male & Female	1936-1955	Lykken, Bouchard et al. (1990)	Minnesota Twins Registry	Yes
WW2 Veterans	Male only	1917-1927	Behrman, Taubman et al. (1977)	World War II Veterans Twin Registry (NAS-NRC)	No
SRI	Male & Female	1911-1985	Obtained from Investigator	Twin Research Registry at SRI International	No
United Kingdom (1)	Mixed [3]	1917-1950	Calculated from Sample	TwinsUK	No
United Kingdom (2)	Mixed	1951-1985	Calculated from Sample	TwinsUK	No

[3] We include the UK samples as mixed-sex estimates due to an insufficient number of males to calculate sex-by-cohort correlations. However, the precise ratio of males to females is known.

Table 3: Twin Correlations and Variance Component Estimates

Nationality ***(k=34)***	**Sex**	**Cohort**	***r*(MZ)**	**N_{mz}**	***r*(DZ)**	**N_{dz}**	h^2	c^2	e^2
							$2(r_{MZ} - r_{DZ})$	$r_{MZ} - h^2$	$1 - r_{MZ}$
Australia (1)	Male	1	**0.70**	216	**0.53**	94	0.34	0.36	0.30
	Female	1	**0.77**	520	**0.55**	299	0.44	0.33	0.23
Australia (2)	Male	2	**0.74**	226	**0.47**	161	0.54	0.20	0.26
	Female	2	**0.75**	479	**0.49**	290	0.52	0.23	0.25
Australia (3)	Male	2	**0.674**	282	**0.532**	164	0.284	0.39	0.326
	Female	2	**0.705**	320	**0.319**	158	0.772	-0.067	0.295
Denmark	Male	2	**0.62**	4370	**0.444**	7068	0.352	0.268	0.38
Finland	Male	1	**0.83**	1506	**0.58**	3504	0.50	0.33	0.17
	Female	1	**0.86**	2028	**0.62**	3870	0.48	0.38	0.14
Germany	Male	Mixed	**0.680**	133	**0.306**	47	0.748	-0.068	0.320
	Female	Mixed	**0.717**	421	**0.479**	172	0.476	0.241	0.283
Italy	Male	Mixed	**0.71**	752	**0.61**	406	0.20	0.51	0.29
	Female	Mixed	**0.79**	1342	**0.7**	712	0.18	0.61	0.21
Norway (1)	Male	1	**0.86**	259	**0.77**	313	0.18	0.68	0.14
	Female	1	**0.89**	405	**0.75**	425	0.28	0.61	0.11
Norway (2)	Male	1	**0.82**	253	**0.48**	284	0.68	0.14	0.18
	Female	1	**0.85**	342	**0.68**	400	0.34	0.51	0.15
Norway (3)	Male	2	**0.85**	370	**0.47**	463	0.76	0.09	0.15
	Female	2	**0.89**	518	**0.66**	576	0.46	0.43	0.11
Spain	Male	Mixed	**0.758**	128	**0.519**	155	0.478	0.28	0.242
	Female	Mixed	**0.821**	228	**0.562**	231	0.518	0.303	0.179
Sweden	Mixed	1	**0.76**	2492	**0.55**	3368	0.42	0.34	0.24
United States									
AddHealth	Male	2	**0.611**	100	**0.477**	94	0.268	0.343	0.389
	Female	2	**0.623**	117	**0.650**	93	-0.054	0.677	0.377
Vietnam Veterans	Male	1	**0.76**	1019	**0.54**	907	0.44	0.32	0.24
Minnesota	Male	Mixed	**0.65**	512	**0.42**	772	0.46	0.19	0.35
	Female	Mixed	**0.72**	758	**0.57**	1154	0.30	0.42	0.28
WW2 Veterans (NAS-NRC)	Male	1	**0.764**	1234	**0.545**	1167	0.438	0.326	0.236
MIDUS	Male	Mixed	**0.668**	164	**0.538**	124	0.293	0.375	0.332
	Female	Mixed	**0.707**	186	**0.561**	198	0.260	0.447	0.293
SRI	Male	Mixed	**0.65**	170	**0.48**	28	0.34	0.31	0.35
	Female	Mixed	**0.68**	390	**0.50**	123	0.36	0.32	0.32
United Kingdom (1)	Mixed	1	**0.717**	457	**0.521**	393	0.391	0.326	0.283
United Kingdom (2)	Mixed	2	**0.593**	388	**0.474**	247	0.238	0.355	0.407
			Total	*23085*		*28460*			

Table 4: Correlations and Heritability Variance Components, Full Sample *(Reference category: American males born pre-1950)*

	***r*(MZ)**		***r*(DZ)**		***h²***		***c²***		***e²***	
	1	***2***	***1***	***2***	***1***	***2***	***1***	***2***	***1***	***2***
Grand Mean	0.747***		0.551***		0.400***		0.361***		0.253***	
	(0.015)		(0.018)		(0.024)		(0.026)		(0.015)	
Nationality										
Australia		0.022		-0.023		0.097		-0.074		-0.022
		(0.018)		(0.033)		(0.076)		(0.069)		(0.018)
Denmark		-0.055+		-0.009		-0.095		0.038		0.055+
		(0.030)		(0.046)		(0.114)		(0.098)		(0.030)
Finland		0.116***		0.036		0.155*		-0.042		-0.116***
		(0.016)		(0.029)		(0.067)		(0.061)		(0.016)
Germany		-0.012		-0.096		0.177		-0.187		0.012
		(0.023)		(0.066)		(0.146)		(0.136)		(0.023)
Italy		0.044*		0.134***		-0.181*		0.222***		-0.044*
		(0.020)		(0.031)		(0.075)		(0.066)		(0.020)
Norway		0.147***		0.119***		0.055		0.092+		-0.147***
		(0.013)		(0.023)		(0.054)		(0.049)		(0.013)
Spain		0.084***		0.015		0.137		-0.056		-0.084***
		(0.025)		(0.047)		(0.106)		(0.097)		(0.025)
Sweden		0.032		-0.014		0.085		-0.057		-0.032
		(0.024)		(0.039)		(0.091)		(0.081)		(0.024)
United Kingdom		-0.056*		-0.053		-0.014		-0.040		0.056*
		(0.020)		(0.043)		(0.103)		(0.091)		(0.020)
Female		0.035***		0.065***		-0.061		0.096*		-0.035***
		(0.007)		(0.018)		(0.041)		(0.038)		(0.007)
Post-1950 Birth Cohort		-0.036*		-0.082*		0.079		-0.118*		0.036*
		(0.014)		(0.027)		(0.061)		(0.056)		(0.014)
Intercept		0.711***		0.532***		0.366***		0.349***		0.289***
		(0.012)		(0.014)		(0.047)		(0.042)		(0.012)
Q	1010.08***	67.626***	514.497***	74.873***	143.114***	66.164***	152.918***	70.771***	1010.08***	67.626
σ_θ^2	0.007	0.000	0.008	0.001	0.012	0.004	0.013	0.004	0.007	0.000
I²	96.73	67.47	93.59	70.62	76.94	66.75	78.42	68.91	96.73	67.47
95% CI	96.08-97.28	49.79-78.92	91.97-94.87	55.16-80.75	68.1-83.34	48.56-78.51	70.32-84.32	52.27-79.76	96.08-97.28	49.79-78.92

*** p<0.001, ** p<0.01, * p<0.05, + p<0.1 Standard errors in parentheses

Table 5: Correlations and Heritability Variance Components, Comparison by Region

	***r*(MZ)**		***r*(DZ)**		***h²***		***c²***		***e²***	
	1	***2***	***1***	***2***	***1***	***2***	***1***	***2***	***1***	***2***
Scandinavia *(k=10)*										
Grand Mean	0.823*** (0.027)		0.601*** (0.033)		0.432*** (0.036)		0.385*** (0.042)		0.177*** (0.027)	
Female		0.072 (0.053)		0.110 (0.066)		-0.058 (0.122)		0.133 (0.117)		-0.072 (0.053)
Post-1950 Birth Cohort		-0.041 (0.054)		-0.195 (0.068)		0.083 (0.118)		-0.136 (0.113)		0.041 (0.054)
Intercept		0.803*** (0.039)		0.581*** (0.050)		0.434** (0.090)		0.367** (0.086)		0.197** (0.039)
Q	679.442***	387.970***	402.630***	165.609***	74.492***	75.335***	94.409***	88.626***	679.442***	387.970***
σ^2_θ	0.007	0.000	0.008	0.001	0.012	0.004	0.013	0.004	0.007	0.000
I²	98.675	98.196	97.765	95.773	87.918	90.708	90.467	92.102	98.675	98.196
95% CI	*98.284-98.978*	*97.504-98.696*	*96.961-98.356*	*93.535-97.236*	*79,830-92.763*	*84.100-94.570*	*84.610-94.095*	*86.814-95.269*	*98.284-98.481*	*97.504-98.696*

*** p<0.001, ** p<0.01, * p<0.05, + p<0.1 Standard errors in parentheses

Table 5: Correlations and Heritability Variance Components, Comparison by Region

	***r*(MZ)**		***r*(DZ)**		***h²***		***c²***		***e²***	
	1	***2***	***1***	***2***	***1***	***2***	***1***	***2***	***1***	***2***
Scandinavia *(k=10)*										
Grand Mean	0.823*** (0.027)		0.601*** (0.033)		0.432*** (0.036)		0.385*** (0.042)		0.177*** (0.027)	
Female		0.072 (0.053)		0.110 (0.066)		-0.058 (0.122)		0.133 (0.117)		-0.072 (0.053)
Post-1950 Birth Cohort		-0.041 (0.054)		-0.195 (0.068)		0.083 (0.118)		-0.136 (0.113)		0.041 (0.054)
Intercept		0.803*** (0.039)		0.581*** (0.050)		0.434** (0.090)		0.367** (0.086)		0.197** (0.039)
Q	679.442***	387.970***	402.630***	165.609***	74.492***	75.335***	94.409***	88.626***	679.442***	387.970***
σ^2_θ	0.007	0.000	0.008	0.001	0.012	0.004	0.013	0.004	0.007	0.000
I²	98.675	98.196	97.765	95.773	87.918	90.708	90.467	92.102	98.675	98.196
95% CI	*98.284-98.978*	*97.504-98.696*	*96.961-98.356*	*93.535-97.236*	*79,830-92.763*	*84.100-94.570*	*84.610-94.095*	*86.814-95.269*	*98.284-98.481*	*97.504-98.696*

*** p<0.001, ** p<0.01, * p<0.05, + p<0.1 Standard errors in parentheses

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