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Genes, Gender Inequality, and Educational Attainment

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Abstract

Women's opportunities have been profoundly altered over the past century by reductions in the social and structural constraints that limit women's educational attainment. Do social constraints manifest as a suppressing influence on genetic indicators of potential, and if so, did equalizing opportunity mean equalizing the role of genetics? We address this with three cohort studies: the Wisconsin Longitudinal Study (WLS; birth years 1939 to 1940), the Health and Retirement Study, and the National Longitudinal Study of Adolescent Health (Add Health; birth years 1975 to 1982). These studies include a "polygenic score" for educational attainment, providing a novel opportunity to explore this question. We find that within the WLS cohort, the relationship between genetics and educational outcomes is weaker for women than for men. However, as opportunities changed in the 1970s and 1980s, and many middle-aged women went back to school, the relationship between genetic factors and education strengthened for women as they aged. Furthermore, utilizing the HRS and Add Health, we find that as constraints limiting women's educational attainment declined, gender differences in the relationship between genetics and educational outcomes weakened. We demonstrate that genetic influence must be understood through the lens of historical change, the life course, and social structures like gender.

Keywords

genetics, gender, education, biodemography

Women's opportunities have been profoundly altered over the past century by reductions in the social and structural constraints that limited their access to higher education. Did the constraints that limited women's educational attainment manifest as a suppressing influence on genetic indicators of potential? If so, did equalizing opportunity mean equalizing the role of genetics? Examining gene-environment interplay in this way can help reveal more precisely how gender inequalities in educational outcomes have changed over time, and how the influence of individual traits on attainments is shaped by socially contingent environments.

Past empirical approaches, such as conventional twin studies, have been limited in their ability to examine gene-environment interplay, but the development of a polygenic score for educational attainment provides

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social scientists with a more direct tool to interrogate how social conditions shape genetic influence. In this article, we utilize three cohort studies: the Wisconsin Longitudinal Study (WLS; birth years 1939 to 1940), the Health and Retirement Study (HRS; birth years 1931 to 1956), and the National Longitudinal Study of Adolescent Health (Add Health; birth years 1975 to 1982). We look at whether gender moderates the relationship between a polygenic score for educational attainment and educational outcomes, not only across the three cohorts, but also within the WLS cohort, as women experienced varying structural and social constraints that shaped their schooling patterns over their life course. Prior work, particularly using polygenic scores, has paid little attention to either gender or the life course. We demonstrate that genetic influence must be understood in the context of history and cohort, the life course, and social structures like gender. The influence of genetics on educational attainment cannot be understood outside of this context.

PERSPECTIVES ON GENES, ENVIRONMENT, AND EDUCATIONAL ATTAINMENT

We outline contrasting perspectives on the relationship between genes, environment, and educational attainment. We do so to provide a heuristic for understanding the assumptions that underlie the existing empirical work devoted to this broader topic, as well as more broadly the dynamics of gene-environment interplay. Our point is not to put particular scholars or particular disciplines in one box or another, as empirical evidence continues to accrue and perspectives change as that evidence accrues.[1]

Genes, Environment, and Educational Attainment: "Strong" Genetic Influences

Most research involving genetics and educational attainment is rooted in a perspective that assumes, tacitly or explicitly, that any genetic influence is separable from that of environments: nature and nurture can be disentangled (Plomin 2018). Within economics and sociology, a robust literature seeks to separate genetic and environmental influences as a way of clarifying the role of the latter in educational differences. The popularity of sibling models (e.g., Conley and Bennett 2000; Haas and Fosse 2008; Hauser and Wong 1989; Warren and Hauser 1997; Warren, Sheridan, and Hauser 2002) and the growing use of "exogenous" shocks, such as changes in mandatory schooling laws (e.g., Black, Devereux, and Salvanes 2008; Glymour et al. 2008; Oreopoulos 2006), reflect the underlying assumption among many social scientists that genetic differences may influence educational outcomes. The goal with this modeling is to rule out genetics as an explanation. Although many of these scholars would have tacitly acknowledged the likelihood of gene-environment interactions, they lacked the tools to test them.

While this segment of the social sciences employed empirical approaches to rule out genetic explanations, in order to specify environmental influences on educational attainment, another segment, behavioral genetics, attempted to demonstrate what Turkheimer (2016:25) calls "strong" genetic explanations, specifically that "an observed phenotypic difference is a manifestation of a specific latent genetic mechanism." Turkheimer further argues that "the history of behavior genetics can be seen as an extended attempt to proceed from weak to strong genetic explanation." Empirically, this involved using twin models to estimate heritability: that is, the fraction of the variance in a trait, like educational attainment, due to genetic variation between individuals in the population. Indeed, until the past few years, twin studies have provided what evidence we have regarding the influence of genetic factors on educational attainment (for a review, see Branigan, McCallum, and Freese 2013). On average, these studies show that approximately 40 percent of the variance in educational attainment can be

accounted for by genetics (Branigan et al. 2013).

To be clear, "strong" genetic explanations for educational attainment do not rule out environmental influences (Krapohl and Plomin 2016; Plomin 2018; Scarr and McCartney 1983; Turkheimer 1998). In addition to the possibility of independent environmental influences acknowledged by this perspective, a key assumption about environment is that one's genes can influence the environment in which one lives, which in turn influences educational attainment (e.g., if genetics make it harder to read, as is the case with dyslexia, one reads less) (Jencks 1980). Indeed, this perspective assumes this may explain a large part of the "effect" of environment (Kong et al. 2018; Plomin 2018; Scarr and McCartney 1983).

Another embedded assumption in this perspective is an "atomistic" view of environment—what Boardman, Daw, and Freese (2013) call micro-environments—in which the consideration of environmental influence centers on family, in part reflecting the use of twin and sibling models to parse out heritability (Plug and Vijverberg 2003). As Plomin (2018:ix) notes, "for most of the twentieth century environmental factors were called nurture because the family was thought to be crucial in determining who we become." Though the literature specifically focused on education has attended to the possibility of school influences and family influences, the twin design has typically not allowed for the disentanglement of these influences (Bartels et al. 2002). More generally, this perspective, and the twin model empirical approach that grounds it, constrains the ability to examine broader environments ranging from institutions, such as educational systems (not simply schools), to broader social forces, such as gender (Boardman et al. 2013; Jencks 1980; Wedow et al. 2018).

Finally, a key assumption underlying this perspective is that complex social outcomes—like education—do not change over time. For there to be a "strong" genetic influence on educational attainment, education itself will not fundamentally change over time. The phenotype—in this case educational attainment—is historically fixed. Or perhaps more precisely, environments simply do not change enough—the variance is not large enough—to alter the influence of genetics, at least at this point in history. Plomin (2018:vii) succinctly expresses this view, arguing that genetics can be used to help us "understand who we are, and predict who we will become."

One of the challenges of assessing the role of broader environments has been the use of twin studies. We will detail those limitations here, but we do want to emphasize that these studies have real advantages (from a causal inference perspective) and have been used to demonstrate novel interactions between genes and some broader environments, like birth cohorts (Boardman, Alexander, and Stallings 2011; Boardman, Blalock, and Button 2008; Boardman, Blalock, and Pampel 2010), as well as the influence of family socioeconomic differences in moderating genetic influences on cognition (e.g., Turkheimer et al. 2003). Nonetheless, they have some limitations, including (1) the lack of directly measured genotypes and (2) how the small and select samples (which often have more limited measures of "environment" compared to typical longitudinal cohort studies used by social scientists) make it generally difficult to explore how broader environments may modify genetic influences (Barnes and Boutwell 2013; Boardman and Fletcher 2015; Conley et al. 2013; Felson 2014; Fosse, Joseph, and Jones 2016; Haberstick et al. 2015; Petersen et al. 2011).

Genes, Environment, and Educational Attainment: A Sociocontextual Perspective

A sociocontextual perspective acknowledges that genetic differences play a role in shaping a complex social outcome like educational attainment, but it emphasizes that genetic influence must be understood in context, such as history and cohort, the life course, and social forces like gender. This perspective posits that the influence of genetic factors on educational attainment is filtered, altered, and shaped by broader complex environments.

Genetic factors that predict education may vary over time in response to changes in the environment. For example, if schools systematically place a greater emphasis on math over reading skills, the genetic factors predicting overall academic performance might change (Boardman et al. 2013; Jencks 1980).

This perspective contextualizes individual experiences in historical periods and birth cohorts (Boardman et al. 2013; Schmitz and Conley 2017; Walter et al. 2016; Wedow et al. 2018). For example, a burgeoning literature examines how cohorts and historical periods modify the influence of genetic variants on health behavior outcomes like smoking, producing evidence that genetic factors have had greater influence in more recent cohorts as laws and social norms have made it harder to smoke (Domingue et al. 2016; Wedow et al. 2018). As for educational attainment, its social and economic value has grown over time, as a consequence of economic and cultural changes (Brown 2001). Consequently, the influence of genetics—when the outcome is historically and culturally contingent—is bound to change over time, according to this view. And though limited, there is some evidence to support the view that the relationship between genetic factors and educational attainment varies across cohorts (Branigan et al. 2013; Conley and Domingue 2016).

A sociocontextual perspective views environment as "longitudinal"—environments change over the life course, and they can vary as a function of the life course stage (Boardman et al. 2013). As Short, Yang, and Jenkins (2013:S93) note, there is an "intertwining of social and biological variation over the life course." Social and structural conditions change over the life course, as a function of age (e.g., the institutions that are dominant in our lives vary as we age) and historical change (e.g., different cohorts experienced the women's rights movement at different points in the life course).

To date, we know relatively little about the relationships between age, genetics, and educational attainment. Does the influence of genetic differences vary over the life course? Research shows that genetic influences on academic achievement change through childhood (Rice et al. 2018; Rimfeld et al. 2018), but no existing work explores how genetic influences vary on subsequent changes in educational attainment and outcomes through midlife. As we will detail, the process of obtaining postsecondary schooling continues through midlife, particularly for women.

Key to this viewpoint is that social forces, like gender, are "fundamental determinants of vulnerability and exposure"—the specific vulnerabilities and exposures are not fully separable from each other (Boardman et al. 2013:S65; Link and Phelan 1995). Gender, for example, is "a dynamic social, cultural, and institutional environment" (Short et al. 2013:S98), or "a social structure that is embedded . . . throughout social life" (Risman 2004:431). One need not disentangle this intricate web to demonstrate how gender may modify downstream genetic factors, ultimately altering their influence on a complex outcome like educational attainment. For example, gender influenced educational attainment throughout the first part of the twentieth century via institutional factors (e.g., women's restricted access to postsecondary schooling), cultural factors (e.g., social norms that ostracized women who attempted to pursue higher education), and social factors (e.g., the expectation of women's primary role in childrearing). The upstream influence, however, is gender.

Nonetheless, although a growing body of empirical work explores gene-environment interactions, especially in the context of cohort, the literature has much less to say about gender as a social construct—and its possible role in modifying genetic influences, not only on educational attainment, but on a wide array of other health, behavioral, and social outcomes (Short et al. 2013). But, as Short and colleagues (2013:S98) argue, gender differences can occur when "genetic variants are expressed in different contexts, biological or social, including 'gendered' environments."

A few additional clarifications are needed. A sociocontextual perspective does not

assume genetic differences do not matter, or even that their influence is especially limited. For example, the idea that genetic differences influence environments is not necessarily contrary to this perspective (Jencks 1980). The key is that the environment is still easily alterable (Jencks 1980). A recent study, for instance, found that students with the highest polygenic scores were more likely to be tracked into higher math levels in high resourced compared to low resourced schools, but students with high scores were equally likely to persist in these classes across low and highly resourced schools; students with lower polygenic scores, however, were more likely to persist in higher math levels in higher rather than lower resourced schools (Harden et al. 2019).

This perspective does not rule out "strong" genetic explanations. For example, Down syndrome has a "strong" genetic explanation that links a precise genetic mechanism to lower cognitive functioning and lower educational attainment. Yet even here, there is robust evidence of environmental modification. Children with Down syndrome largely did not learn to read 50 years ago, but because of the Disability Rights Movement, which led to the passage of laws stipulating that children with disabilities have a right to an education, a majority today do learn to read (Naess et al. 2012). That said, the sociocontextual perspective is likely more dominant for outcomes where there is a "weak" genetic explanation: "an outcome [that] has a heritable basis, but the mechanisms that transmit it are largely unknown" (Dar-Nimrod and Heine 2011:5; Turkheimer 1998, 2016).

The Dawn of the Polygenic Score

The major empirical constraint on the sociocontextual perspective has been the lack of data to explore the dynamic processes by which environment and genetic factors may interact to influence educational attainment. The mapping of the human genome and the rapid decline in the cost of genotyping has opened the door for new ways to examine how environments influence the role of genetics in behaviors of interest to social scientists (Conley and Fletcher 2017). This has led to the integration of genome-wide data into population-based studies commonly used to examine, among other things, gender differences in socioeconomic outcomes like educational attainment and earnings (Herd, Carr, and Roan 2014; McQueen et al. 2015; Sonnega et al. 2014).

Because any single genetic variant plays a limited role in complex health or behavioral outcomes (Chabris et al. 2015), there is a growing consensus around the development of polygenic scores to capture genetic "risk" (Boyle, Li, and Pritchard 2017; Dudbridge 2016; Krapohl et al. 2017). Genome-wide association studies find associations between the most common kind of genetic variation (specifically, single nucleotide polymorphisms [SNPs]) and outcomes ranging from height and cancers to personality and educational attainment. For each individual in these studies, somewhere between 1 and 2.5 million variants (SNPs) have been identified (Lee et al. 2018). The genome-wide analysis then identifies the strength of the associations between each variant and the outcome, like educational attainment. Large samples are needed because the estimated effect sizes for any particular variant are small. The study used to generate the education polygenic score included 1.1 million individuals (Lee et al. 2018). The polygenic scores are effectively a summary score for each individual that is based on the strength of the association between the genetic variants and the outcome, in this case educational attainment.

Polygenic scores that provide robust, out-of-sample predictions have been developed for outcomes such as height, body mass index (BMI), psychiatric disorders, and smoking (Belsky et al. 2012; Conley and Fletcher 2017; Domingue et al. 2014; Domingue et al. 2016; Kendler 2006; Koboldt et al. 2013; Liu and Guo 2015; Wood et al. 2014). The education polygenic score is comparable in its predictive strength to the polygenic scores for obesity (Cesarini and Visscher 2017), and to the predictive strength of one parent's

educational attainment in predicting a child's educational attainment (Lee et al. 2018), although there is debate over its multigenerational influences (Belsky et al. 2018; Liu 2018). To date, studies have demonstrated that the education polygenic score predicts educational attainment in entirely independent samples, including in samples that compare one full sibling to another (Domingue et al. 2015; Okbay et al. 2016; Rietveld et al. 2014; Selzam et al. 2017). Yet, although the predictive validity of the education polygenic score is well demonstrated, we are only just beginning to explore how social and environmental factors modify genetic influences on educational attainment.

GENDER INEQUALITIES IN EDUCATIONAL ATTAINMENT OVER THE TWENTIETH CENTURY: CHANGE ACROSS COHORTS AND OVER THE LIFE COURSE

Changing gender inequalities in educational attainment over the twentieth century are well-documented (Gamoran 2001). Gender differences have largely been driven by differences in postsecondary schooling, rather than differences in obtaining a high school degree. Until the 1960 birth cohort, women were far less likely than men to obtain bachelor's degrees (DiPrete and Buchmann 2013). Women's educational attainment lagged men's for decades due to institutional constraints ranging from outright discrimination to specific policies, such as the GI Bill, which overwhelmingly benefited men (Buchmann, DiPrete, and McDaniel 2008; Gamoran 2001). Weakening institutional constraints, increasing age at first marriage, and increasing opportunities for women in the labor force due to legislative and cultural changes all contributed to women's increased attainment of postsecondary schooling (Goldin, Katz, and Kuziemko 2006). Substantial complexity surrounding the mechanisms is embedded in this history, but the changes in gender differences in educational attainment over time show they are the result of alterable social conditions. The studies we use, specifically the WLS, HRS, and Add Health (b. 1931 to 1982), represent the cohorts that experienced this dramatic change over the course of the twentieth century.

The size of the gender gap in postsecondary attainment was at its peak in the 1930s. In the 1930 birth cohort, 17 percent of men obtained a college degree by age 28, compared to only 8 percent of women. The gap was just as large for the 1940 birth cohort, although the overall likelihood of obtaining a college degree increased. These differences were largely eliminated, however, for individuals born in 1959. The HRS sample includes birth cohorts from 1931 to 1959 and thus covers the era when the bulk of this shift occurred (Gamoran 2001).

Women in later cohorts faced a very different set of structural conditions and social expectations when they finished high school. The barriers women born in the 1930s through the 1950s faced, from sex discrimination in postsecondary admissions and in the workplace to a more robust gendered distribution of household labor, had weakened. Leaving high school, women in later cohorts could expect to access employment commensurate with their educational attainment, that marriage would not end their labor force participation, and that they would likely continue in the labor market even after having children (Goldin et al. 2006). Indeed, women began to surpass men's rates for bachelor's degree completion (DiPrete and Buchmann 2013). The reasons for women's increased attainment are well understood, but the flattening of men's attainment as women's attainment continues to grow is less clear and is increasingly a focus of research (Buchmann et al. 2008; DiPrete and Buchmann 2013; Fortin, Oreopoulos, and Phipps 2015; Legewie and DiPrete 2012). The Add Health participants are representative of this more advantaged group of women, having been born between 1975 and 1982.

Research shows robust differences across cohorts, but less often detailed are changes

that occurred over the life course of women who came of age when gender inequalities in college completion were at their peak, in the 1950s, but hit midlife in the 1980s, as gender inequalities in postsecondary schooling had substantially softened for younger women. The 1939 to 1940 birth cohort, of which members of the Wisconsin Longitudinal Study (WLS) are a part, provides a compelling case study for gender differences in educational outcomes. This cohort approached early adulthood during the period of peak gender differences in educational attainment, particularly postsecondary attendance and attainment. But by the time they were in their 30s and 40s, the postsecondary educational gap among younger cohorts was beginning to close and would eventually flip so that women, on average, obtained higher levels of education than men (Goldin 2006). As the WLS cohort approached middle age, women in their 20s were attending college at similar rates to their male peers. In summary, for women in the 1939 cohort, late adolescence and early adulthood was characterized by limited educational opportunities, but by their early middle age these opportunities were enhanced. Importantly, this intersected with the point in their life course when they were released from primary gendered responsibilities for raising children.

Indeed, there were gender differences in the age at which individuals completed schooling, particularly postsecondary schooling. Women in the 1939 to 1940 birth cohort were less likely to obtain college degrees before age 30 than were their male peers (during the late 1950s and 1960s), but by their 40s (during the 1980s) they went back and obtained postsecondary schooling at higher rates than did men. In the WLS, 25 percent of female college graduates, compared to 11 percent of male college graduates, earned their degree after age 30. This mirrors national postsecondary attendance statistics over this period (Levin and Levin 1991). In 1972, 25 percent of men and 21 percent of women engaged in postsecondary schooling were age 30 or older. By 1990, this had fallen to 21 percent of men but had risen to 31 percent of women (U.S. Census 2016). This likely is a function of both a period effect—that middle-aged women in the 1980s were experiencing the same reduction in structural constraints as were younger women—and an age effect—that middle-aged women were freed of gendered caregiving responsibilities for children, which had constrained them in their 20s and 30s.

GENE-ENVIRONMENT INTERACTIONS: DOES GENDER MODIFY THE INFLUENCE OF GENETICS ON EDUCATIONAL ATTAINMENT AND OUTCOMES ACROSS COHORTS AND OVER THE LIFE COURSE?

We have robust evidence that genetics influence educational attainment, and that structural and social factors reduced women's educational attainment, but we do not know whether the relationship between genetics and educational outcomes differs by gender. Examining changes across cohorts and over the life course provides a mechanism to consider how varying environments filter genetic influences on educational attainment.

Focusing on the 1939 to 1940 birth cohort, were genetic influences as consequential for women's educational outcomes as for men's? How did that influence vary over their life course? During their 20s, women in this cohort engaged in postsecondary schooling at lower rates than men, but while men's rates flattened in midlife, women's rates continued to grow—although they never did match men's attainment. Did genetic influences on educational outcomes change as women in this cohort hit midlife and gendered barriers to postsecondary schooling for women, both institutional and familial, began to fall? Evidence shows that genetic influences change over the life course, especially during childhood and adolescence, but no existing work explores how genetic influences vary on subsequent changes in educational attainment

through midlife, even though the process of obtaining postsecondary schooling continues through midlife.

While the WLS allows us to examine within cohort change, the HRS and Add Health allow for examining changes across cohorts. Do gender differences in the relationship between the education polygenic score and educational attainment and outcomes weaken in younger cohorts when the social and structural constraints that limited women's educational attainment weakened? While there is some evidence that the influence of genetics on educational attainment has changed across cohorts, we do not know whether this influence varies by gender.

A Sociocontextual Explanation: The Exceptional Is Suppressed

Social conditions, which vary across cohorts and over the life course within particular cohorts, may have influenced women's propensity to gain higher educational attainment via social constraints acting to undermine the genetic influence on women's educational attainment—decreasing the probability to realize their "actualized" potential as compared to men. In a widely-cited study, Turkheimer and colleagues (2003) report that with an economically diverse sample of twins, heritability of cognitive performance was highest among those raised in highly educated and well-off households, and it was lowest in less educated and poorer households. The explanation was that in highly resourced households, genetic factors could manifest into observable differences in cognition. In more poorly resourced households, however, resource constraints did not allow this particular talent to manifest (for earlier anticipations of this idea, see Rowe, Jacobson, and van den Oord 1999; Scarr-Salapatek 1971).

While no existing work uses direct genetic measures (specifically, genetic variants identified from large genome-wide association studies) to examine gender differences, there is some evidence from twin studies regarding gender differences in the heritability of educational attainment. For example, a recent meta-analysis of twin studies testing the heritability of educational attainment, which included published and unpublished findings, found that heritability estimates drawn from twin studies range from .18 to .77 (Branigan et al. 2013). On average, educational attainment was more heritable for men than for women, although the finding varied considerably across samples.

Cohort differences may explain this variance in findings regarding the influence of gender. Earlier work by Heath and colleagues (1985) found a pattern of results across cohorts that would support the hypothesis that gender—as a social force—modifies the influence of genetic factors on educational attainment. The heritability of educational attainment in Norway did not differ between men and women prior to 1940. However, as more generous education policies emerged after the war (cohorts born between 1940 and 1960), heritability increased among men yet stayed the same for women, creating a gender difference. Men were able to avail themselves of the new policies in ways women were not. This reduced environmental constraints for men, but not for women, resulting in genetics exerting a greater influence on educational attainment for men as compared to women (Heath et al. 1985).

The insights on gender differences from twin heritability studies are intriguing, yet their generalizability has been called into question (Barnes and Boutwell 2013; Petersen et al. 2011). For example, there is evidence that twins differ from non-twin children on traits such as language development, personality, and internalizing behavioral problems (Kendler et al. 1995; Pulkkinen et al. 2003; Shinwell, Haklai, and Eventov-Friedman 2009; Voracek and Haubner 2008). Most relevant for this study, there is some evidence that twins have lower levels of cognitive functioning, academic performance, and educational attainment compared to singletons, which may be partially a function of genetic factors (Tsou et al. 2008; Voracek and Haubner 2008).

The inferences drawn from this suppression model, however, would lead to the following hypotheses:

Hypothesis 1a: The education polygenic score better predicts educational outcomes for men than for women, as the structural and social constraints faced by women in the WLS cohort (born 1939 to 1940) suppress genetic influences on postsecondary schooling.

Hypothesis 2a: The gender gap in the influence of the education polygenic score on educational outcomes lessens over time as women in the WLS cohort went back and obtained more postsecondary schooling in midlife, reflecting declines in institutional and social barriers, ranging from changed social norms to women being released from gendered child caregiving responsibilities (as their children grew older).

Hypothesis 3a: Gender differences in the influence of the education polygenic score on educational outcomes will decline among the youngest cohorts in the HRS, with no evidence of a difference in the Add Health cohort.

"Strong" Genetic Effects

An alternative model, reflecting assumptions regarding "strong" genetic effects, predicts that genetic influences on higher education should prove resistant to environment. Indeed, while some twin studies back up Turkheimer and colleagues' (2003) findings that the heritability of cognitive performance is stronger among children from high-SES households compared to those from low-SES households, other studies have found the reverse (Asbury, Wachs, and Plomin 2005; see also the meta-analysis by Tucker-Drob and Bates 2016).

Findings from studies focused specifically on academic outcomes like reading ability and academic performance are mixed as to whether they support the suppression model (Daw, Guo, and Harris 2015; Figlio et al. 2017; Hart et al. 2013; Shakeshaft et al. 2013; Taylor et al. 2010; van den Oord and Rowe 1997). Friend and colleagues (2009) actually found higher heritability for high reading ability among children born to low-SES parents compared to those born to high-SES parents.

Regarding gender, some studies support a "strong" genetic explanation. For example, a sample of more than 13,000 twins in a 1990s birth cohort in the United Kingdom found no gender differences in the heritability of a range of education-related outcomes (Krapohl et al. 2014). Moreover, Baker and colleagues (1996), comparing twins in Australia born before and after 1950, found no gender differences in the heritability of educational attainment. There was also no gender difference in a Finnish 1936 to 1950 birth cohort (Silventoinen et al. 2004). And Petrill and Thompson (1994) found the heritability of scholastic achievement in secondary schooling was greater for women than for men.

A key limitation to these studies, however, it that they rely heavily on twin models and heritability differences to test for gender differences in genetic influences (Jencks 1980). The polygenic score, however, provides a more flexible test of the "strong" genetic effect proposition. A "strong" effect may be demonstrated if a set of variants predict divorce equally—across environments. A critical assumption, as Plomin (2018) argues, is that we not only will be able to "understand who we are" in the present, but we can predict "who we will become" in the future. Thus, even if "heritability" remains unchanged, if the underlying genetic mechanism predicting that outcome changes, or if the polygenic score varies in its ability to predict an outcome across "environments," this does not qualify as a "strong" genetic explanation.

Consequently, inferences drawn from the strictly "strong" genetic model would lead to the following hypotheses:

Hypothesis 1b: There are no gender differences in how the polygenic score for educational attainment predicts postsecondary school outcomes in the WLS.

Hypothesis 2b: The influence of the polygenic score for educational attainment does not vary in its influence on postsecondary school outcomes over the life course of WLS participants.

Hypothesis 3b: Gender differences in the influence of the education polygenic score on educational outcomes will show no evidence of change across cohorts based on the HRS and Add Health data.

DATA AND METHODS

Data

WLS. The Wisconsin Longitudinal Study (WLS) allows us to examine, within a single cohort born from 1939 to 1940, whether there are gender differences in how the relationship between the education polygenic score and educational attainment changes within individuals over the life course. HRS and Add Health (detailed below) only allow for educational attainment measured at a single point in the life course. WLS is a one-third sample of all 1957 Wisconsin high school graduates (Hauser et al. 1992; Herd et al. 2014). These respondents were originally empaneled with an in-person questionnaire at age 18 (1957), which was followed with a mail survey of parents in 1964, a telephone survey in 1975, mail and telephone surveys in 1993 and 2004, and in-person interviews in 2011. The WLS has a high response rate, exceeding 80 percent in most rounds of data collection. Data, documentation, and other material are available at http://www.ssc.wisc.edu/wlsresearch/.

The WLS includes a wide range of administrative and prospectively collected data from early life through adulthood. The original 1957 survey collected information regarding graduates' high school experiences, including administrative data such as the Henmon-Nelson IQ test and high school rank. Relevant for our analyses, these data provide a full record of post high school education, making the dataset uniquely suited for testing changes in educational attainment over the full life course.

From 2006 to 2007, WLS first collected saliva samples from respondents using Oragene kits and a mailback protocol patterned closely on a previous study (Rylander-Rudqvist et al. 2006). Additional sample collection was conducted in 2011 during in-person interviews for participants who did not submit samples in 2006 to 2007. Compliance to the DNA request was about 5 percentage points higher among men but broadly matched response rates for other data in the WLS (Herd et al. 2014). After quality control, a total of 9,012 graduate and sibling respondents were genotyped at ~710,000 markers (before imputation) utilizing the Omni-Express BeadChip. Only graduate respondents were included in this study; thus, the analytic sample included 5,654 participants, which constitutes 56 percent of the original sample frame, or 69 percent of surviving participants. It is important to keep in mind that the WLS is unusual in having a 100 percent response rate from the sample frame. The highest initial response rate for an HRS cohort, for example, is 80 percent for the 1931 to 1939 birth cohort. Consequently, the total response rate is higher in the WLS, even as the length of the study (60 years) is substantially longer.

Genotyping was completed at Johns Hopkins' Center for Inherited Disease Research (CIDR), and data cleaning was performed in collaboration with the Genetic Analysis Center at the University of Washington. The detailed procedures used to generate these data are available on the WLS website (https://www.ssc.wisc.edu/wlsresearch/documentation/GWAS).

HRS. The Health and Retirement Study (HRS) provides data to examine whether gender differences in the relationship between the education polygenic risk score and educational attainment changes across cohorts. The original sample, interviewed in 1992 and every subsequent two years, includes individuals born between 1931 and 1941. Refresher cohorts were added in 1998 (War Babies, b. 1942 to 1947), 2004 (Early Baby Boomers, b. 1948 to 1953), and 2010 (Mid Baby Boomers, b. 1954 to 1959). We excluded the older cohorts (b. before 1930) to keep the analysis comparable with the WLS cohort. The older cohorts also suffer from significantly more mortality selection. Response rates for these cohorts were 81 percent, 70 percent, 75

percent, and 69 percent, respectively. Genetic data are available for 62 percent of the entire HRS sample, or over 80 percent of the sample that had survived and had been retained to the period when saliva samples were collected. Of the 14,774 respondents with genetic data, collected between 2006 and 2010, we used 9,073 individuals of European and non-Hispanic descent (Ware et al. 2018). Genetic data from 404 white non-Hispanic individuals were not usable due to quality control issues. Given the risk of non-response and mortality selection, weights were developed to specifically account for these potential biases in the sample with genetic data (Domingue et al. 2017). Detailed protocol on the saliva collection can be found in Crimmins and colleagues (2015). Genotyping was done by the Center for Inherited Disease Research (CIDR) using the illumina HumanOmni2.5 BeadChip. Imputation was conducted by the University of Michigan. Detailed information on the HRS biosamples can be found in Ware and colleagues (2018).

Add Health. Add Health allows us to examine cohort differences by adding the youngest cohort as compared to HRS (b. 1931 to 1959). Add Health is a nationally representative cohort drawn from a probability sample of 80 U.S. high schools and 52 U.S. middle schools, representative of U.S. schools in 1994 to 1995 with respect to region, urban setting, school size, school type, and race or ethnic background (n = 20,745 at Wave 1 in 1994 to 1995). Waves 3 (2001 to 2002) and 4 (2008 to 2009) data collections included 15,197 (mean age 22.3 years) and 15,701 (then ages 24 to 32 years, mean age 28.9 years) individuals, respectively. The sample was genotyped (via Oragene saliva collection) at Wave 4 of the study (for details, see Belsky et al. 2018; Domingue et al. 2018). Among participants, 15,072 provided saliva samples and 12,058 provided consent for genotyping. After quality control during genotyping, 9,975 of these were retained. This left 5,692 participants who had European ancestry and were non-Hispanic. The final sample size was 5,514 after restricting it to respondents with valid educational data and who were born prior to 1983, to ensure individuals were a minimum of 25 years old. The resulting sample is comparable to the overall Add Health sample (Domingue et al. 2015).

Measures

Outcomes in WLS. The first detailed educational attainment report in WLS came from parents in 1964. In 1975, respondents themselves reported on all schooling obtained since graduating high school. In every subsequent wave, participants were asked retrospective questions regarding any changes in their educational attainment (including additional years of schooling, even if no additional degree was obtained). The collected measures also capture starting and ending dates for postsecondary schooling. Over half of the sample did not pursue education past high school. We use three different measures: "years" of schooling, which is a summary measure derived from highest degree of postsecondary schooling, whether an individual engaged in any postsecondary schooling, and whether an individual attained a four-year bachelor's degree.

Outcomes in HRS. The HRS collected educational attainment data on each new cohort when they were first interviewed (in 1992, 1998, 2004, and 2010). We measure education based on the number of years of schooling and highest degree attained. The measure was harmonized to replicate the WLS measure of "years of schooling."

Outcomes in Add Health. Because this cohort is younger than the WLS and HRS cohorts, we only include respondents who are at least age 25 or were born before 1983. We measure educational attainment as the highest degree completed by the time of interview at Wave 4, when respondents were asked, "What is the highest level of education that you have achieved to date?"[2]

Covariates

Polygenic score for educational attainment. Polygenic scores summarize predictive information in the genome with respect to a particular trait. The scores use weights based on genome-wide association studies (GWAS) conducted in other samples. The polygenic score for educational attainment that we use is based on a 1.1-million-person GWAS (Lee et al. 2018).[3] Weights of individual variants are multiplied by the count of trait-associated alleles for each SNP and summed across all variants. Detailed information on construction of the education polygenic scores can be found in supporting documentation on the WLS website (https://www.ssc.wisc.edu/wlsresearch/documentation/GWAS) as well as in additional papers (Belsky et al. 2018; Domingue et al. 2015; Trejo et al. 2018). The score is standardized to have a mean of 0 and SD of 1. Note that the GWAS study that was used to develop the polygenic score had less representation of younger cohorts (Lee et al. 2018). It is thus possible that for the Add Health cohort, relevant variants specific to younger cohorts in predicting educational attainment are not included in the score. However, if a lack of a gender difference in the predictive capacity of the education polygenic score in Add Health is, in part, due to a cohort difference in the genetic variants that predict educational attainment, the broader hypothesis—that environments modify genetic influence—still holds true. That said, as we will detail, the robustness of the relationship between the polygenic score and educational attainment is comparable across these studies in aggregate.

Figure A.1 in the online supplement shows the relationship between the polygenic score for educational attainment and educational attainment in the WLS. Individuals who completed only high school, for example, have a mean PGS of –.17. In contrast, respondents who completed college have a mean score of .34. Figure A.2 in the online supplement shows a robust relationship in the HRS. Figure A.3 presents the same analysis for Add Health, similarly showing a robust relationship between the polygenic score and educational attainment in this sample.

Additional covariates. Additional variables include age and biological sex. We also include controls for the first 10 principal components estimated from the genome-wide SNP data to account for allele differences across ancestral groups (population stratification) in our analytic sample (McQueen et al. 2015). Population stratification is a key issue in studies of this kind. In short, there is the risk that results are confounded by ancestry differences. Even in the Wisconsin Longitudinal Study, which includes respondents largely of Northern European ancestry, there are clear ancestral differences, for example, between individuals of Polish versus British descent (see Figure A.4 in the online supplement). Consequently, study findings, even in the WLS, can be sensitive to the inclusion of controls for population stratification.

Due to known problems associated with the application of polygenic scores in diverse populations (Martin et al. 2017), we limit our analyses to individuals who self-identify as non-Hispanic white and whose genotypes are consistent with European-ancestry populations. We do so because of potential concerns with population stratification and because patterns of linkage disequilibrium (LD)—the correlation between nearby genetic variants—vary considerably across socially defined racial and ethnic groups. Most notably, groups with significant African ancestry have more genetic variation and thus lower LD, which creates problems for comparing associations of SNPs across groups—a problem that is compounded when creating genome-wide polygenic scores.

Statistical Model

Our analysis of polygenic scores involved running OLS or logistic models (as appropriate for the outcome), stratified by gender, based on the following form:[4]

$$\mathrm{E}\left(y_i|\mathrm{PGS},\mathrm{X}\right) = \mathrm{f}\left(b_0 + b_1 \times \mathrm{PGS}_i + \mathrm{X'B}\right)$$

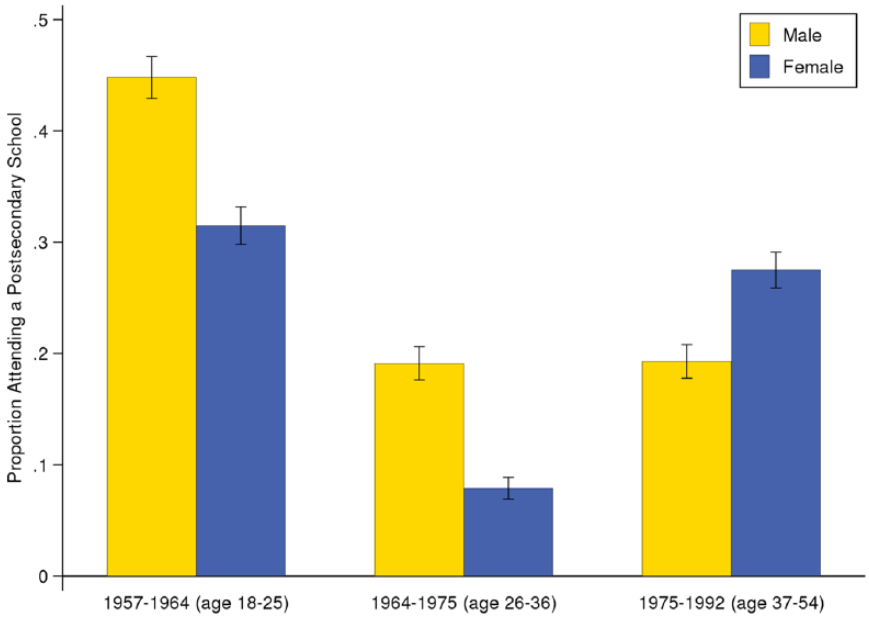

Figure 1. Gender Differences in Enrollment in Postsecondary Schooling in the WLS

where y_i is the outcome, in this case educational attainment; b_1 is the polygenic score; and X is the matrix of control variables, which includes age and controls for population stratification (10 principal components). Analyses in the HRS are weighted to account for nonresponse bias and mortality selection. The complex survey design, as well as differential response rates across cohorts, make this a critical adjustment in the HRS. Tables A.6a and A.6b in the online supplement present weighted and unweighted analyses for the HRS. The differences in the estimates are nearly exclusively driven by non-response, which varied across cohorts, and complex sample survey design, not mortality (Domingue et al. 2017).

RESULTS

Gender Differences in Educational Attainment in the Wisconsin Longitudinal Study (WLS) Cohort

Figures 1 and 2 present gender differences in educational attainment over the life course of WLS participants. These differences are similar to trends for the broader cohort. Figure 1 presents gender differences in postsecondary schooling attendance, not completion. Women's enrollment, although lower than men's in early life, was higher than men's in midlife, which reflects women's delayed postsecondary schooling pattern. Between ages 18 and 25, 45 percent of men were enrolled in postsecondary schooling, compared to just over 31 percent of women. Between ages 26 and 36 the fraction of men and women enrolled was 18 and 9 percent, respectively. By midlife (ages 37 to 54), however, a higher proportion of women than men were enrolled (27 versus 19 percent). Women's enrollment levels in midlife were almost equivalent to what they had been in their 20s.

Enrollment in postsecondary schooling tells us part of the story, but completion of a degree, particularly a bachelor's degree, is also important for gender differences in educational attainment for this cohort. As Panel a in Figure 2 details, men were significantly more likely to earn a college degree. Yet, many participants did not actually attain college degrees until they were in their 30s and 40s. Just 30 percent of men and 18 percent of women had achieved a college degree prior to

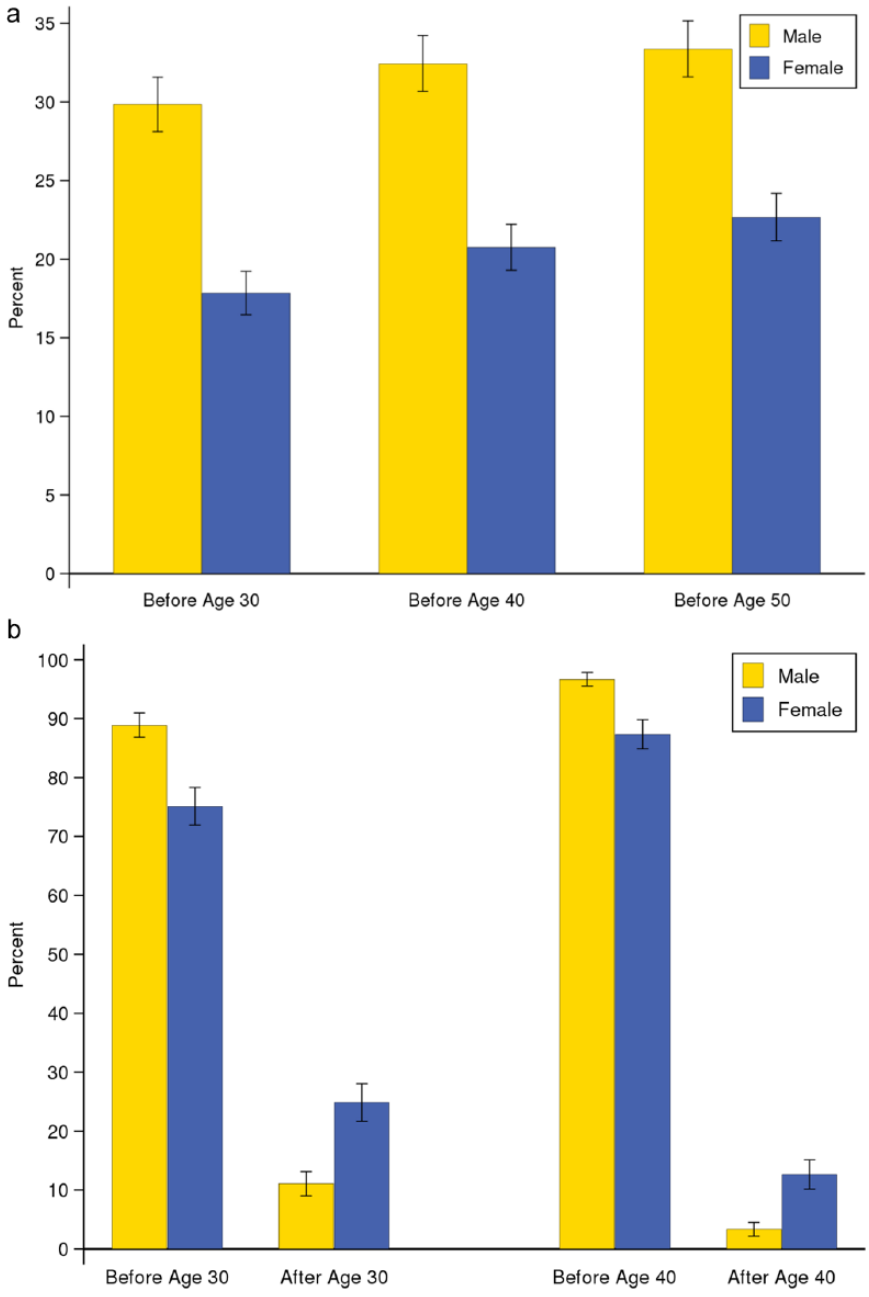

Figure 2. College Degree Attainment and Timing of Attainment, by Gender in the WLS
Panel a. Percent with a Bachelor's Degree
Panel b. Timing of Degree among Respondents with a Bachelor's Degree

age 30. By age 50, however, 33 percent of men and 23 percent of women had obtained a college degree, slightly shrinking the educational attainment gap between men and women. Panel b in Figure 2 further clarifies gender differences in the timing of college degree completion. Among respondents who attained college degrees, women were significantly more likely than men to have attained that degree at older ages, with 25 percent of

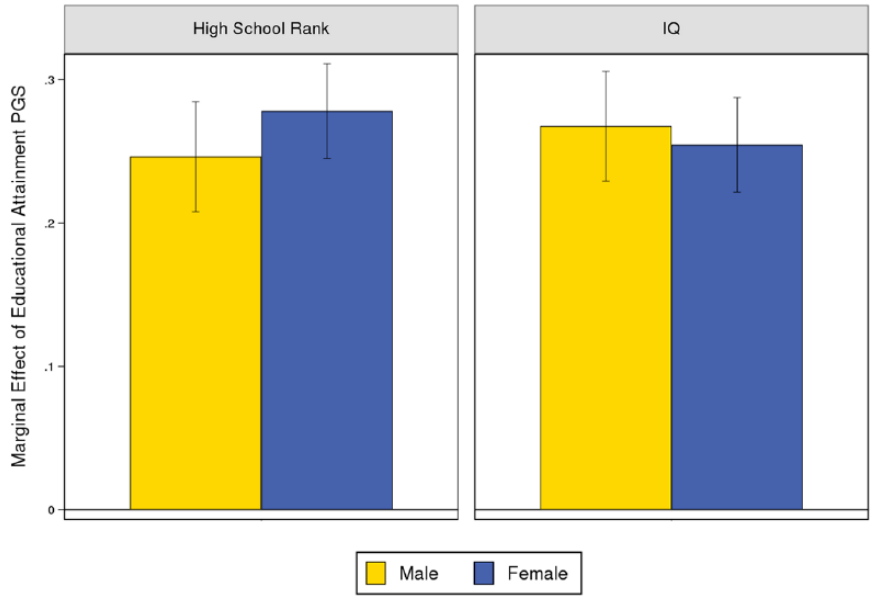

Figure 3. The Education Polygenic Score Regressed on High School Rank and Adolescent IQ in the WLS

women and 11 percent of men obtaining their degree after age 30.

Gender, the Education Polygenic Score, and High School Academic Ability in the WLS

Academic performance in high school and general cognitive ability are two of the strongest predictors of college attendance and completion (Borghans et al. 2016; Zax and Reese 2002). Figure 3 (estimates provided in Table A.1 in the online supplement) shows that, in the WLS, a standard deviation increase in the education polygenic score is associated with a range of about .25 to .28 difference in cumulative high school rank and Henmon-Nelson IQ scores. However, we find no evidence of gender differences in the predictive value of the education polygenic score for either.

Gender Differences in the Polygenic Score's Prediction of Educational Outcomes in the WLS

We test whether gender modifies the relationship between the education polygenic score and educational outcomes in Figures 4, 5, and 6. We find support for Hypothesis 1a, not Hypothesis 1b: the relationship between the education polygenic score and schooling is larger for men than for women. Figure 4 (estimates provided in Table A.2 in the online supplement) provides the marginal effects produced from models using the education polygenic score to predict educational attainment at ages 25, 36, 54, and 65. The effect is stronger for men than for women regardless of age. Even so, patterns differ for women and men as they age. For men, the relationship between the education polygenic score and educational attainment strengthens considerably between ages 25 and 36, but it is largely stable thereafter. For women, the increased association between ages 25 and 36 is more moderate, but it strengthens again between ages 36 and 53, enough so that the difference between ages 25 and 54 is statistically significant ($p < .001$).[5]

In support of Hypothesis 2a, rather than Hypothesis 2b, we found gender differences in the relationship between the education polygenic score and going back for any kind of postsecondary schooling over the life

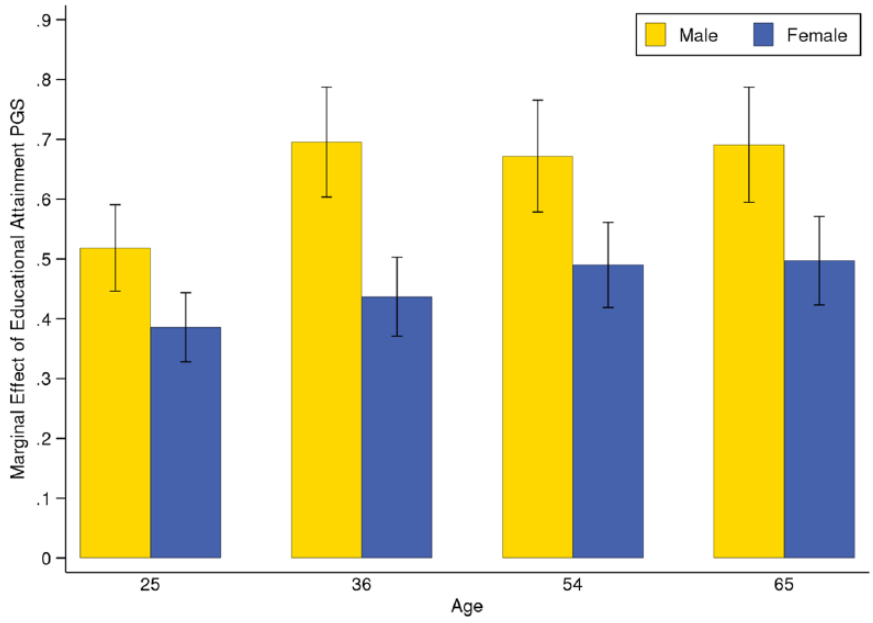

Figure 4. Gender Differences in the Relationship between the Education Polygenic Score and Years of Schooling over the Life Course

Note: Vertical lines depict the 95 percent confidence interval with robust standard errors. The models control for age and the first 10 principal components of the genomic data. Sample is limited to individuals of European ancestry.

course. Panel a in Figure 5 (estimates provided in Table A.3 in the online supplement) shows how the education polygenic score predicts enrollment in any postsecondary schooling between the ages of 18 to 25, 25 to 36, 36 to 53, and 53 to 65. From ages 18 to 25 (1957 to 1963) and 25 to 36 (1964 to 1975), the education polygenic score is more predictive of men going back to school than it is for women going back to school, with evidence of a larger gap among individuals with the highest genetic propensity for obtaining higher levels of educational attainment. From age 36 to 53 (1976 to 1992), the reverse is true: the education polygenic score is more predictive of enrollment in any postsecondary schooling for women than for men. Moreover, the gender gap is widest among respondents with the highest genetic propensity for higher educational attainment. The same trend, although much weaker, holds for ages 53 to 65 (1993 to 2004).

Panel b in Figure 5 (estimates provided in Table A.4 in the online supplement) details gender differences in the probability of obtaining an additional degree at different points in the life course, rather than any engagement in postsecondary schooling. Note that the range is smaller for panel b, 0 to .4, compared to panel a, 0 to .8. The pattern is nearly identical except for ages 18 to 26. Supplemental analyses indicate that this is influenced by women obtaining two-year degrees (for fields like nursing) during this period. In summary, the findings presented in Figures 4 and 5 support hypotheses that women's genetic potential for higher educational attainment was suppressed.[6]

How does the shift in the relationship between gender, the polygenic score, and obtaining more education in midlife influence the magnitude of overall gender differences in the relationship between the polygenic score and educational attainment? Figure 6 shows the percentage of men and women obtaining a college degree across sextiles of the education polygenic score. The bars are segmented based on whether the degree was

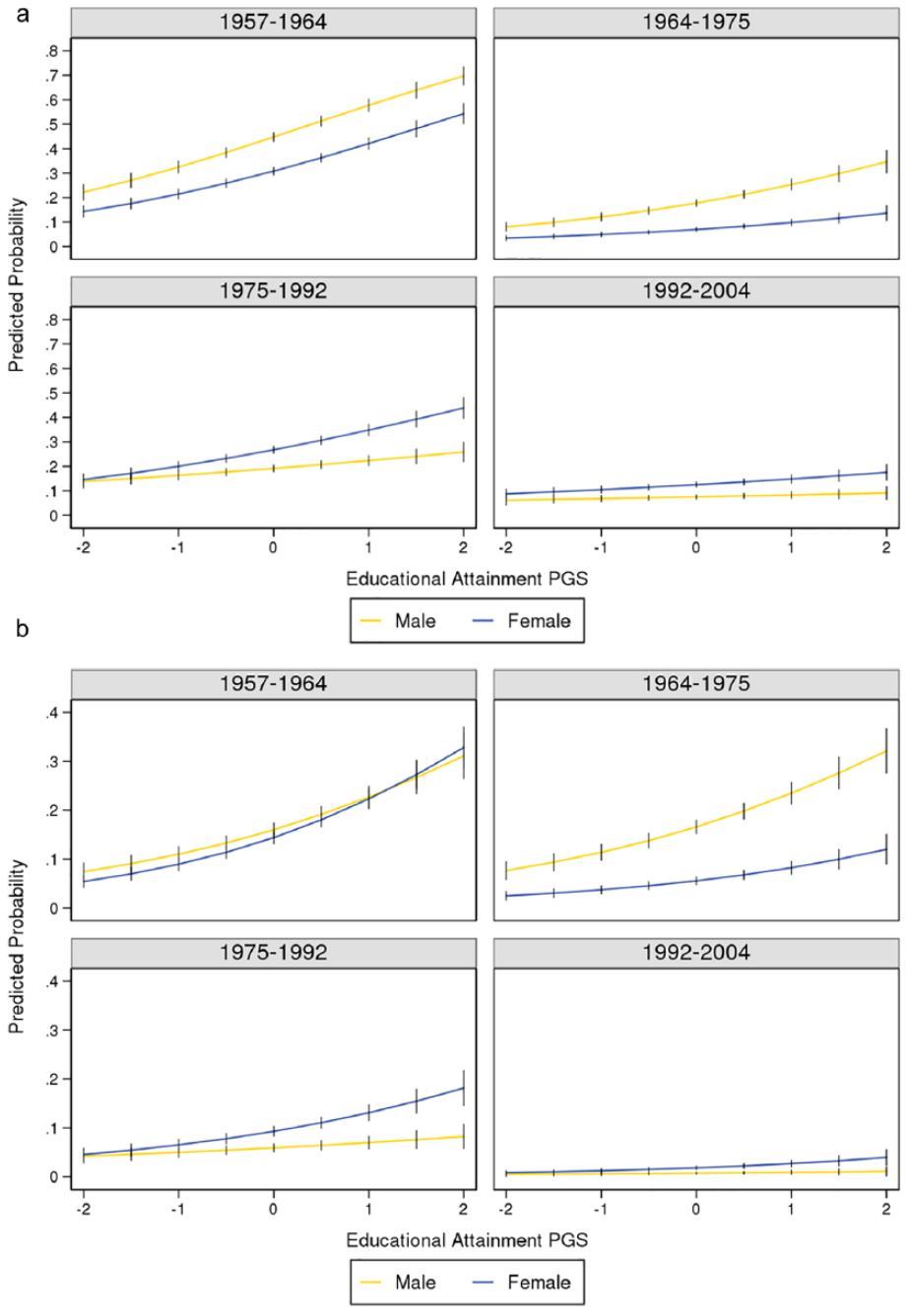

Figure 5. Predictive Value of Education Polygenic Score over the Life Course, 1957 to 2004
Panel a. Returning for Any Postsecondary Schooling
Panel b. Obtaining an Additional Degree

completed prior to, or after, age 30. This figure shows that gender differences in the strength of the relationship between the education polygenic score and completing a bachelor's degree are slightly smaller once you account for respondents who obtained a college degree after age 30. The reduction is especially present in the top of the education

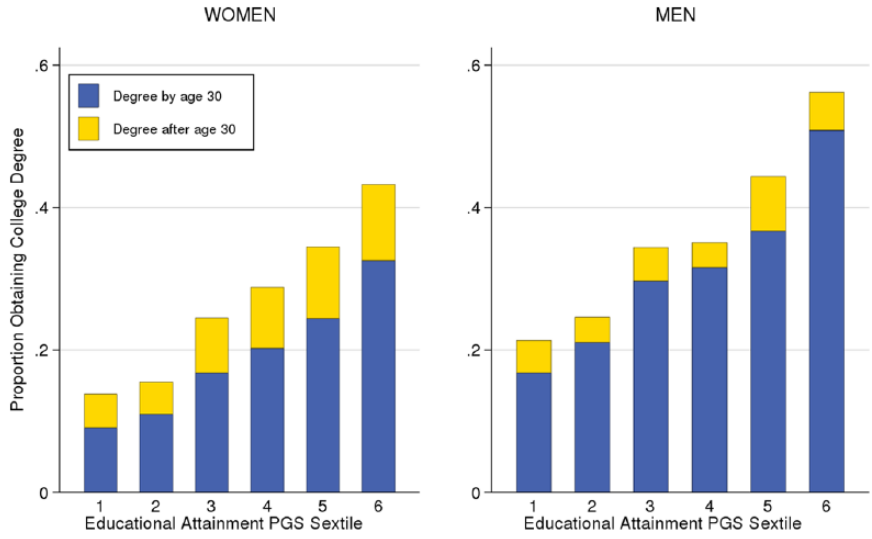

Figure 6. Proportion Finishing a Bachelor's Degree, by Gender and Education Polygenic Score Sextiles

polygenic score distribution, with little to no change in the difference at the bottom. The difference in the gender gap declines by 1 percentage point at the first sextile and there is no difference at the second sextile. This compares to a 3 and 5 percentage point decline in the gender gap at the fifth and sixth sextiles. In percent terms, this is a difference of a 6 percent versus 25 percent reduction in the bottom and top two sextiles, respectively. The difference at the top sextile, after accounting for individuals who obtained a college degree after age 30, was statistically significant ($p < .01$).

Gender Differences in the Polygenic Score's Prediction of Educational Attainment in the Health and Retirement Study (HRS)

The HRS allowed us to test Hypothesis 3a, specifically, whether gender differences in the relationship between the education polygenic score and educational attainment weakened across cohorts, rather than within a cohort. We found evidence for such a decline, which better supports Hypothesis 3a rather than 3b. The HRS includes individuals born between 1931 and 1959. Figure 7 depicts estimates based on five-year rolling cohorts. For example, the coefficient for 1931 is an average of coefficients from 1931 to 1936. The shaded areas represent 84 percent confidence intervals; thus, when the shaded areas do not overlap, the differences are significant at the $p <$ .05 level (see Table A.7 in the online supplement for point estimates and 95 percent confidence intervals). The findings generally demonstrate that gender differences in the relationship between the education polygenic score and educational attainment varied across cohorts, with a more robust positive relationship for men compared to women evident among older cohorts, and the relationship reversing among the youngest cohorts. The gender differences were statistically significant ($p < .05$) for the 1938 to 1943 birth cohort, with a more robust positive relationship between the education polygenic score and educational attainment for men than for women. The pooled estimates show that gender differences were statistically significant ($p < .05$) for the full 1931 to 1941 HRS birth cohort, reflecting the reduced standard errors due to the larger sample (see Table A.6a in the online supplement).

By the 1952 to 1957 birth cohort, this relationship had reversed, with a stronger positive

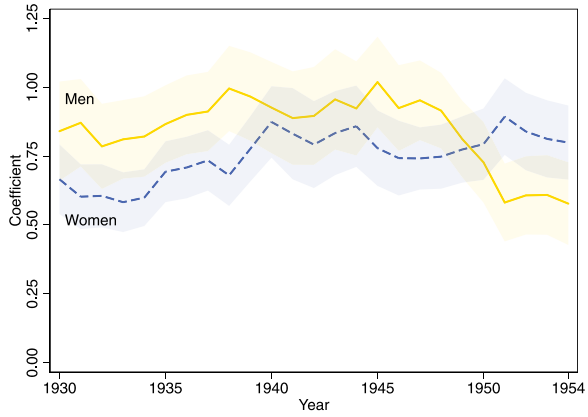

Figure 7. Relationship between the Education Polygenic Score and Years of Education in the Health and Retirement Study, Five-Year Rolling Cohort Windows
Note: The coefficients reflect five-year rolling cohorts. For example, 1930 is an average of 1930 through 1935, and 1931 is an average of 1931 through 1936. The shaded areas represent 84 percent confidence intervals; no overlap thus approximates a difference in coefficients at the $p < .05$ level.

relationship for women than for men. The estimates produced from rolling cohorts parallel the HRS defined cohorts (for detailed estimates, see Table A.6a in the online supplement). One important caveat is that the HRS only measured educational attainment when individuals had reached late midlife. As we showed in the WLS, when capturing within-individual changes over the life course in schooling, the differences between men and women varied over the life course, but were weakest in midlife, at least for the 1939 birth cohort. The pattern, however, of higher fractions of women compared to men engaged in postsecondary schooling at later ages continued for later cohorts, which suggests this pattern might hold for younger cohorts as well (U.S. Census 2016). In summary, we could infer, based on the WLS analyses, that the gender differences might be stronger in the HRS if educational attainment was measured earlier in the life course.

One of the more striking trends in this set of analyses is the large decline in the relationship between the education polygenic score and educational attainment for men, which is largely driven by the 1954 to 1959 birth cohort (the difference in coefficients for men in the 1954 to 1959 [$b = .577$] birth cohort compared to the 1948 to 1953 [$b = .915$] birth cohort is significant at the $p < .05$ level). This decline actually meant that the relationship between the polygenic score and educational attainment was larger for women than for men. This may reflect that the 1954 to 1959 male birth cohort, unlike the two prior cohorts, was not subject to a draft or military service for the Vietnam War, and thus did not have the same incentive to engage in postsecondary schooling. There is evidence that the draft, and to a lesser extent GI benefits, increased postsecondary schooling for individuals in the 1948 to 1953 cohort, although the decline in postsecondary schooling for men in the 1970s was related to changing economic conditions (Card and Lemieux 2001). As shown in Table A.5 in the online supplement, there is evidence of a flattening of the increasing trend in postsecondary schooling attainment among men for the cohorts that came of age in the early and mid-1970s in the HRS, as well as in Census data (CPS 2019). It is possible that a combination of the ending of the draft and weakening economic conditions in the 1970s decreased men's incentive to participate in postsecondary schooling, thus weakening the relationship between the polygenic score and attainment for men. That said, Tables A.6a and

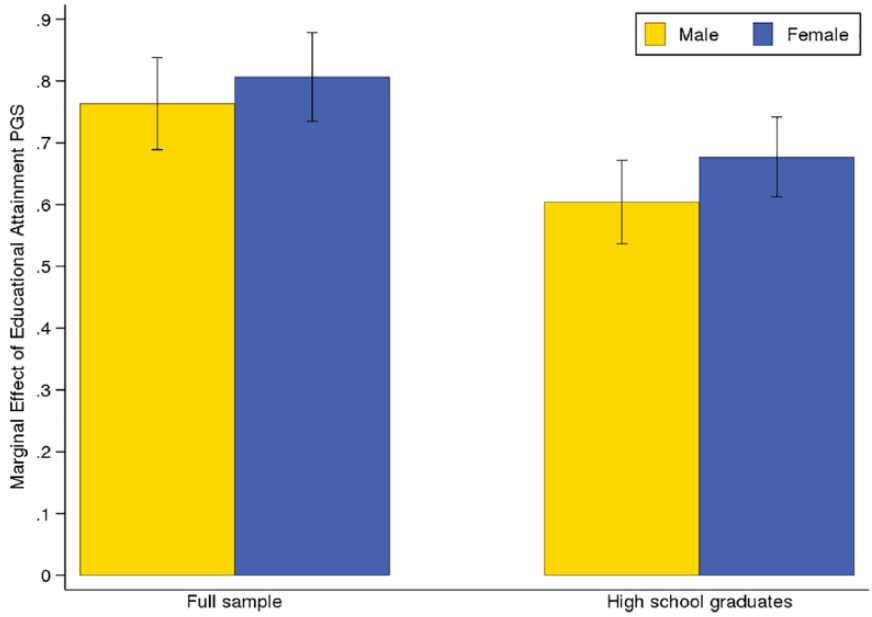

Figure 8. Relationship between the Education Polygenic Score and Years of Education in Add Health

Note: The education polygenic risk score has been residualized using the first 10 principal components of the genomic data. Sample is limited to individuals of European ancestry.

A.6b in the online supplement show that estimates for men in this cohort are quite sensitive to weights that account for selection and attrition. A replication of the results, in a different U.S.-based sample, for the 1954 to 1959 male birth cohort would be a valuable test.

Gender Differences in the Polygenic Score's Prediction of Educational Attainment in Add Health

Add Health (b. 1975 to 1982) allows us to examine cohort differences using the youngest available cohort. The social and structural constraints that hampered women's educational attainment have effectively been removed for this cohort. Indeed, as of Wave 4, for the 1982 birth cohort, 56 percent of women and 44 percent of men had completed a college degree. Although we cannot assess changes in midlife for this cohort, as they are not old enough, we can assess gender differences in the strength of the relationship between the education polygenic score and educational attainment for these respondents in their late 20s and early 30s. We find support for Hypothesis 3a rather than 3b: there are no gender differences in the relationship between the education polygenic score and educational attainment. Figure 8 shows the relationship for respondents with high school degrees, as well as the relationship for the full cohort, including respondents who did not complete high school.[7]

DISCUSSION

This study provided a novel analysis testing how history and cohort, the life course, and social structures like gender influence the relationship between the education polygenic score and educational attainment. Our results indicate that the role of genetics in shaping educational attainment is strongly patterned by gender, a social structure embedded throughout social life (Risman 2004). Within the WLS cohort (~1939), the relationship between genetics and educational outcomes is

weaker for women than for men, especially between the 1950s and 1960s when a series of structural and social barriers limited women's engagement in higher education. However, the relationship between genetic factors and education strengthened for women in middle age as they went back to school as their childrearing responsibilities wound down and new schooling opportunities emerged. Furthermore, analyses of the HRS (1931 to 1959) and Add Health (1975 to 1982) cohorts demonstrate that gender differences in how well polygenic information predicts educational attainment weakened substantially among younger cohorts. There is little evidence, among the youngest cohorts, that genetics has a greater influence on men's versus women's postsecondary schooling outcomes.

These findings provide more support for sociocontextual models that suggest environments play a large role in modifying the influence of genetics on educational attainment, rather than for strong genetic models that assume environments do not meaningfully alter genetic influences on education. We also find evidence that contextual factors may suppress genetic advantages. Larger social constraints reduced genetic influences on women's postsecondary educational outcomes, thus decreasing their probability of realizing their "genetic" potential as compared to men. Only when social conditions were altered to reduce the structural and social barriers limiting women's participation in higher education did gender differences weaken.

For the WLS cohort, changes in social and structural conditions that played out over their life course occurred at multiple contextual levels, as gender is embedded throughout social life (Risman 2004). In particular, the strengthening genetic influence on educational attainment among WLS women during their 40s and 50s may, in part, reflect an age and social context effect: specifically, women were released from key gendered social constraints linked to childbearing and childrearing as their childbearing years ended and their children got older. This age-specific experience occurred in the context of a particular period that saw a rapid increase in women obtaining postsecondary schooling. This return to schooling for older women is reflected in larger trends. In the early 1960s, 9.2 percent of male college students were age 30 and older, compared to just 3.8 percent of female college students. By the mid-1980s, 25.3 percent of female college students were age 30 and older, compared to 16.7 percent of male college students (CPS 2017). The influence of genetics on educational attainment for this cohort played out, and varied, in the context of how gender structured age and period effects.

The approach and methods used here reflect current scientific standards, but some caveats apply, not just to this analysis, but to most existing work that uses polygenic scores to examine gene by environment interactions. Polygenic scores might simultaneously underestimate the influence of both environment and genetic differences on outcomes. Regarding the former, current GWAS that are used to generate polygenic scores include samples across multiple cohorts, environments, and periods. Some scholars have speculated that this approach leads to polygenic scores disproportionately reflecting genetic variants that are least likely to be influenced by broader social conditions and environments (Conley 2016). To address this, researchers have begun to propose GWAS that, rather than predicting means, predict high levels of variance in the relationship between genetic variants and outcomes (Al Kawam et al. 2018; Conley et al. 2018). The implications of this for our study are that we may have produced a lower-bound estimate of the role of gender in modifying the relationship between the education polygenic score and educational attainment.

Furthermore, existing heritability estimates from twin studies would indicate that the education polygenic score used in these analyses explains only part of the estimated total genetic influence on education (Cessarini and Visscher 2017). The polygenic score used in this study predicts educational attainment about as well as one can by using the attainment of one parent (but not both; Lee et al.

2018). An important test going forward will be whether larger GWAS sample sizes identify additional genetic variants linked to educational attainment that explain even more of the variance (McClellan, Lehner, and King 2017; Visscher et al. 2017). Nonetheless, there is little reason to believe that underestimates of heritability would substantially affect the relative differences by gender that we observe.

Another concern is the possibility that genetic variants linked to education in the WLS cohort might be different than those in the Add Health cohort. The youngest cohorts were somewhat underrepresented in the GWAS that produced the education polygenic score (Lee et al. 2018). That said, the overall strength of the relationship between the polygenic score and educational attainment was comparable between the WLS and Add Health. Moreover, the findings from HRS, given that individuals from comparable birth years to the HRS cohorts were better represented in the education GWAS, further support the cross-cohort differences we find. Finally, even if genetic mechanisms changed over time, a major point of this article is that genetic influences are filtered through social conditions. Even if the sum genetic influence did not vary across cohort/gender, a changing relationship between the polygenic score used in this article and schooling outcomes (e.g., in Add Health compared to WLS) would instead show that the genetic mechanisms do vary (e.g., a "weak" genetic effect).

One of the most promising areas of genetics research going forward will be to formally test whether genetic variants linked to varying phenotypes change over time. Existing empirical work has not had the statistical power to formally test whether there are cohort differences in the specific genetic variants linked to educational attainment, so this presents a significant opportunity to disentangle gene-environment interplay (Lee et al. 2018). A recent *ASR* article has proposed a novel method (rGxE) that, as larger datasets become available, can help parse out whether genetic mechanisms vary across cohorts (Wedow et al. 2018).

A significant weakness—of both this article and social genetics research more broadly—is the lack of attention to populations outside of European ancestry. Our analysis focused on European ancestry populations because nearly 90 percent of individuals in genome-wide association studies and genotyped samples have European ancestry (Mills and Rahal 2019). Note, however, that even as calls for a broader inclusion of ancestry groups in genetic studies grows, we would not focus on "race" differences because race is a social, rather than biological, construct (Lee 2009). The resurgence in "race" genetics is deeply concerning, including researchers using genetic data to make claims about white supremacy. Indeed, the American Society of Human Genetics was recently compelled to release a statement denouncing "attempts to link genetics and racial supremacy" (ASGH 2018:636).

Finally, although we cannot unequivocally rule out some underlying biological explanation linked to sex for these findings, we would argue this is unlikely for four reasons. First, the dramatic change in the relationship between gender and educational attainment over the past 50 years, a relatively short period, undermines the possibility of genetic sex differences or evolutionary explanations. Second, when the education polygenic score is separately derived for women versus men, the correlation between the two is nearly one, thus indicating that the underlying mechanisms are quite similar for men and women (Lee et al. 2018). Third, the change in the relationship between the educational polygenic score and educational attainment within a single cohort as that cohort ages further weakens support for biological claims. Fourth, the empirical tests in this article demonstrated that there were no gender differences in how the education polygenic score predicted the capacity for higher educational attainment, as measured by adolescent IQ and high school academic performance.

CONCLUSIONS

What are the broader implications of this research? Educational attainment remains one of the most studied social determinants and outcomes in social science research. Yet,

examining the influence of genetics on educational attainment remains controversial. These results, however, provide evidentiary support for a relatively limited role of genetics: genetics appear to account for 9 to 12 percent of the variance in educational outcomes, and the evidence points to how the influence of genetics cannot be understood independent of social structures, like gender.

The discomfort in the social sciences regarding the use of genetic data to study questions related to educational inequalities is grounded in social sciences' role in the eugenics movement, as well as the ongoing attempts to use these data to justify existing inequalities as rooted in genetics (Bliss 2018; Duster 2004; Roberts 2011). White supremacists, including those in academia, are using genetic data, falsely, to support their claims. For example, they claim racial differences in intelligence based on genetics, a strategy that dates back to the Progressive era. In short, they aim to dehumanize black people based on assumptions of how intelligence determines the capacity to be fully human. Their use of these data and interpretation of existing research is wrong, and it remains dangerous. Consequently, these concerns are well-founded and the debate and critique are, and will continue to be, essential as the field continues to develop.

Discomfort with this research, however, may also reflect an uneasiness with the role of genetics in educational ability. Nearly 14 percent of children age 3 to 18 have a diagnosed disability, many of which have a clear genetic basis, that influences their ability to learn in current educational institutions (Zablotsky et al. 2019). Even among the most advantaged, genetic differences play a role in differentiating who achieves higher levels of educational attainment. Indeed, it may matter even more for this group (Turkheimer et al. 2003). Just like we do not all have equal probabilities of becoming a pianist at birth, we do not have equal probabilities of obtaining a PhD at birth either. When we gush over the most prominent scholars among us, while we acknowledge the role that race, gender, and class play in shaping those careers, we also acknowledge the "raw" talent.

McMillan Cottom (2019:27) observes that "[s]mart is only a construct of correspondence, between one's ability, one's environment, and one's moment in history." Being "smart" has never been more valued than in this historical moment, both socially and monetarily. We also have a long and robust history of dehumanizing and discrimination against those with cognitive disabilities, against those defined as not smart enough. We generally do not have the same uneasiness regarding the role of genetics in the development of abilities like music or art—even though some complicated interplay of genes and broader environments is present here, just as it is for academic ability. We rarely insult people based on their lack of artistic ability, whereas we commonly insult people based on their lack, or perceived lack, of intelligence. Acknowledging the role of genetics in this historical moment, especially when some (falsely) claim it is immutable, justifiability causes unease. We caution, however, that this discomfort may also reflect unexamined assumptions about the role of "smart" in defining who is—and is not—fully human and equal.

Yet, a sociological perspective in this research is essential. Decades of theory and empirical work allows sociologists to think critically about how to conceptualize and measure environment when examining the interplay between genes and environment for an outcome like educational attainment. Social scientists, including sociologists, bring methodological expertise as well, with the development of causal inference strategies, such as exogenous school policy changes, that allow for analyses that would not be confounded by, for example, gene by environment correlations (Conley and Fletcher 2017).

Nonetheless, although sociology has played an enormously valuable role in the critical analyses of genetic research, with important exceptions (e.g., Boardman et al. 2008; Conley et al. 2013; Mills, Barban, and Tropf 2018; Wedow et al. 2018), sociology has been mostly absent from genetics research itself, ceding it to the psychological sciences.

One example of how this has muddied the water is the tendency in the psychological literature to view IQ and educational attainment as interchangeable (Plomin 2018). In this worldview, education is a psychological trait, like schizophrenia, and environments play a limited role in explaining variation in these traits. Sociologists, of course, know well the difference between a psychological trait and a life course attainment, as the latter occurs only as the product of an extended chain of interactions between individual behavior and environmental response (Freese 2008). In fact, it was a group of sociologists who demonstrated, in a meta-analysis of twin studies, the vastly different (and much larger) role of environment in heritability studies of education versus IQ (Branigan et al. 2013).

Yet, the view, based on twin studies that severely limit analyses of gene-environment interplay, that genetics plays the dominant role in determining educational attainment has infiltrated policy debates in the United Kingdom. For example, a key advisor to the English Secretary of Education, after extensively citing research on twins by psychologists like Robert Plomin, claimed that schools or teacher quality had little influence on educational outcomes, specifically test scores (Merrick 2013). Although his read of the science was dubious at best, twin studies and heritability estimates make it far more difficult to have nuanced discussions of how environments modify genetic influences. In short, if the concern is that engaging in this research will validate the view that only genetics matter, and that policy interventions are a fruitless mechanism to improve and equalize educational outcomes, we now actually have an empirical tool to counteract this view.

The advent of polygenic scores provides the opportunity for sociologists to have a louder voice in these debates by broadening the body of empirical work focused on the role of environments in modifying genetic influences. It is far more difficult to contradict empirical evidence—and to have a credible voice in these debates—without engaging in the relevant research. In 1980, Jencks presciently argued that heritability—and the twin models that provide its empirical basis—limits our ability to understand the role of environment in patterning genetic influences on social outcomes like educational attainment. Today, however, we now have an empirical tool that will allow us to test and better understand gene-environment interplay. The growing body of work, which increasingly includes sociology, is just beginning to help us better understand the role of environments, from cohort to socioeconomic conditions (Domingue et al. 2016; Liu 2018; Trejo et al. 2018; Wedow et al. 2018). But to get there, we need the expertise of sociologists who have the theoretical and empirical knowledge of "environment" to properly do this work.

Our analysis provides an example of how sociologists can engage. We show that gender influences the expression of "genetic potential" into educational attainment, over the life course and across cohorts. The findings from this study, however, emphasize that you cannot understand the role of genetics in educational attainment if you do not examine the social environments in which genetic factors are operating. Genetics does not explain the history of gender inequalities in educational attainment. Gender explains the varied relationship between the polygenic score and educational attainment across the life course and cohorts. It is only by acknowledging the complicated interplay of genetics and environment that we can come closer to understanding precisely how environments and institutions shape stratification and inequality.

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Notes

1. Of course, it is also true that one can look at the same evidence and come to different conclusions about which perspective it supports. Debates

between Turkheimer and Plomin beautifully illustrate this point (Turkheimer 2016).

2. Response options and their numeric values (in parentheses) were 8th grade or less (8), some high school (10), high school graduate (12), some vocational/technical training (13), completed vocational/technical training (14), some college (14), completed college (16), some graduate school (17), completed a master's degree (18), some graduate training beyond a master's degree (19), completed a doctoral degree (20), some post-baccalaureate professional education (18), and completed post-baccalaureate professional education (19).
3. The WLS and Add Health were both part of the GWAS sample used in Lee and colleagues (2018), but we obtained customized weights to use in creating our scores that excluded each sample when constructing polygenic scores.
4. Code for the analyses presented in this study is publicly available at https://osf.io/4fvu2/. The data we use are available but cannot be posted by us. WLS and Add Health analyses use restricted data that are available to investigators but require prospective users to submit and receive approval for their secure use. Our HRS analyses are based on their public-use files, but users still must register with HRS to obtain and use these data.
5. Note that when testing for statistical differences across these groups, the samples are not independent. We are comparing differences over time among the same group of women.
6. We cannot rule out the possibility that mortality selection influenced these findings, given higher mortality rates for men and for individuals with lower educational attainment. Existing evidence, however, shows that these kinds of selection effects are not large, particularly within a cohort (Domingue et al. 2017). In our HRS analyses, where some researchers have worried that mortality risk may pose a larger problem, we used weights to address the risk (Domingue et al. 2017).
7. While we thought it useful to provide the stratified analysis for Add Health, we did not exclude respondents with less than high school degrees from the HRS analyses because our goal was to compare across an extended number of cohorts within the HRS sample. Given differential retention rates across cohorts, and weight construction, attempting to parse the analyses this way would likely have led to significant measurement error. Moreover, in the younger HRS cohorts, gender differences in high school graduation rates, which had not differed for the 1930s birth cohorts, started to emerge, with more women than men completing high school degrees (Heckman and LaFontaine 2010).

References

Al Kawam, Ahmad, Mustafa Alshawaqfeh, James J. Cai, Erchin Serpedin, and Aniruddha Datta. 2018. "Simulating Variance Heterogeneity in Quantitative Genome Wide Association Studies." *BMC Bioinformatics* 19(3):72.

American Society of Human Genetics (ASGH). 2018. "ASGH Denounces Attempts to Link Genetics and Racial Supremacy." *American Journal of Human Genetics* 103(5):636.

Asbury, Kathryn, Theodore D. Wachs, and Robert Plomin. 2005. "Environmental Moderators of Genetic Influence on Verbal and Nonverbal Abilities in Early Childhood." *Intelligence* 33(6):643–61.

Baker, Laura A., Susan A. Treloar, Chandra A. Reynolds, Andrew C. Heath, and Nicholas G. Martin. 1996. "Genetics of Educational Attainment in Australian Twins: Sex Differences and Secular Changes." *Behavior Genetics* 26(2):89–102.

Barnes, J. C., and Brian B. Boutwell. 2013. "A Demonstration of the Generalizability of Twin-Based Research on Antisocial Behavior." *Behavior Genetics* 43(2):120–31.

Bartels, Meike, Marjolein J. H. Rietveld, G. Caroline, M. Van Baal, and Dorret I. Boomsma. 2002. "Heritability of Educational Achievement in 12-Year-Olds and the Overlap with Cognitive Ability." *Twin Research and Human Genetics* 5(6):544–53.

Belsky, Daniel W., Benjamin W. Domingue, Robbee Wedow, Louise Arseneault, Jason D. Boardman, Avshalom Caspi, Dalton Conley, et al. 2018. "Genetic Analysis of Social-Class Mobility in Five Longitudinal Studies." *Proceedings of the National Academy of Sciences* 115(31):E7275–84.

Belsky, Daniel W., Terrie E. Moffitt, Renate Houts, Gary G. Bennett, Andrea K. Biddle, James A. Blumenthal, James P. Evans, et al. 2012. "Polygenic Risk, Rapid Childhood Growth, and the Development of Obesity: Evidence from a 4-Decade Longitudinal Study." *Archives of Pediatrics & Adolescent Medicine* 166(6):515–21.

Black, Sandra E., Paul J. Devereux, and Kjell G. Salvanes. 2008. "Staying in the Classroom and Out of the Maternity Ward? The Effect of Compulsory Schooling Laws on Teenage Births." *Economic Journal* 118(530):1025–54.

Bliss, Catherine. 2018. *Social by Nature: The Promise and Peril of Sociogenomics*. Stanford, CA: Stanford University Press.

Boardman, Jason D., Kari B. Alexander, and Michael C. Stallings. 2011. "Stressful Life Events and Depression among Adolescent Twin Pairs." *Biodemography and Social Biology* 57(1):53–66.

Boardman, Jason D., Casey L. Blalock, and Tanya M. M. Button. 2008. "Sex Differences in the Heritability of Resilience." *Twin Research and Human Genetics* 11(1):12–27.

Boardman, Jason D., Casey L. Blalock, and Fred C. Pampel. 2010 "Trends in the Genetic Influences on Smoking." *Journal of Health and Social Behavior* 51(1):108–23.

Boardman, Jason D., Jonathan Daw, and Jeremy Freese. 2013. "Defining the Environment in Gene–Environment

Research: Lessons from Social Epidemiology." *American Journal of Public Health* 103(S1):S64–S72.

Boardman, Jason D., and Jason M. Fletcher. 2015. "To Cause or Not to Cause? That Is the Question, but Identical Twins Might Not Have All of the Answers." *Social Science & Medicine* 127:198–200.

Borghans, Lex, Bart H. Golsteyn, James J. Heckman, and John Eric Humphries. 2016. "What Grades and Achievement Tests Measure." *Proceedings of the National Academy of Sciences* 113(47):13354–9.

Boyle, Evan A., Yang Li, and Jonathan Pritchard. 2017. "An Expanded View of Complex Traits: From Polygenic to Omnigenic." *Cell* 169(7):1177–86.

Branigan, Amelia R., Kenneth J. McCallum, and Jeremy Freese. 2013. "Variation in the Heritability of Educational Attainment: An International Meta-analysis." *Social Forces* 92(1):109–40.

Brown, David K. 2001. "The Social Sources of Educational Credentialism: Status Cultures, Labor Markets, and Organizations." *Sociology of Education* 74(extra issue):19–34.

Buchmann, Claudia, Thomas A. DiPrete, and Anne McDaniel. 2008. "Gender Inequalities in Education." *Annual Review of Sociology* 34:319–37.

Card, David, and Thomas Lemieux. 2001. "Can Falling Supply Explain the Rising Return to College for Younger Men? A Cohort-Based Analysis." *Quarterly Journal of Economics* 116(2):705–46.

Cesarini, David, and Peter M. Visscher. 2017. "Genetics and Educational Attainment." *NPJ Science of Learning* 2(1):4.

Chabris, Christopher F., James J. Lee, David Cesarini, Daniel J. Benjamin, and David I. Laibson. 2015. "The Fourth Law of Behavior Genetics." *Current Directions in Psychological Science* 24(4):304–12.

Conley, Dalton. 2016. "Socio-Genomic Research Using Genome-Wide Molecular Data." *Annual Review of Sociology* 42:275–99.

Conley, Dalton, and Neil G. Bennett. 2000. "Is Biology Destiny? Birth Weight and Life Chances." *American Sociological Review* 65(3):458–67.

Conley, Dalton, and Benjamin W. Domingue. 2016. "The Bell Curve Revisited: Testing Controversial Hypotheses with Molecular Genetic Data." *Sociological Science* 3:520–39.

Conley, Dalton, and Jason Fletcher. 2017. *The Genome Factor: What the Social Genomics Revolution Reveals about Ourselves, Our History, and the Future*. Princeton, NJ: Princeton University Press.

Conley, Dalton, Rebecca Johnson, Benjamin W. Domingue, Christopher Dawes, Jason D. Boardman, and Mark L. Siegal. 2018. "A Sibling Method for Identifying vQTLs." *PloS One* 13(4):e0194541.

Conley, Dalton, Emily Rauscher, Christopher Dawes, Patrik K. E. Magnusson, and Mark L. Siegal. 2013. "Heritability and the Equal Environments Assumption: Evidence from Multiple Samples of Misclassified Twins." *Behavior Genetics* 43(5):415–26.

Conley, Dalton, Mark L. Siegal, Benjamin W. Domingue, Kathleen Mullan Harris, Matthew B. McQueen, and Jason D. Boardman. 2014. "Testing the Key Assumption of Heritability Estimates Based on Genome-wide Genetic Relatedness." *Journal of Human Genetics* 59(6):342.

Crimmins, Eileen M., Jessica D. Faul, Jung Ki Kim, and David R. Weir. 2015. "Documentation of Biomarkers in the 2010 and 2012 Health and Retirement Study." Ann Arbor: Survey Research Center, University of Michigan.

Current Population Survey (CPS). 2017. "CPS Historical Time Series Tables on School Enrollment. Table A-6. Age Distribution of College Students 14 Years Old and Over, by Sex: October 1947 to 2016" (https://www.census.gov/data/tables/time-series/demo/school-enrollment/cps-historical-time-series.html).

Current Population Survey (CPS). 2019. "Historical Time Series Visualizations. Figure 6: Percent of Population 25 and Older, and 25 to 29 Years Old, with Bachelor's Degree or Higher by Sex: 1947–2018" (https://www.census.gov/content/dam/Census/library/visualizations/time-series/demo/fig6.jpg).

Dar-Nimrod, Ilan, and Steven J. Heine. 2011. "Genetic Essentialism: On the Deceptive Determinism of DNA." *Psychological Bulletin* 137(5):800–18.

Daw, Jonathan, Guang Guo, and Kathleen Mullan Harris. 2015. "Nurture Net of Nature: Re-evaluating the Role of Shared Environments in Academic Achievement and Verbal Intelligence." *Social Science Research* 52:422–39.

DiPrete, Thomas A., and Claudia Buchmann. 2013. *The Rise of Women: The Growing Gender Gap in Education and What It Means for American Schools*. New York: Russell Sage Foundation.

Domingue, Benjamin W., Daniel W. Belsky, Dalton Conley, Kathleen Mullan Harris, and Jason D. Boardman. 2015. "Polygenic Influence on Educational Attainment: New Evidence from the National Longitudinal Study of Adolescent to Adult Health." *AERA Open* (https://doi.org/10.1177/2332858415599972).

Domingue, Benjamin W., Daniel W. Belsky, Jason M. Fletcher, Dalton Conley, Jason D. Boardman, and Kathleen Mullan Harris. 2018. "The Social Genome of Friends and Schoolmates in the National Longitudinal Study of Adolescent to Adult Health." *Proceedings of the National Academy of Sciences* 115(4):702–07.

Domingue, Benjamin W., Daniel W. Belsky, Amal Harrati, Dalton Conley, David R. Weir, and Jason D. Boardman. 2017. "Mortality Selection in a Genetic Sample and Implications for Association Studies." *International Journal of Epidemiology* 46(4):1285–94.

Domingue, Benjamin W., Daniel W. Belsky, Kathleen Mullan Harris, Andrew Smolen, Matthew B. McQueen, and Jason D. Boardman. 2014. "Polygenic Risk Predicts Obesity in Both White and Black Young Adults." *PloS One* 9(7):e101596.

Domingue, Benjamin W., Dalton Conley, Jason Fletcher, and Jason D. Boardman. 2016. "Cohort Effects in the Genetic Influence on Smoking." *Behavior Genetics* 46(1):31–42.

Dudbridge, Frank. 2016. "Polygenic Epidemiology." *Genetic Epidemiology* 40(4):268–72.

Duster, Troy. 2004. *Backdoor to Eugenics*. New York: Routledge.

Felson, Jacob. 2014. "What Can We Learn from Twin Studies? A Comprehensive Evaluation of the Equal Environments Assumption." *Social Science Research* 43:184–99.

Figlio, David N., Jeremy Freese, Krzysztof Karbownik, and Jeffrey Roth. 2017. "Socioeconomic Status and Genetic Influences on Cognitive Development." *Proceedings of the National Academy of Sciences* 114(51):13441–46.

Fortin, Nicole M., Philip Oreopoulos, and Shelley Phipps. 2015. "Leaving Boys Behind: Gender Disparities in High Academic Achievement." *Journal of Human Resources* 50(3):549–79.

Fosse, Roar, Jay Joseph, and Mike Jones. 2016. "Schizophrenia: A Critical View on Genetic Effects." *Psychosis* 8(1):72–84.

Freese, Jeremy. 2008. "Genetics and the Social Science Explanation of Individual Outcomes." *American Journal of Sociology* 114:S1–S35.

Friend, Angela, John C. DeFries, Richard K. Olson, Bruce Pennington, Nicole Harlaar, Brian Byrne, and Stefan Samuelsson. 2009. "Heritability of High Reading Ability and Its Interaction with Parental Education." *Behavior Genetics* 39(4):427–36.

Gamoran, Adam. 2001. "American Schooling and Educational Inequality: A Forecast for the 21st Century." *Sociology of Education* 74(extra issue):135–53.

Glymour, M. Maria, Ichiro Kawachi, Christopher S. Jencks, and Lisa F. Berkman. 2008. "Does Childhood Schooling Affect Old Age Memory or Mental Status? Using State Schooling Laws as Natural Experiments." *Journal of Epidemiology & Community Health* 62(6):532–7.

Goldin, Claudia. 2006. "The Quiet Revolution That Transformed Women's Employment, Education, and Family." *American Economic Review* 96(2):1–21.

Goldin, Claudia, Lawrence F. Katz, and Ilyana Kuziemko. 2006. "The Homecoming of American College Women: The Reversal of the College Gender Gap." *Journal of Economic Perspectives* 20(4):133–56.

Haas, Steven A., and Nathan Edward Fosse. 2008. "Health and the Educational Attainment of Adolescents: Evidence from the NLSY97." *Journal of Health and Social Behavior* 49(2):178–92.

Haberstick, Brett C., Andrew Smolen, Redford B. Williams, George D. Bishop, Vangie A. Foshee, Terence P. Thornberry, Rand Conger, et al. 2015. "Population Frequencies of the Triallelic 5HTTLPR in Six Ethnically Diverse Samples from North America, Southeast Asia, and Africa." *Behavior Genetics* 45(2):255–61.

Harden, K. Paige, Benjamin W. Domingue, Daniel W. Belsky, Jason D. Boardman, Robert Crosnoe, Margherita Malanchini, Michel Nivard, Elliot M. Tucker-Drob, and Kathleen Mullan Harris. 2019. "Genetic Associations with Mathematics Tracking and Persistence in Secondary School." *bioRxiv* (https://www.biorxiv.org/content/10.1101/598532v1).

Hart, Sara A., Brooke Soden, Wendy Johnson, Christopher Schatschneider, and Jeanette Taylor. 2013. "Expanding the Environment: Gene × School-Level SES Interaction on Reading Comprehension." *Journal of Child Psychology and Psychiatry* 54(10):1047–55.

Hauser, Robert M., William H. Sewell, John A. Logan, Taissa S. Hauser, Carol Ryff, Avshalom Caspi, and Maurice M. MacDonald. 1992. *The Wisconsin Longitudinal Study: Adults as Parents and Children at Age 50*. University of Wisconsin-Madison.

Hauser, Robert M., and Raymond Sin-Kwok Wong. 1989. "Sibling Resemblance and Intersibling Effects in Educational Attainment." *Sociology of Education* 62(3):149–71.

Heath, Andrew C., Kare Berg, Lindon J. Eaves, Marit H. Solaas, Linda A. Corey, Jon Sundet, Per Magnus, and Walter E. Nance. 1985. "Education Policy and the Heritability of Educational Attainment." *Nature* 314(6013):734.

Heckman, James J., and Paul A. LaFontaine. 2010. "The American High School Graduation Rate: Trends and Levels." *The Review of Economics and Statistics* 92(2):244–62.

Herd, Pamela, Deborah Carr, and Carol Roan. 2014. "Cohort Profile: Wisconsin Longitudinal Study (WLS)." *International Journal of Epidemiology* 43(1):34–41.

Jencks, Christopher. 1980. "Heredity, Environment, and Public Policy Reconsidered." *American Sociological Review* 45(5):723–36.

Kendler, Kenneth S. 2006. "'A Gene for . . .': The Nature of Gene Action in Psychiatric Disorders." *American Journal of Psychiatry* 162(7):1243–52.

Kendler, Kenneth S., Nicholas G. Martin, Andrew C. Heath, and Lindon J. Eaves. 1995. "Self-Report Psychiatric Symptoms in Twins and Their Nontwin Relatives: Are Twins Different?" *American Journal of Medical Genetics* 60(6):588–91.

Koboldt, Daniel C., Karyn Meltz Steinberg, David E. Larson, Richard K. Wilson, and Elaine R. Mardis. 2013. "The Next-Generation Sequencing Revolution and Its Impact on Genomics." *Cell* 155(1):27–38.

Kong, Augustine, Gudmar Thorleifsson, Michael L. Frigge, Bjarni J. Vilhjalmsson, Alexander I. Young, Thorgeir E. Thorgeirsson, Stefania Benonisdottir, et al. 2018. "The Nature of Nurture: Effects of Parental Genotypes." *Science* 359(6374):424–8.

Krapohl, Eva, Hamel Patel, Stephen Newhouse, Charles J. Curtis, Sophie von Stumm, Philip S. Dale, Delilah Zabaneh, Gerome Breen, Paul F. O'Reilly, and Robert Plomin. 2017. "Multi-polygenic Score Approach to Trait Prediction." *Molecular Psychiatry* 23:1368–74.

Krapohl, Eva, and Robert Plomin. 2016. "Genetic Link between Family Socioeconomic Status and Children's Educational Achievement Estimated from Genome-wide SNPs." *Molecular Psychiatry* 21(3):437–43.

Krapohl, Eva, Kaili Rimfeld, Nicholas G. Shakeshaft, Maciej Trzaskowski, Andrew McMillan, Jean-Baptiste Pingault, Kathryn Asbury, et al. 2014. "The High Heritability of Educational Achievement Reflects Many Genetically Influenced Traits, Not Just

Intelligence." *Proceedings of the National Academy of Sciences* 111(42):15273–8.

Lee, Catherine. 2009. "'Race' and 'Ethnicity' in Biomedical Research: How Do Scientists Construct and Explain Differences in Health?" *Social Science & Medicine* 68(6):1183–90.

Lee, James J., Robbee Wedow, Aysu Okbay, Edward Kong, Omeed Maghzian, Meghan Zacher, Tuan Anh Nguyen-Viet, et al. 2018. "Gene Discovery and Polygenic Prediction from a Genome-wide Association Study of Educational Attainment in 1.1 Million Individuals." *Nature Genetics* 50(8):1112–21.

Legewie, Joscha, and Thomas A. DiPrete. 2012. "School Context and the Gender Gap in Educational Achievement." *American Sociological Review* 77(3):463–85.

Levin, Jack, and William C. Levin. 1991. "Sociology of Educational Late Blooming." *Sociological Forum* 6(4):661–79.

Link, Bruce G., and Jo C. Phelan. 1995. "Social Conditions as Fundamental Causes of Disease." *Journal of Health and Social Behavior* (extra issue):80–94.

Liu, Hexuan. 2018. "Social and Genetic Pathways in Multigenerational Transmission of Educational Attainment." *American Sociological Review* 83(2):278–304.

Liu, Hexuan, and Guang Guo. 2015. "Lifetime Socioeconomic Status, Historical Context, and Genetic Inheritance in Shaping Body Mass in Middle and Late Adulthood." *American Sociological Review* 80(4):705–37.

Martin, Alicia R., Christopher R. Gignoux, Raymond K. Walters, Genevieve L. Wojcik, Benjamin M. Neale, Simon Gravel, Mark J. Daly, Carlos D. Bustamante, and Eimear E. Kenny. 2017. "Human Demographic History Impacts Genetic Risk Prediction across Diverse Populations." *American Journal of Human Genetics* 100(4):635–49.

McClellan, Jon M., Thomas Lehner, and Mary-Claire King. 2017. "Gene Discovery for Complex Traits: Lessons from Africa." *Cell* 171(2):261–4.

McMillan Cottom, Tressie. 2018. *Thick: And Other Essays*. New York: The New Press.

McQueen, Matthew B., Jason D. Boardman, Benjamin W. Domingue, Andrew Smolen, Joyce Tabor, Ley Killeya-Jones, Carolyn T. Halpern, Eric A. Whitsel, and Kathleen Mullan Harris. 2015. "The National Longitudinal Study of Adolescent to Adult Health (Add Health) Sibling Pairs Genome-wide Data." *Behavior Genetics* 45(1):12–23.

Merrick, Jane. 2013. "Michael Gove Held Talks with 'IQ Genes' Professor." *The Independent*, October 13 (https://www.independent.co.uk/news/education/education-news/michael-gove-held-talks-with-iq-genes-professor-8876500.html).

Mills, Melinda C., Nicola Barban, and Felix C. Tropf. 2018. "The Sociogenomics of Polygenic Scores of Reproductive Behavior and Their Relationship to Other Fertility Traits." *RSF: The Russell Sage Foundation Journal of the Social Sciences* 4(4):122–36.

Mills, Melinda C., and Charles Rahal. 2019. "A Scientometric Review of Genome-wide Association Studies." *Communications Biology* 2(1):9.

Næss, Kari-Anne B., Monica Melby-Lervåg, Charles Hulme, and Solveig-Alma Halaas Lyster. 2012. "Reading Skills in Children with Down Syndrome: A Meta-analytic Review." *Research in Developmental Disabilities* 33(2):737–47.

Okbay, Aysu, Jonathan P. Beauchamp, Mark Alan Fontana, James J. Lee, Tune H. Pers, Cornelius A. Rietveld, Patrick Turley, et al. 2016. "Genome-wide Association Study Identifies 74 Loci Associated with Educational Attainment." *Nature* 533(7604):539–42.

Oreopoulos, Philip. 2006. "Estimating Average and Local Average Treatment Effects of Education When Compulsory Schooling Laws Really Matter." *American Economic Review* 96(1):152–75.

Petersen, Inge, Torben Martinussen, Matthew McGue, Paul Bingley, and Kaare Christensen. 2011. "Lower Marriage and Divorce Rates among Twins Than among Singletons in Danish Birth Cohorts 1940–1964." *Twin Research and Human Genetics* 14(2):150–7.

Petrill, Stephen A., and Lee Anne Thompson. 1994. "The Effect of Gender upon Heritability and Common Environmental Estimates in Measures of Scholastic Achievement." *Personality and Individual Differences* 16(4):631–40.

Plomin, Robert. 2018. *Blueprint: How DNA Makes Us Who We Are*. Cambridge, MA: MIT Press.

Plug, Erik, and Wim Vijverberg. 2003. "Schooling, Family Background, and Adoption: Is It Nature or Is It Nurture?" *Journal of Political Economy* 111(3):611–41.

Pulkkinen, Lea, Inka Vaalamo, Risto Hietala, Jaakko Kaprio, and Richard J. Rose. 2003. "Peer Reports of Adaptive Behavior in Twins and Singletons: Is Twinship a Risk or an Advantage?" *Twin Research and Human Genetics* 6(2):106–18.

Rice, Mabel L., Stephen R. Zubrick, Catherine L. Taylor, Lesa Hoffman, and Javier Gayán. 2018. "Longitudinal Study of Language and Speech of Twins at 4 and 6 Years: Twinning Effects Decrease, Zygosity Effects Disappear, and Heritability Increases." *Journal of Speech, Language, and Hearing Research* 61(1):79–93.

Rietveld, Cornelius A., Tõnu Esko, Gail Davies, Tune H. Pers, Patrick Turley, Beben Benyamin, Christopher F. Chabris, et al. 2014. "Common Genetic Variants Associated with Cognitive Performance Identified Using the Proxy-Phenotype Method." *Proceedings of the National Academy of Sciences* 111(38):13790–4.

Rimfeld, Kaili, Margherita Malanchini, Eva Krapohl, Laurie J. Hannigan, Philip S. Dale, and Robert Plomin. 2018. "The Stability of Educational Achievement across School Years Is Largely Explained by Genetic Factors." *NPJ Science of Learning* 3(1):16.

Risman, Barbara J. 2004. "Gender as a Social Structure: Theory Wrestling with Activism." *Gender & Society* 18(4):429–50.

Roberts, Dorothy. 2011. *Fatal Invention: How Science, Politics, and Big Business Re-create Race in the Twenty-first Century*. New York: New Press/ORIM.

Rowe, David C., Kristen C. Jacobson, and Edwin J. C. G. Van den Oord. 1999. "Genetic and Environmental Influences on Vocabulary IQ: Parental Education Level as Moderator." *Child Development* 70(5):1151–62.

Rylander-Rudqvist, Tove, Niclas Håkansson, Gunnel Tybring, and Alicja Wolk. 2006. "Quality and Quantity of Saliva DNA Obtained from the Self-Administrated Oragene Method—A Pilot Study on the Cohort of Swedish Men." *Cancer Epidemiology and Prevention Biomarkers* 15(9):1742–5.

Scarr, Sandra, and Kathleen McCartney. 1983. "How People Make Their Own Environments: A Theory of Genotype→ Environment Effects." *Child Development* 54(2):424–35.

Scarr-Salapatek, Sandra. 1971. "Race, Social Class, and IQ." *Science* 174(4016):1285–129.

Schmitz, Lauren L., and Dalton Conley. 2017. "The Effect of Vietnam-Era Conscription and Genetic Potential for Educational Attainment on Schooling Outcomes." *Economics of Education Review* 61:85–97.

Selzam, Saskia, Eva Krapohl, Sophie von Stumm, P. F. O'Reilly, K. Rimfeld, Y. Kovas, P. S. Dale, J. J. Lee, and R. Plomin. 2017. "Predicting Educational Achievement from DNA." *Molecular Psychiatry* 22(2):267–72.

Shakeshaft, Nicholas G., Maciej Trzaskowski, Andrew McMillan, Kaili Rimfeld, Eva Krapohl, Claire M. A. Haworth, Philip S. Dale, and Robert Plomin. 2013. "Strong Genetic Influence on a UK Nationwide Test of Educational Achievement at the End of Compulsory Education at Age 16." *PloS one* 8(12):e80341.

Shinwell, Eric, T. Haklai, and Smadar Eventov-Friedman. 2009. "Outcomes of Multiplets." *Neonatology* 95(1):6–14.

Short, Susan E., Yang Claire Yang, and Tania M. Jenkins. 2013. "Sex, Gender, Genetics, and Health." *American Journal of Public Health* 103(S1):S93–S101.

Silventoinen, Karri, Robert F. Krueger, Thomas J. Bouchard, Jaakko Kaprio, and Matt McGue. 2004. "Heritability of Body Height and Educational Attainment in an International Context: Comparison of Adult Twins in Minnesota and Finland." *American Journal of Human Biology: The Official Journal of the Human Biology Association* 16(5):544–55.

Sonnega, Amanda, Jessica D. Faul, Mary Beth Ofstedal, Kenneth M. Langa, John W. R. Phillips, and David R. Weir. 2014. "Cohort Profile: The Health and Retirement Study (HRS)." *International Journal of Epidemiology* 43(2):576–85.

Taylor, Jeanette, Alysia D. Roehrig, Soden Hensler, C. M. Connor, and Christopher Schatschneider. 2010. "Teacher Quality Moderates the Genetic Effects on Early Reading." *Science* 328(5977):512–4.

Trejo, Sam, Daniel W. Belsky, Jason D. Boardman, Jeremy Freese, Kathleen Mullan Harris, Pamela Herd, Kamil Sicinski, and Benjamin W. Domingue. 2018. "Schools as Moderators of Genetic Associations with Life Course Attainments: Evidence from the WLS and Add Health." *Sociological Science* 2(5):513–40.

Tsou, Meng-Ting, Meng-Wen Tsou, Ming-Ping Wu, and Jin-Tan Liu. 2008. "Academic Achievement of Twins and Singletons in Early Adulthood: Taiwanese Cohort Study." *BMJ* 337:a438.

Tucker-Drob, Elliot M., and Timothy C. Bates. 2016. "Large Cross-National Differences in Gene × Socioeconomic Status Interaction on Intelligence." *Psychological Science* 27(2):138–49.

Turkheimer, Eric. 1998. "Heritability and Biological Explanation." *Psychological Review* 105(4):782–91.

Turkheimer, Eric. 2016. "Weak Genetic Explanation 20 Years Later: Reply to Plomin et al. (2016)." *Perspectives on Psychological Science* 11(1):24–28.

Turkheimer, Eric, Andreana Haley, Mary Waldron, Brian d'Onofrio, and Irving I. Gottesman. 2003. "Socioeconomic Status Modifies Heritability of IQ in Young Children." *Psychological Science* 14(6):623–8.

U.S. Bureau of the Census. 2016. "Table A-6, Age Distribution of College Students by Sex: October 1947–2015; Source: U.S. Census Bureau, Current Population Survey, 1947 to 2015" (https://www.census.gov/data/tables/time-series/demo/school-enrollment/cps-historical-time-series.html).

Van den Oord, Edwin J. C. G., and David C. Rowe. 1997. "An Examination of Genotype-Environment Interactions for Academic Achievement in an U.S. National Longitudinal Survey." *Intelligence* 25(3):205–28.

Visscher, Peter M., Naomi R. Wray, Qian Zhang, Pamela Sklar, Mark I. McCarthy, Matthew A. Brown, and Jian Yang. 2017. "10 Years of GWAS Discovery: Biology, Function, and Translation." *American Journal of Human Genetics* 101(1):5–22.

Voracek, Martin, and Tanja Haubner. 2008. "Twin–Singleton Differences in Intelligence: A Meta-Analysis." *Psychological Reports* 102(3):951–62.

Walter, Stefan, Iván Mejía-Guevara, Karol Estrada, Sze Y. Liu, and M. Maria Glymour. 2016. "Association of a Genetic Risk Score with Body Mass Index across Different Birth Cohorts." *JAMA* 316(1):63–9.

Ware, Erin, Lauren Schmitz, Arianna Gard, and Jessica Faul. 2018. "HRS Polygenic Scores—Release 3: 2006–2012 Genetic Data." Ann Arbor: Survey Research Center, University of Michigan.

Warren, John Robert, and Robert M. Hauser. 1997. "Social Stratification across Three Generations: New Evidence from the Wisconsin Longitudinal Study." *American Sociological Review* 62(4):561–72.

Warren, John Robert, Jennifer T. Sheridan, and Robert M. Hauser. 2002. "Occupational Stratification across the Life Course: Evidence from the Wisconsin Longitudinal Study." *American Sociological Review* 67(3):432–55.

Wedow, Robbee, Meghan Zacher, Brooke M. Huibregtse, Kathleen Mullan Harris, Benjamin W. Domingue, and Jason D. Boardman. 2018. "Education, Smoking, and Cohort Change: Forwarding a Multidimensional

Theory of the Environmental Moderation of Genetic Effects." *American Sociological Review* 83(4):802–32.

Wood, Andrew R., Tonu Esko, Jian Yang, Sailaja Vedantam, Tune H. Pers, Stefan Gustafsson, Audrey Y. Chu, et al. 2014. "Defining the Role of Common Variation in the Genomic and Biological Architecture of Adult Human Height." *Nature Genetics* 46(11):1173.

Zablotsky, Benjamin, Lindsey I. Black, Matthew J. Maenner, Laura A. Schieve, Melissa L. Danielson, Rebecca H. Bitsko, Stephen J. Blumberg, Michael D. Kogan, and Coleen A. Boyle. 2019. "Prevalence and Trends of Developmental Disabilities among Children in the United States: 2009–2017." *Pediatrics* 144(4):e20190811.

Zax, Jeffrey S., and Daniel I. Reese. 2002. "IQ, Academic Performance, Environment, and Earnings." *Review of Economics and Statistics* 84(4):600–616.

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