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The propensity for aggressive behavior and lifetime incarceration risk: A test for gene-environment interaction (G × E) using whole-genome data

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ABSTRACT

Incarceration is a disruptive event that is experienced by a considerable proportion of the United States population. Research has identified social factors that predict incarceration risk, but scholars have called for a focus on the ways that individual differences combine with social factors to affect incarceration risk. Our study is an initial attempt to heed this call using whole-genome data. We use data from the Health and Retirement Study (HRS) (N = 6716) to construct a genome-wide measure of genetic propensity for aggressive behavior and use it to predict lifetime incarceration risk. We find that participants with a higher genetic propensity for aggression are more likely to experience incarceration, but the effect is stronger for males than females. Importantly, we identify a gene-environment interaction (G × E)—genetic propensity is reduced, substantively and statistically, to a non-significant predictor for males raised in homes where at least one parent graduated high school. We close by placing these findings in the broader context of concerns that have been raised about genetics research in criminology.

1. Introduction

The United States is addicted to incarceration (Pratt, 2009). Approximately 2.2 million people are incarcerated by the U.S. criminal justice system on any given day, which translates to nearly 1 of every 110 US adults being behind bars at any given time (Carson, 2014; Cullen & Jonson, 2016). When looked at from the individual level, an estimated 6 to 10% of all U.S. citizens will, at some point in their life, spend time in a state or federal prison (Bonczar, 2003; Muller & Wildeman, 2016). These prevalence rates are alarming because incarceration has consistently been linked to a host of deleterious outcomes such as school failure, job instability, family and relationship difficulties, later involvement in crime, and even premature death (e.g., Bernburg & Krohn, 2003; Laub & Vaillant, 2000; Massoglia & Pridemore, 2015; Turanovic, Rodriguez, & Pratt, 2012; Yi, Turney, & Wildeman, 2016). These associations have been found to remain even after accounting for an extensive range of confounding influences, suggesting there is a causal impact of incarceration on negative life outcomes (Travis, Western, & Redburn, 2014).

Given the far-reaching impact of incarceration on offenders and their families, criminologists have investigated the risk and protective factors leading to behaviors that change one's likelihood of

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experiencing incarceration (e.g., Agnew, 2006; Farrington, Gaffney, & Ttofi, 2016; Moffitt, 2003; Raine, 2013; Sampson & Laub, 1993). Research has shown, for example, that low levels of attachment to parents and school, living in disadvantaged areas, and having delinquent peers are risk factors for the types of behavior that sometimes result in incarceration (e.g., Akers, 1988; Hirschi, 1969; Wilson, 1987). Other work has revealed how the social context can protect against criminogenic influences, thereby lowering incarceration risk. Family socioeconomic status (SES) in particular is known for its association with most—if not all—of the social factors that affect incarceration risk (Barkan & Rocque, 2018). This suggests childhood SES works as a fundamental cause (Link & Phelan, 1995) of incarceration because it affects whether one is "at risk of risks" for later involvement with the criminal justice system (see, generally, Anderson, 1999; Sampson & Wilson, 1995; Wikström & Treiber, 2016). The childhood environment, including childhood SES, is closely tied to and dependent upon parental educational attainment. Thus, we might expect low parental educational attainment to expose one to certain risks for incarceration and high parental educational attainment to compensate for risks (see, generally, Barkan & Rocque, 2018).

At the same time, criminologists have long recognized family SES, by itself, is an insufficient explanation of the observed variation in criminal justice outcomes; individual differences such as aggressive behavior must also be taken into account (Huesmann, Eron, & Dubow, 2002; Tremblay, Hartup, & Archer, 2005). Indeed, individuals who display high levels of aggression in childhood relative to their peers are more likely to commit violent acts as adults, thereby raising their risk of experiencing incarceration in adulthood (Agnew, 2001; Broidy et al., 2003; Farrington, 1991; Huesmann et al., 2002; Kalvin & Bierman, 2017; Lai, Zeng, & Chu, 2015; Loeber, Burke, & Pardini, 2009; Reingle, Jennings, & Maldonado-Molina, 2012; Schaeffer, Petras, Ialongo, Poduska, & Kellam, 2003). Thus, scholars have explored the etiology of aggression to better understand incarceration risk over the life course. Such research finds that variation in aggression emerges early in the life course, often within the first year (Hay et al., 2014). This suggests genetic propensities play a role in the etiology and maintenance of aggressive behavior (see Tremblay, 2003), and behavioral genetic research supports this argument (Asherson & Cormand, 2016; Burt, 2009; Polderman et al., 2015).

But genetic factors are not deterministic for aggression or for its downstream consequences like incarceration risk. On the contrary, genetic influences depend on the social environment, meaning gene-environment interaction (G × E) is the rule and not the exception (Tremblay, 2015). Research into the risk factors for incarceration should incorporate a G × E perspective—a perspective that recognizes the importance of both social and genetic risk factors. We will do so by analyzing whether parental educational attainment moderates the genetic propensity for aggression in predicting lifetime incarceration risk. Doing so affords us the ability to, as Conley and Fletcher (2017, p. 3) noted, "…go from the adage that a gene for aggression lands you in jail if you are from the ghetto but in the boardroom if you are to the manor born, to a scientific research agenda showing how environmental and genetic effects mutually depend upon each other."

The present study will advance the G × E perspective, but we will depart from previous efforts in several important respects (e.g., Wells et al., 2017). First, we integrate empirical and theoretical developments in criminology with the latest advancements in genomic technology. Modern genomic technology enables researchers to analyze markers across the *entire* human genome as opposed to selectively analyzing one or two candidate genes (Freese, 2018). Our study is among the first to capitalize on genome-wide data to study an outcome of interest to criminologists (see also Wertz et al., 2018). We will do so by constructing a genome-wide score that taps into the genetic propensity for aggressive behavior. The genome-wide score for aggressive behavior will then be used to predict incarceration risk. But, as noted above, we anticipate the impact of the genome-wide score will be moderated by socio-environmental inputs. Thus, we will test for a G × E between the genetic propensity for aggression and parental educational attainment in the prediction of incarceration risk.

Our second major departure from prior G × E research is that we will analyze lifetime incarceration risk by drawing information from a prospective longitudinal sample of Americans who have passed midlife (i.e., the Health and Retirement Study [HRS]). Doing so allows us to avoid problems of "right-censoring" that may have impacted estimates from much of the available criminological literature that relied on samples of adolescents and young adults to study the risk factors for incarceration (Cullen, 2011).

To setup the framework for our analysis, we now turn to a review of research that links aggressive behavior with incarceration risk. In doing so, we highlight the complications that have been faced by prior work and how our approach—using a genome-wide score of the propensity for aggressive behavior—helps to address those complications.

1.1. The impact of aggressive behavior on incarceration risk

Aggressive behavior, which can broadly be defined as direct or indirect acts of physical and social harm (Hartup, 2005), has consistently been identified as a risk factor for coming into contact with the criminal justice system generally and with incarceration risk specifically. Research has shown that highly aggressive individuals are more likely to engage in behaviors that result in incarceration (Agnew, 2001; Brame, Nagin, & Tremblay, 2001; Broidy et al., 2003; Coie & Dodge, 1998; Loeber et al., 2009; Loeber & Stouthamer-Loeber, 1998; Nagin & Tremblay, 1999; Schaeffer et al., 2003). For instance, after revealing a robust link between aggressive behavior and a range of criminal justice outcomes, Huesmann et al. (2002, p. 200) concluded that, "… how aggressively children behave by age eight is the best predictor of how likely they are over the next 22 years to be arrested, how many times they will be arrested, how serious will be their crimes, how many times they will be convicted and how long they will serve in prison."

Understanding the relationship between aggressive behavior and incarceration, however, presents both theoretical and methodological challenges. First, there is a substantial level of stratification across sex within the criminal justice system, including incarceration (Barnes, Jorgensen, Beaver, Boutwell, & Wright, 2015; Brame, Bushway, Paternoster, & Turner, 2014; Wilson & Herrnstein, 1985). Aggression research indicates that males and females display aggressive behavior using different techniques. Males tend to show aggression physically, while females are more likely to aggress using social means (Archer, 2004).

In addition, the relationship between aggressive behavior and incarceration is bi-directional. Research has shown that aggression heightens incarceration risk (e.g., Huesmann et al., 2002), but there is also evidence that incarceration increases aggressive behavior later in life (e.g., Listwan, Sullivan, Agnew, Cullen, & Colvin, 2013; Nagin, Cullen, & Jonson, 2009). Relatedly, aggressive behavior is time dependent (Anderson, 1999). Using measures of aggressive behavior taken at different time points may result in variation in estimates of the impact of aggressive behavior on incarceration risk.

Finally, another concern that is nearly always present in observational research is the aggressive behavior-incarceration relationship may be confounded by omitted variables. Without sufficiently considering possible confounders, estimation of the impact of aggressive behavior on incarceration risk could be biased.

We propose a novel approach to study the aggressive behavior-incarceration relationship: measure the genetic propensity for aggressive behavior and use it to predict incarceration risk. Doing so allows us to minimize the concerns discussed above as genetic propensity is established at conception and does not change over time. Thus, the direction of its association with incarceration risk cannot be reversed and there are very few antecedent causal factors that could confound the relationship.

Given these advantages, genetic propensity for aggressive behavior is a worthwhile measure to use when trying to understand the link between aggressive behavior and incarceration risk. But, before we present our analysis, it is important to establish that a propensity for aggressive behavior can be measured at the genetic level. In the next section, we consider the role of genetic influences on human behavior in general and aggressive behavior specifically. We then discuss how analytic approaches that are now common in genomics research can be used to test our hypotheses.

1.2. The genetic architecture of human behavior

The human genome is about 3 billion base pairs long (a base pair is a single A-T or C-G pairing) and has millions of locations (known as loci) that vary from person-to-person (Snustad & Simmons, 2016). Geneticists have recently developed analytic tools that allow researchers to observe the locations where individuals vary (typically these locations are called single nucleotide polymorphisms, or SNPs) and analyze whether variants at those loci are predictive of a particular outcome. This approach, which is known as a genome-wide association study (GWAS),[2] is one of the most rapidly developing areas in all of science (Visscher et al., 2017). While most of the work in GWAS has focused on medical and mental health conditions such as Alzheimer's disease (Lambert et al., 2013), BMI (Locke et al., 2015), height (Wood et al., 2014), and schizophrenia (Ripke et al., 2014), there is a growing body of work that applies GWAS to outcomes of interest to behavioral scientists. For instance, there are now GWAS examining the genomic predictors of educational attainment (Lee et al., 2018; Okbay, Beauchamp, et al., 2016; Rietveld et al., 2013), subjective well-being (Okbay, Baselmans, et al., 2016), and risk tolerance (Linnér et al., 2018).[3]

GWAS are especially noteworthy because their results can be used to generate a polygenic score (PGS) for the outcome of focus. PGSs are compound measures that aggregate the estimates produced by GWAS into a single score. That score can be thought of as a weighted average of an individual's genetic propensity for the specified outcome. In this way, it is appropriate to think of the PGS as a latent genetic propensity for the outcome focus.

The first PGS analysis was conducted in a study of schizophrenia (Purcell et al., 2009) and since then, PGSs have proven to be robust predictors of many human outcomes (e.g., Belsky et al., 2016; Belsky et al., 2018; Conley, Laidley, Belsky, et al., 2016; Conley & Domingue, 2016; Conley, Laidley, Boardman, & Domingue, 2016; Liu, 2018; Liu & Guo, 2015; Wedow et al., 2018). For example, a recent study found that a PGS for educational attainment could explain >10% of the variance in participants' observed education levels (Lee et al., 2018). This finding is quite remarkable when we consider that parental education—previously, the best-known predictor of one's own education levels—typically accounts for about the same amount of variance. Of interest to criminologists is a recent study by Wertz et al. (2018). Their analysis revealed that a PGS for educational attainment predicted criminal behavior and offending trajectory. Thus, the prospect that genomic technology in general, and PGSs in particular, can be integrated into criminological explanations of offending has already been realized.

What has not yet been assessed is whether a genome-wide PGS for aggressive behavior can be used to predict incarceration risk across the life course. Thus, the first hypothesis to be tested in the present study is:

Hypothesis 1. A genome-wide polygenic score (PGS) for aggressive behavior will be positively associated with lifetime incarceration risk.

In order to create a genome-wide PGS for aggressive behavior, we draw on the results from a recent GWAS conducted by Pappa et al. (2016). Pappa et al. (2016) performed GWAS on the outcome of childhood aggressive behavior by analyzing data from 9 cohorts, for a combined sample size of approximately 19,000 individuals. We rely on the estimates provided by Pappa et al. (2016) and combine it with the genome-wide information in our data to create a PGS for aggressive behavior. In other words, we construct a genome-wide measure of latent genetic propensity for aggressive behavior (see also Elam, Chassin, & Pandika, 2018).

1.3. Gene-environment interaction (G × E)

Developmental research reveals that a large majority of children follow a general pattern of aggression over their life course (e.g., Alink et al., 2006; Côté, Vaillancourt, LeBlanc, Nagin, & Tremblay, 2006; Nærde, Ogden, Janson, & Zachrisson, 2014; NICHD, 2004; Tremblay et al., 2004). Typically, children begin displaying aggressive behavior early but quickly desist in late childhood and early adolescence (Alink et al., 2006; Broidy et al., 2003; Nagin & Tremblay, 1999; NICHD, 2004; Tremblay et al., 2004). But not all children follow this pathway. Of interest is the relatively small group of individuals—which is almost exclusively made up of males (Moffitt, Caspi, Rutter, & Silva, 2001)—who display a higher frequency of aggressive behavior than others in their age group during toddlerhood and are then observed to continue their aggressive behavior at a fairly high and stable rate throughout adolescence and adulthood (Brame et al., 2001; Broidy et al., 2003; Keenan & Wakschlag, 2000; Moffitt, 1993; Nagin & Tremblay, 1999). Individuals who follow this trajectory tend to present with a range of developmental, personal, and legal problems throughout the life course (Jennings, Rocque, Fox, Piquero, & Farrington, 2016; Moffitt, 1993; Moffitt, Caspi, Harrington, & Milne, 2002; Raine et al., 2005). Not surprisingly, persistently aggressive individuals are more likely to experience incarceration than their peers (Jennings et al., 2016; Martinez, Lee, Eck, & O, 2017; Wolfgang, Figlio, & Sellin, 1972).

Persistently aggressive individuals almost always show heightened signs of physical aggression as children. But Robins's (1978) paradox makes an important observation: although persistently antisocial individuals almost always show signs of problematic behavior early in the life course, most children who display such behaviors will eventually desist. In other words, aggressive behavior and its consequences—incarceration being our focus—are not due solely to genetic risk factors. Rather, genetic influences are contingent upon the environment in which individuals live. As such, we anticipate a gene-environment interaction (G × E).

There are several perspectives that guide the development of G × E hypotheses.[4] One, known as the social compensation model, is

[2] To be specific, GWAS focuses on single nucleotide polymorphisms (SNPs) and not on specific genes. For the present purposes, a SNP can be thought of as a unique marker in the human genome. There are approximately 3 billion loci in the human genome. But only about 30 million or so are expected to vary from person-to-person, meaning there are roughly 30 million SNPs in the human genome. A gene—of which there are approximately 20,000 in the human genome—can be "tagged" by SNPs. Some genes will contain more than one SNP. The mechanics of GWAS are straightforward. In essence, the analysis is conducted by estimating a correlation (or a regression coefficient) between each SNP and the outcome of interest. Because there are potentially millions of tests performed, GWAS researchers use a corrected *P*-value threshold of 5×10^{-8}. This has proven effective at limiting false-discoveries and increasing rates of replication (Visscher et al., 2017).

[3] There is even a large scale consortium dedicated to applying GWAS to the study of social science outcomes (SSGAC): https://www.thessgac.org.

[4] It is worth noting that there have been other, more elaborate, developments in the G × E literature. For instance, Belsky et al. (2007) proposed the differential susceptibility model. This model suggests individuals with certain genetic profiles may be more responsive to their environment in a "for better and for worse" fashion; they respond well to positive environments, but they respond

applicable here (Boardman, Daw, & Freese, 2013; Monroe & Simons, 1991; Shanahan & Hofer, 2005; Zuckerman, 1999). As Shanahan and Hofer (2005, p. 67) pointed out, we can think of the social compensation model as identifying how positive or neutral environments *compensate* for genetic risk factors. This observation inspired our second hypothesis:

Hypothesis 2. The impact of the genome-wide polygenic score (PGS) for aggressive behavior on lifetime incarceration risk will be moderated by childhood SES.

Childhood SES is one of the most consistent socio-environmental predictors of contact with the criminal justice system. Indeed, childhood SES has recently been classified as a fundamental cause of offending (Barkan & Rocque, 2018) because studies have shown that low childhood SES is associated with numerous risk factors, such as toxic stress (English, 1998; Mersky & Reynolds, 2007), improper parenting (Bornstein & Bradley, 2012), poor neighborhood conditions (Anderson, 1999; Sampson, 2013), contact with delinquent peers (Akers, 1988; Fergusson & Horwood, 1999; Heimer, 1997), and low levels of attachment to family and school (Hay, Fortson, Hollist, Altheimer, & Schaible, 2006). Each of these risk factors is known to predict involvement with the criminal justice system, so it is reasonable to think childhood SES could moderate the relationship between genetic propensity for aggressive behavior and incarceration risk.

One indicator that can be used to tap into childhood SES is parent educational attainment because SES is an outcome that, for most, is at least partially the result of education level. Thus, criminologists have recognized the robust ability of education level to serve as a proxy for SES and to predict offending more generally (e.g., Derzon, 2010; Osgood, Wilson, O'Malley, Bachman, & Johnston, 1996; Wilcox & Clayton, 2001). For our purposes, parent educational attainment will serve as an indicator of the focal participant's childhood SES.

In particular, we propose that individuals with high parent educational attainment have less exposure to risk factors, which is to say higher parental educational attainment may be able to "compensate" or dampen the association between one's genetic propensity for aggressive behavior and their risk of incarceration over the life course. The conceptual model that will guide our analysis is presented in Fig. 1.

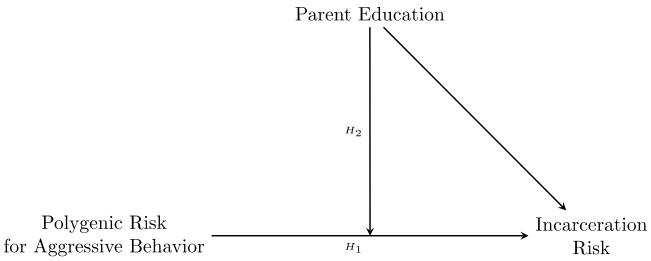

Fig. 1. Theoretical model.

2. Methods

2.1. Data

Data for this analysis come from the Health and Retirement Study (HRS). HRS is a nationally representative longitudinal study of Americans over age 50 conducted every two years from 1992 to 2014. HRS consists of six birth cohorts: 1) the Study of Assets and Health Dynamics Among the Oldest Old (AHEAD [born before 1924]); 2) Children of Depression (CODA [born 1924–1930]); 3) HRS [born 1931–1941]; 4) War Babies (WB [born 1942–1947]); 5) Early Baby Boomers (EBB [born 1948–1953]); and 6) Mid Baby Boomers (MBB [born 1954–60]).

DNA samples in HRS were collected from participants between 2006 and 2010 using the Illumina Human Omni-2.5 Quad beadchip, which covers approximately 2.5 million single nucleotide polymorphisms (SNPs) across the human genome. In other words, each HRS participant was genotyped for 2.5 million genetic markers. Of the 16,000 participants who submitted DNA for genotyping, 15,708 passed the University of Washington Genetics Coordinating Center's (GCC) standardized quality control processes.[5] We will draw on the genotype data as well as the survey data collected as part of the normal interview process.

The genome-wide PGS created in this study was based on the findings from GWAS that relied on samples of European descent (Pappa et al., 2016). Geneticists have long noted that findings from one population group may not be replicable in other ancestral groups due to population stratification and differential patterns of ancestral makers that are unique to each group (Conley & Fletcher, 2017). To minimize confounding effects due to these influences, we follow convention in genomics research and restrict our analysis to participants whose genotypes were consistent with European-ancestry populations and who self-reported as non-Hispanic Whites (Martin et al., 2017). Doing so removes a large portion of the variation that is link to ancestral populations and, therefore, does not allow us to explore any specific hypotheses relating to race/ethnicity. We will also include controls for the first 10 ancestry principal components (Price et al., 2006).

2.2. Measures

2.2.1. Lifetime incarceration

The dependent variable in this study was measured during the most recent waves of data collection, which were conducted between 2012 and 2014. All participants were asked the following question: "Have you ever been an inmate in a jail, prison, juvenile detention center, or other correctional facility?" Respondents replied with *no* (coded 0) or *yes* (coded 1). As reported in Table 1, approximately 5% of the sample indicated they had experienced incarceration, but those respondents were almost exclusively drawn from the male subsample, which had 9% prevalence compared to 2% for females. This prevalence estimate is consistent with prior literature (Bonczar, 2003; Muller & Wildeman, 2016). Given the HRS sample had a mean age of ≈71 when this survey question was asked, it is appropriate to consider it a lifetime measure of incarceration.

Of course, there may be concern over recall bias if some participants had forgotten about an incarceration experience and, therefore, underreported their involvement. But two pieces of evidence suggest this concern is unlikely to have impacted our analysis. First, it is well documented that incarceration experiences are traumatic and, in many ways, life-altering (Blevins, Listwan, Cullen, & Jonson, 2010). An experience that has such far-reaching implications is unlikely to be systematically forgotten. Second, evidence reveals that survey

(*footnote continued*)
badly to negative/stressful environments. Although the Belsky et al. (2007) differential susceptibility model has helped scholars gain insight into certain phenotypes, practical considerations have limited researchers' ability to distinguish empirical evidence that would support Belsky's model from evidence that would support the simpler social compensation model. For this reason, along with several other considerations that come with analyzing whole-genome data, we will draw on the social compensation model in the present study. But readers should not interpret this as evidence against the differential susceptibility model. Rather, our analysis cannot offer an appropriate test of the differential susceptibility model.

[5] All genomic data are put through a rigorous process of quality control (QC) before they are released to analysts (Laurie et al., 2010). The QC process typically involves several rounds of algorithmic pruning of genomic "calls" that are likely to be errors followed by a round of human processing that is aimed at finding errors. In short, the QC process tries to rule out all sources of noise that could be attributed to complications that arise during genomic data collection.

Table 1
Descriptive statistics.

	Males Proportion/mean (standard deviation)	Females Proportion/mean (standard deviation)	Overall Proportion/mean (standard deviation)
Lifetime incarceration (=1)	0.093 (–)	0.018 (–)	0.048 (–)
High parental educational attainment (=1)	0.670 (–)	0.610 (–)	0.634 (–)
Polygenic score for aggressive behavior	−0.004 (0.001)	−0.004 (0.001)	−0.004 (0.001)
Age	71.738 (9.717)	71.183 (10.375)	71.408 (10.116)
Birth year	1941.433 (9.905)	1941.978 (10.490)	1941.757 (10.259)
N	2724	3992	6716

Note: Statistics for the polygenic score for aggressive behavior are based on raw values prior to standardization.

respondents—especially those who have an established rapport with the researchers, as the HRS participants likely do after being enrolled in the study for multiple years—overwhelmingly respond in a truthful manner to questions about their criminal histories (Henry, Moffitt, Caspi, Langley, & Silva, 1994; Krohn, Lizotte, Philips, Thornberry, & Ball, 2013). Taken together, these points suggest there is little reason to be concerned that the HRS participants have systematically misrepresented their incarceration experiences.

2.2.2. High parental educational attainment

Garnering a suitable measure of childhood SES among a sample of elderly Americans presents several challenges. Despite the usual concerns of measuring SES (e.g., the complications of defining SES), we faced the added burden of measuring childhood SES among participants who were 50+ years removed from their childhood. Thus, we use parental educational attainment as a proxy for childhood SES. Given the role of educational attainment as an antecedent variable in the cascade of factors (like income, wealth, and job status) that underlie SES, it stands to reason that parental education level can serve as a robust and salient proxy of childhood SES. Nonetheless, it was important to keep in mind that education levels were quite different in the early parts of the 20th century, so we coded our measure relative to what a high education would have represented during those times.

HRS participants were asked to report "the number of years of education completed" separately for both of their parents. We created an indicator of exposure to high parental educational attainment by taking the highest level of education reported for either parent. For example, if a respondent reported her mother completed 12 years and father completed 10 years, we coded the measure as 12. Next, we dichotomized the measure so it indicated whether at least one parent completed high school (12 or more years). Thus, parental educational attainment was coded such that *neither parent completed high school* (coded 0) or *at least one parent completed high school* (coded 1). In cases where information was missing for one parent (e.g., a single-parent household), the level of education for the single parent was counted as the highest level of education.

The HRS participants' parents reached their formative years during a time when graduating high school represented a relatively high level of education. We see evidence of this in the HRS sample: only 39% of HRS participants were raised in homes where both parents graduated high school and >30% were raised in a home where neither graduated high school. Thus, our measure of parental educational attainment is not a constant across all participants and a substantial portion of our sample (≈30%) are identified as being in the "risk" group.[6]

2.2.3. Polygenic score (PGS) for aggressive behavior

Analyses in this study are based on a genome-wide polygenic score (PGS) for aggressive behavior that was computed using summary statistics from a recent GWAS on aggressive behavior (Pappa et al., 2016).[7] The PGS takes the following form:

$$PGS_i = \sum_{j=1}^{J} \beta_j G_{ij}$$

where PGS_i represents the polygenic score for an individual HRS participant i, β_j is the beta (or weight) coefficient for each genetic variant j estimated by the Pappa et al. (2016) GWAS, and G_{ij} is the number of "risk" variants (i.e., the variant positively associated with aggressive behavior) that HRS participant i possesses at variant j. In short, the genome-wide PGS for HRS participant i is computed as a weighted sum of all the "risk" variants that s/he carries.

Polygenic scoring for this analysis was conducted according to the conventional methods described by Dudbridge (2013) using the PRsice software package (Euesden, Lewis, & O'Reilly, 2015).[8] Note that we coded all β_j so that higher values are associated with a higher propensity for aggression. Following this routine, the computed PGS was normally distributed in the full sample and in the male/female subsamples. We provide descriptive statistics for the unstandardized PGS in Table 1 in order to give a glimpse into the data, but the PGS was standardized to have a mean of 0 and a standard deviation of 1 for all other analyses.

2.2.4. Covariates

Confounding influences must be controlled in observational studies, and genome-wide research is no different. But genome-wide research is different in terms of the range of potential confounders that must be considered. While there are many factors antecedent to incarceration, very few are antecedent to the PGS. Nonetheless, it is imperative to include controls for population stratification—essentially, ancestry (Barnes, 2018)—when performing a genome-wide analysis (Price et al., 2006). We followed current convention and conducted a principal components analysis on the genome-wide data. We then controlled for the largest 10 principal components (PCs) in order to rule out confounding influences due to population stratification (Price et al., 2006). The PCs were constructed on the basis of genome-wide SNPs with pair-

[6] We recognize parental educational attainment is an imperfect proxy of childhood SES, so we performed several rounds of robustness checks to ensure our findings were not sensitive to a particular measurement or coding strategy. We found the substantive results were not affected, regardless of how we coded the parental education measure (e.g., leaving the measure as a count, including just one parent, and using an ordinal count to index both parents). Also, we found an alternative measure of childhood SES—the respondent's retrospective perception of their family's financial well-being when they were a child—resulted in the same substantive conclusions.

[7] The measurements of aggressive behavior used by Pappa et al. (2016) were all based on validated questionnaires which assessed aggressive behavior through items such as cruelty to animals, destruction of things, disobedience, defiance, mean to others, high tempered, angry moods, hitting or hurting others, irritable, easily frustrated, threatens others, and fighting, to name a few. The SNP heritability was estimated to range from 10 to 54%.

[8] We performed several sensitivity checks to assess whether different strategies for polygenic scoring altered the substantive results. For instance, we computed the PGS using only the "top hits", meaning the SNPs that had the largest effects, from the Pappa et al. (2016) GWAS. We also explored different techniques of pruning and clumping. None of these approaches altered the substantive conclusions of our analysis.

wise squared correlation (R^2) smaller than 0.20.

Because the HRS includes multiple cohorts of participants, we control for each respondent's age at the most recent wave of data collection and we include a control for birth year to account for cohort effects. As reported in Table 1, the mean (and median) age of respondents included in the analysis was approximately 71 and this did not vary across sex. Finally, it is possible that confounding can affect a test for G × E (see Motz, Tanksley, Liu, Mersha, & Barnes, 2019). We consider this possibility in our analysis with our second robustness check (discussed in more detail in the Robustness Checks section).

2.3. Analysis plan

The analysis unfolded in two steps. First, we estimated the association between the genome-wide PGS for aggressive behavior and lifetime incarceration experience (Hypothesis 1) by logistic regression. Second, to test for a G × E (Hypothesis 2), we included a multiplicative term between the genome-wide PGS and parental educational attainment in the prediction of lifetime incarceration.

Before moving to the analysis, two other points must be addressed. First, it is important to acknowledge the severe level of stratification across sex that is traditionally observed on criminal justice outcomes like incarceration (Barnes et al., 2015; Brame et al., 2014; Wilson & Herrnstein, 1985). Aggression research has shown that males are more likely to aggress physically while females aggress using social means (e.g., Archer, 2004). Physical aggression is far more likely to trigger a response from the criminal justice system and, thus, we have reason to believe the PGS for aggressive behavior could differentially predict lifetime exposure to incarceration by sex. As such, we performed all analyses stratified by sex, an approach that is consistent with recent genetically informed analyses in the social sciences (Perry, 2016).

Second, two robustness checks were conducted to assess whether known sources of confounding in genomic studies impacted our estimates. Specifically, we assessed a) whether the genome-wide PGS for aggression provided unique predictive power compared to the well-established PGS for educational attainment (Wertz et al., 2018) and b) whether gene-environment correlation complicated our results (Scarr & McCartney, 1983). These analyses revealed that our results were robust and, therefore, do not reflect the most likely sources of bias. We discuss these results in more detail at the end of the Findings section.

3. Findings

3.1. Primary results

Table 2 provides the results gleaned from our logistic regression models that tested for an association between the genome-wide PGS for aggressive behavior and lifetime incarceration risk, stratified by sex. As can be seen in Models 1M and 1F, consistent with Hypothesis 1, respondents who scored higher on the PGS for aggressive behavior had greater risk of experiencing incarceration at some point in their lifetime. Yet the association was statistically significant only in the male subsample ($P < 0.05$). For males, a one standard deviation increase in the PGS for aggressive behavior increased the odds of experiencing incarceration by 15% ($100 \times [e^{0.140} - 1]$). After adding covariates to the models, the results remained substantively unchanged (see Models 2M and 2F).

Models 3M and 3F reveal the results of our tests for a G × E between the genome-wide PGS for aggressive behavior and parental educational attainment (i.e., Hypothesis 2). For males, the parameter estimate for the interaction term was $b = -0.295$ and it was statistically significant ($P < 0.05$). Consistent with the prediction of the social compensation G × E model, the impact of the PGS for aggressive behavior was significantly weaker among those who were raised in a household where at least one parent completed high school. For this group, the effect of the PGS on incarceration risk was reduced to 0.056 (0.351–0.295), an estimate that was not statistically distinguishable from 0.00 ($P > 0.05$).[9] For males who were raised in a household where neither parent completed high school, the impact of the PGS on lifetime incarceration risk was $b = 0.351$ (odds ratio = 1.420, $P < 0.05$). A one standard deviation increase in the PGS increased the odds of incarceration by 42% for males born into households with relatively low parental educational attainment.

Fig. 2 displays predicted probabilities of lifetime incarceration risk as a function of PGS scores for the male subsample. These values were computed for males who grew up in a household where neither parent completed high school (demarcated as the line with the boxes as markers) and for males who grew up in a household where at least one parent completed high school (demarcated as the line with triangles as markers). As can be seen, the function is positive for males from low parental education households. But for males from high parental education households, the function is relatively flat. These results were anticipated by the social compensation G × E model, which suggests protective environments can suppress genetic risks for an outcome.

The dashed vertical line in the figure reveals the location where the two prediction lines become statistically distinguishable. That location was at the 1.1 PGS standard deviation mark. What this means is that PGS scores for aggressive behavior do not lead to statistically different predictions for incarceration risk until the PGS values are quite high (i.e., 1.1 standard deviation [or more] above zero). When viewed from the perspective of the environmental moderator, the vertical line reveals the location where parental educational attainment has a statistically significant impact on men's incarceration risk. Parental educational attainment is unable to statistically distinguish between the incarceration risks of two males who have low polygenic risk for aggressive behavior. But parental educational attainment is able to differentiate the incarceration risks of two individuals who have high polygenic risk. This implies that raising parental educational attainment might reduce incarceration risk for men with the highest genetic risk for aggressive behavior.

Returning to Table 2, the impact of the PGS for aggressive behavior—and the G × E with parental educational attainment—for females can be observed in model 3F. The focal coefficients (i.e., the coefficients for the PGS, parental educational attainment, and their interaction) were in the expected directions (positive for the PGS, negative for parental educational attainment, and negative for the interaction term), but only the coefficient for parental educational attainment achieved statistical significance.

A potential explanation for the sex differences observed here might be that males in the HRS carry more genetic risk than females. We assessed this possibility by observing the distribution of the PGS across sex. As shown in Fig. 3, there was no substantive difference in the distribution of the PGS for aggressive behavior between males and females in the HRS sample. A *t*-test confirmed there is no meaningful difference in genetic risk across sex ($P > 0.05$).

3.2. Robustness checks

Our analysis uncovered a statistically significant interaction between parental educational attainment and the PGS for aggressive behavior in predicting lifetime incarceration risk for males. It is important, therefore, to try and rule out the factors most likely to bias our

[9] We were concerned that this null effect may have emerged as a result of limited variation on the PGS among respondents who were born into homes where at least one parent graduated from high school. In other words, if all the genomic risk for aggressive behavior was clustered among those from low educated households, then we might expect a null result for respondents from well-educated homes. But this does not appear to be the explanation because the range and standard deviations for the PGS were nearly identical across the two subsamples (scores on the standardized PGS were between −3 and 4 and the standard deviation was approximately 1.00 for both subsamples).

Table 2
Logistic regression of lifetime incarceration on the polygenic score for aggressive behavior, high parental educational attainment, and their interaction, by sex (Hypotheses 1 and 2).

	Male			Female		
	(1M)	(2M)	(3M)	(1F)	(2F)	(3F)
	b (*SE*)	*b* (*SE*)	*b* (*SE*)	*b* (*SE*)	*b* (*SE*)	*b* (*SE*)
Polygenic score for aggressive behavior	0.140** (0.070)	0.140* (0.071)	0.351*** (0.129)	0.089 (0.129)	0.021 (0.134)	0.081 (0.229)
High parental educational attainment (=1)	–	−0.134 (0.155)	−0.078 (0.161)	–	−0.495* (0.286)	−0.494* (0.286)
Polygenic score for aggressive behavior × high parental educational attainment	–	–	−0.295** (0.150)	–	–	−0.087 (0.273)
Age	–	−0.085 (0.072)	−0.088 (0.072)	–	−0.176 (0.133)	−0.175 (0.133)
Birth year	–	−0.031 (0.070)	−0.033 (0.070)	–	−0.036 (0.130)	−0.036 (0.130)
Intercept	−2.040*** (0.254)	4.110 (5.159)	4.199 (5.169)	−4.815*** (1.166)	7.253 (9.703)	7.212 (9.701)
N	2724	2724	2724	3992	3992	3992
Log likelihood	−834.499	−805.887	−803.946	−345.355	−293.095	−293.044
AIC	1692.997	1641.773	1639.893	714.709	616.191	618.088

Note: All models control for the largest 10 principal components to adjust for population stratification; *b* = unstandardized regression parameter estimate; *SE* = standard error; AIC = Akaike information criterion.
* $P < 0.1$, two-tailed.
** $P < 0.05$, two-tailed.
*** $P < 0.01$, two-tailed.

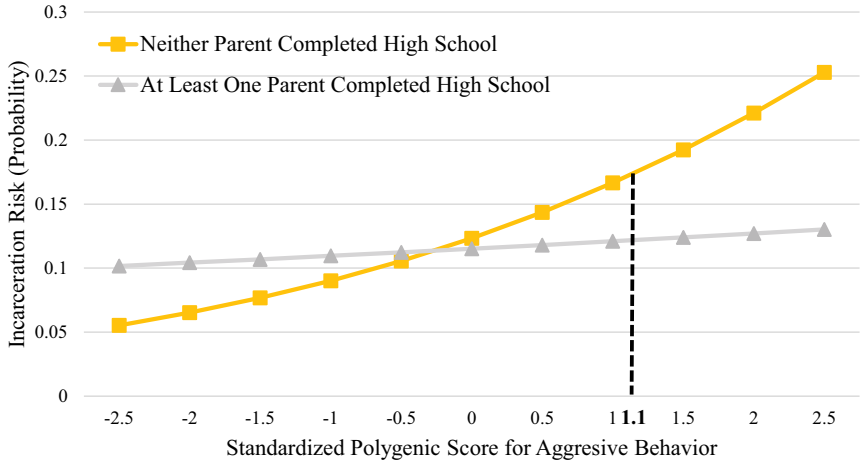

Fig. 2. The effect of the polygenic score for aggressive behavior on lifetime incarceration risk across levels of parental educational attainment (male subsample).
Note: The predicted probabilities are statistically distinguishable for all polygenic score values that lie to the right of the dashed line.

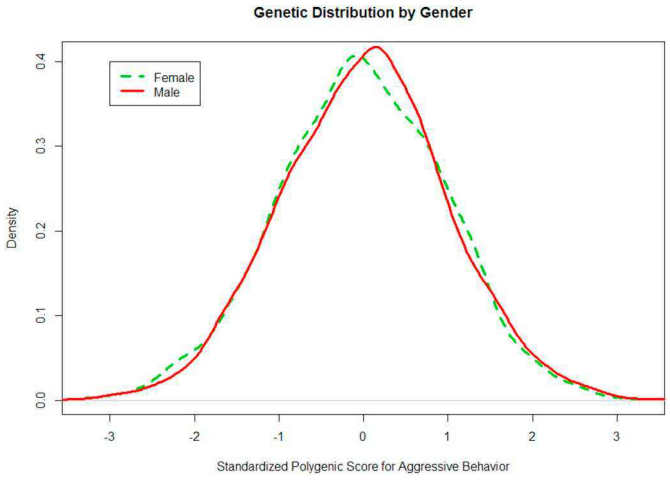

Fig. 3. Distribution of the polygenic score for aggressive behavior for females and for males.
Note: A *t*-test was conducted to test for differences in the mean of the polygenic score between males and females. There was no evidence of a sex difference ($P = 0.124$, two-tailed).

results. First, we analyzed whether the PGS for aggressive behavior tapped into genetic risk that was independent of the genetic risk captured by another, well-established PGS: the PGS for educational attainment (Okbay, Beauchamp, et al., 2016). This was an important exercise because recent evidence demonstrates a robust and consistent relationship between the PGS for educational attainment and criminal offending (Wertz et al., 2018). We analyzed the association between the PGS for educational attainment and the PGS for aggressive behavior and found that the correlation was substantively small in the full sample ($r = -0.052$, $P < 0.05$) and in the male subsample ($r = -0.045$, $P < 0.05$). This suggests the PGS for aggressive behavior is unlikely to be acting as a proxy for genetic influences on educational attainment.

Second, passive gene-by-environment correlation (*r*GE) could have played a role in impacting our estimates. Passive *r*GE occurs when parents' genotypes are correlated with their environment and, thus, the child's genotype is also correlated with those same environments. In the present case, individuals with low polygenic risk for aggressive behavior could be more likely to be raised by parents with higher levels of education, creating a correlation between the child's genotype and his/her exposure to high parental educational attainment. If such *r*GE exists, then the interpretation of the G × E becomes more complicated (Fletcher & Conley, 2013; Jaffee & Price, 2007; Scarr & McCartney,

1983; Wagner, Li, Liu, & Guo, 2013). To assess this possibility, we compared the mean of the PGS for aggressive behavior between respondents who were raised in a household where neither parent completed high school and those who were raised in a household where at least one parent completed high school. The mean PGS scores did not differ by parental educational attainment for the full sample ($t = 0.66$, $P > 0.05$) but there was a marginally statistically significant mean difference in the male subsample ($t = 1.97$, $P = 0.05$). The mean difference between the two groups was substantively small (0.016 in standard deviation units), suggesting our G × E finding is unlikely to be driven by passive *r*GE.

4. Discussion

Incarceration is rarely a positive turning point. Spending time in prison or jail is associated with a host of negative life outcomes, including employment problems, family disruption, future antisocial behavior, and even early death (Turanovic et al., 2012; Uggen & McElrath, 2014). These links have serious implications for US citizens, a country with the highest incarceration rate in the world (Glaze & Herberman, 2013). Because incarceration touches so many, particularly those who come from disadvantaged environments, it is understandable that the bulk of the literature on the risk factors for incarceration has focused on social and structural forces (Pratt, 2009; Vogel & Porter, 2016). These sources are undoubtedly important, but a full explanation of the risks for incarceration may necessitate a broader search: one that embraces the possibility that socio-environmental factors "trigger" or "compensate" for risk factors that can be traced to individual differences.

Accordingly, the present study investigated the possibility that the impact of genetic propensity for aggression on lifetime incarceration risk can be suppressed by exposure to a protective environment. Drawing on genome-wide data, we found evidence of a positive association between a genome-wide polygenic score for aggressive behavior and lifetime incarceration risk. Evidence to support the social compensation G × E model also emerged, revealing that the polygenic risk for aggressive behavior was positively associated with incarceration risk but only for participants who were born into a home where neither parent had completed high school. The polygenic score did not predict incarceration for those who were born into a home where at least one parent had completed high school. With this in mind, we highlight four contributions from this study that warrant consideration. We then move to a discussion of the broader implications of genomic research in criminology.

4.1. Contributions

First, we drew from the extensive literature on the relationships between aggressive behavior, SES, parental educational attainment, and contact with the criminal justice system and integrated it with the social compensation G × E model (Shanahan & Hofer, 2005) to develop and test two hypotheses. This is notable because, as Perry (2016, p. 1685) stated, there has been "…a striking absence of social theory in the genetics literature, where G × E effects are often reported with little speculation about social mechanisms underlying them (Freese & Shostak, 2009; Moffitt, Caspi, & Rutter, 2005; Rutter, Moffitt, & Caspi, 2006)." Our findings provided support for the social compensation G × E model, which has implications for our understanding of how the social environment shapes individuals' lives. Adverse environments may trigger genetic risks, while favorable environments may suppress or compensate for genetic risks.

These results suggest that children with a high genetic propensity for aggression may learn to control their behavior through disciplinary practices before they reach ages where criminal offending is likely (see, generally, Tremblay et al., 2005). It may be that highly educated households are more effective at disciplining out the aggressive behavior of their genetically at-risk children, effectively silencing the impact of the genetic predisposition. An alternative explanation is that, given equivalent expression of aggressive behavior, individuals from socioeconomically advantaged families are less likely to be incarcerated. But if this were the driving force, we would expect individuals from highly educated households to have the lowest risks of incarceration across all levels of the polygenic score. But, in our sample, higher parental education was associated with lower incarceration risk, but only among males with relatively high polygenic scores for aggressive behavior. This means that without genetic information, differences in incarceration risk across parental education levels were unobservable. This finding underscores the significance of incorporating genetic factors into empirical and theoretical models of the social causes of criminological outcomes (Li, Liu, & Guo, 2015; Liu, Li, & Guo, 2015). As Simons et al. (2011, p. 884) pointed out, "… genetic research thus does not challenge the importance of environmental factors in determining human behavior; instead, it shows how social scientific explanations might be made more precise by incorporating genetic information (Guo, Roettger, & Cai, 2008; Guo, Tong, & Cai, 2008; Shanahan, Vaisey, Erickson, & Smolen, 2008)."

A second feature of this study is that we found genetic influences on incarceration risk were only statistically significant for the male subsample. Explanations for this pattern of findings could invoke gendered theories of criminality (e.g., Mears, Ploeger, & Warr, 1998) and perhaps even evolutionary psychology. For instance, it may be the case that the expression of aggression is contingent on social and physiological contexts. Male aggression is more likely to be of the physical variety compared to the types of aggression expressed by females (Archer, 2004; Björkqvist, Lagerspetz, & Kaukiainen, 1992; Lagerspetz, Björkqvist, & Peltonen, 1988; Moffitt et al., 2001). This may be an evolved mechanism that is tied to the generally larger physical stature of the male body compared to the female body. These physiological differences might have, over time, led to sex differences in aggressive behaviors (see Campbell, 2005), which have ultimately resulted in sex differences in incarceration rates.

Third, we analyzed lifetime incarceration experiences rather than age-specific risk, like risk in early adulthood, that is more commonly assessed in the criminological literature (Cullen, 2011). Results based on samples collected among younger participants may be biased due to right censoring, meaning some individuals may experience incarceration after the observation window has closed. For this reason, we believe the focus on a lifetime risk measure represents an important contribution in its own right.

Fourth, this study is among the first to assess whether a genome-wide polygenic score has any predictive ability for a criminological outcome (see Wertz et al., 2018). This is a significant advancement over conventional indirect approaches like heritability studies (Barnes et al., 2014; Burt & Simons, 2014) and candidate gene studies (Simons et al., 2011; Wells et al., 2017) that have traditionally been employed by criminologists. The genome-wide approach we employ represents a key development for human behavioral research because it is a step toward a more holistic and direct understanding of the link between genetic risks, social risks, and human behavioral outcomes (Freese, 2018). Indeed, our study highlights the possibility of a synergistic relationship between the genome and the social context on human behavioral outcomes.

4.2. Limitations

It is important to remind readers to interpret these findings cautiously. Although we address many of the limitations throughout the manuscript, there are several limitations that are worth noting again. Each of these primarily deals with sample composition. First, the current study limits the sample to non-Hispanic Whites, which reduces the generalizability of our results to minority groups. Non-Hispanic Whites tend to have lower lifetime incarceration risks compared to minorities,

which draws attention to the need for sociogenomics work to identify solutions to the challenges imposed by population stratification that allow for analysis of all population groups. Second, the sample is comprised of older participants, which poses potential missing data problems because some participants may have passed away before they report being incarcerated. This is a well-known concern and researchers have developed sensitivity tests to determine whether this sort of mortality selection bias has impacted study results. To address this limitation, we performed the test recommended by Domingue et al. (2017) and the results suggest that our findings are robust when appropriate corrections are made. We report the results from this robustness check in the supplemental analysis.

4.3. Broader considerations

Because this is one of the first studies to capitalize on genome-wide data to analyze a criminological outcome, it is important that we consider the broader substantive and philosophical concerns that may be sparked by our analysis. We are sensitive to the past harms that have come in the wake of research attempting to link biological factors to criminality (Rafter, Posick, & Rocque, 2016). We must consider, therefore, what our findings do and do not have to say about "born criminals" and whether criminality can be predicted early in the life course.

Fortunately, there are precursors to this discussion (Reimers, Craver, Dozmorov, Bacanu, & Kender, 2019). Directly relevant is Turkheimer's (1998) discussion of weak biologism. We can characterize weak biologism as the recognition that human behaviors, for the most part, all rely on the same biological pathways. Thus, most human outcomes do not have their own unique developmental mechanisms, meaning genetic factors cannot work deterministically for complex outcomes like criminality or incarceration. But we believe it is prudent to make two additional points. First, our findings suggest outcomes characterized by weak biologism can be contingent on environmental inputs. The primary takeaway from our study is that complex outcomes like aggressive behavior and its consequences (i.e., incarceration risk) will only be explainable in probabilistic terms that are contingent on biological *and* environmental processes.

The second point is that genome-wide polygenic scores will continue to be refined, mainly as a function of sample sizes in the GWAS data used to generate them. As they are refined, the predictive power of polygenic scores will improve. This can already be seen by observing the progression of prediction accuracy for educational attainment from the first GWAS in 2013 (Rietveld et al., 2013) to the most recent iteration in 2018 (Lee et al., 2018). In just a few short years, the predictive ability of the polygenic score for educational attainment has increased from $R^2 \approx 2\%$ to $R^2 \approx 10\%$. In doing so, it has established itself as one of the strongest single predictors of one's educational attainment.

The genome-wide polygenic score for aggressive behavior is thus weak, but it has strong potential. It is weak because it does not specifically identify a biological pathway, meaning it will not lead directly to any targeted intervention. But its potential is strong in the sense that, if combined with information about environmental risk/protective factors, it may become an increasingly accurate indicator of those at highest risk for displaying aggressive behavior (probabilistically speaking). The problem we now face is the specter of the capacity to screen without the capacity to treat or prevent. We must begin to consider the ethical and moral questions that will follow if it becomes possible to predict, with any degree of accuracy, the risk for displaying aggressive behavior by combining genomic data with environmental predictors. We anticipate that labeling theory will spring back into popularity (see, generally, Mukherjee, 2016).

Our point here is not to be alarmists but rather to suggest genome-wide polygenic scores will become increasingly more accurate predictors of complex human outcomes, even those characterized by weak biologism. And their power to predict will increase as G × E research reveals the scenarios where the environment can protect or exacerbate the genetic influence. It is thus important that scholars and philosophers begin to consider what this means for social science research, whether this kind of work should play a role in policy discussions, and what role scholars should take in terms of advocating policies to government officials. For the moment, we are comfortable in making one recommendation: no genomic screening should ever take place until effective and ethical responses have been established.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.avb.2019.07.002.

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