Educational Consequences of Early Crime and Punishment: Testing A Genetically Informed Life-course Model Using the Add Health Data

Hexuan Liu[1,2], Ryan T. Motz[1], Peter Tanksley[1], J.C. Barnes[1], Kathleen Mullan Harris[3, 4]

[1] School of Criminal Justice, University of Cincinnati
[2] Institute for Analytics Innovation, University of Cincinnati
[3] Department of Sociology, University of North Carolina at Chapel Hill
[4] Carolina Population Center, University of North Carolina at Chapel Hill

ABSTRACT

In this study, we develop a life-course model to investigate the complex relationships among genetic inheritance, criminal justice (CJ) involvement (e.g., arrest, conviction, or incarceration), and educational outcomes. To test the model, we use whole-genome data from the National Longitudinal Study of Adolescent to Adult Health (Add Health) to conduct an analysis based on the most powerful polygenic score constructed to date for educational attainment (Lee *et al.* 2018). We find that participants with higher polygenic scores for educational attainment were significantly less likely to report CJ involvement during their adolescence. We then show the genetic association with the risk of CJ involvement is attributable to a range of individual and social factors, particularly experiences at school. Finally, we find evidence that adolescent CJ involvement mediates the associations between the education polygenic score and participants' educational outcomes in adulthood (e.g., years of schooling, high school completion, and tertiary education participation). These findings reveal that CJ involvement at an early age may prevent individuals from realizing their full genetic potential for educational attainment. Findings in this study also provide important insights to assess the effect of genetic confounding in research of causal relationships between CJ involvement and later-in-life outcomes.

INTRODUCTION

Sociologists have long been interested in the relationship between social stratification and crime. Education is one of the leading mechanisms of social stratification and mobility in modern societies (Blau and Duncan 1967; Bourdieu and Passeron 1977; Coleman 1988; Featherman and Hauser 1976; Hout 1988; Mare 1993; Sewell *et al.* 1969; Teachman 1987). Criminal behavior and its consequences, particularly involvement with the criminal justice (CJ) system (e.g., arrest, conviction, incarceration, etc.), may play important roles in influencing one's educational opportunities and socioeconomic well-being in later life (Apel and Sweeten 2010; Kirk and Sampson 2013; Kling 2006; Western 2002; Western and Pettit 2005).

Understanding the impact of CJ involvement on education, however, presents critical theoretical and methodological challenges (Kirk and Wakefield 2018). In particular, individuals are not randomly assigned to have contact with the CJ system. Rather, they select into the CJ system on the basis of various individual characteristics and social processes. This means that any relationship between CJ involvement and education could be spurious owing to those selection factors. One important source of selection bias is genetic variation. Genetic factors not only influence individual traits such as cognitive ability (Savage *et al.* 2018) and delinquency (Tielbeek *et al.* 2017), but also play a role in individuals' selection of social environments such as schools and neighborhoods that contribute to both CJ involvement risk and education (Plomin *et al.* 1977; Scarr and McCartney 1983; Wagner *et al.* 2013; Conley and Fletcher 2017). Thus, the relationship between CJ involvement and education might be confounded, at least partially, by genetic heterogeneity (i.e., genetic confounding). Addressing this possibility has proven challenging as few available datasets provide the measures to do so (Barnes *et al.* 2014).

Research has shown that educational attainment is attributable to economic, social, and cultural influences, as well as genetic inheritance (Conley *et al.* 2015; Eckland 1967; Eckland 1979; Liu 2018; Nielsen and Roos 2015; Scarr and Weinberg 1978). According to family-based heritability studies, on average, genetic factors account for 40 percent of the variation in educational attainment (Branigan *et al.* 2013). Yet, specific mechanisms linking genetic factors and educational outcomes are still not well understood. In particular, the role of criminal behavior and its consequences has largely been overlooked. To be more specific, it is unclear whether—and to what degree—criminal behavior and CJ involvement in early life mediate the genetic association with education (e.g., are some genetic factors associated with higher/lower risk of criminal behavior and CJ involvement during adolescence, which in turn, lead to variation in educational outcomes?).

Recent developments in genomic technology have enabled studies to collect genetic measures across the entire genome (i.e., genome-wide data). Using genome-wide data, researchers have successfully identified associations between genetic variants and various human traits of interest to social scientists. In particular, to date, three genome-wide association (GWA) studies have identified more than a thousand genetic variants associated with educational outcomes (Rietveld *et al.* 2013a; Okbay *et al.* 2016a; Lee *et al.* 2018). Based on the results of these GWA studies, polygenic scores (PGSs) have been developed as compound measures that aggregate estimates of multiple genetic effects on education (Belsky *et al.* 2018; Belsky *et al.* 2016; Conley and Domingue 2016; Conley et al. 2015; Domingue *et al.* 2018; Domingue *et al.* 2015; Liu 2018; Wedow *et al.* 2018). Such PGSs offer social scientists opportunities to assess the complex relationships among genetics, CJ involvement, and education.

In this study, informed by recent socio-genomic research, we develop a life-course model that ties together the two lines of inquiry: (1) the extent to which the relationship between CJ involvement and educational outcomes is attributable to genetic confounding, and (2) the mediating effect of CJ involvement during adolescence on the genetic association with educational outcomes in adulthood. To test the model, we leverage the longitudinal design and genome-wide data in the National Longitudinal Study of Adolescent to Adult Health (Add Health) to conduct an analysis based on the most powerful PGS constructed to date for educational attainment (Harris *et al.* 2013; Lee et al. 2018).

BACKGROUND

Educational Consequences of Criminal Justice Involvement

The United States' CJ system imprisons more people per capita than any other nation (Pratt 2009). Currently, approximately one in every 37 adults (i.e., approximately seven million individuals) is in prison, jail, or under some form of community supervision by a probation or parole officer (Kaeble and Glaze 2016). Around 6-to-10% of all people will serve in prison at some point in their lifetime (Muller and Wildeman 2016). These numbers are alarming as involvement with the CJ system has been found to be associated with a host of detrimental consequences that produce social inequalities, including poor educational outcomes (Haskins 2014; Kirk and Sampson 2013), limited employment opportunities and low income (Apel and Sweeten 2010; Harding *et al.* 2018; Ramakers *et al.* 2014; Western and Pettit 2005), reducing marriage and familial stability (Apel 2016; Massoglia *et al.* 2011; Siennick *et al.* 2014; Turney 2015), declining institutional participation (Brayne 2014; Haskins and Jacobsen 2017; Sugie 2015), and health problems (Geller *et al.* 2012; Sugie and Turney 2017). All these will, in turn,

contribute to an increased risk for future criminal involvement and recidivism (Kirk 2015; Kirk and Papachristos 2011; Liberman *et al.* 2014).

Life-course theories of cumulative disadvantage have provided a theoretical roadmap to understand the negative consequences of early CJ involvement. According to Sampson and Laub (1997 :147), "cumulative disadvantage is generated most explicitly by the negative structural consequences of criminal offending and official sanctions for life changes. The theory specifically suggests a 'snowball' effect—that adolescent delinquency and its negative consequences (e.g., arrest, official labeling, incarceration) increasingly 'mortgage' one's future, especially later life chances molded by schooling and employment." Likewise, Moffitt (1993) theorized that some delinquent adolescents become "ensnared" by the consequences of their antisocial behavior, therefore narrowing their chances for conventional behavior. The effect of CJ involvement may be irrevocable such that an individual's opportunity to "turn over a new leaf" is drastically restricted (see also Giordano *et al.* 2002).

The current study focuses on the educational consequences of CJ involvement during adolescence. Specifically, one of the most important mechanisms through which CJ involvement could affect education is through the official labeling of the person as a "criminal," which modifies the way s/he is treated by social institutions. In schools, for example, students with criminal records may be suspended, expelled, or segregated into specialized programs designed for problem youth due to reasons of accountability and school safety. In addition, time spent in court, in juvenile detention, or reporting to a probation officer may lead to absences, a blemished transcript, and an unstable educational trajectory. The stigma of a criminal label is also detrimental to social relationships of the labeled offender to his or her parents, teachers, and prosocial peers. All these are likely to affect the labeled offender's completion of education and

limit their opportunities to pursue higher education in the future (Kirk and Sampson 2013; Ramey 2016). Moreover, weak bonds to family and school may produce problem behavior such as school dropout and truancy (Gottfredson and Hirschi 1990; Hirschi 1969). Individuals who have offended and been involved with the CJ system may reduce their attachment to family and school, and that may lead to dropouts and incomplete education (Sweeten *et al.* 2009). This is all to say that youth who come into contact with the CJ system are at risk of becoming ensnared in a developmental pathway that wagers their future educational opportunities, thereby lowering their average level of education.

Genetics and Risk of Criminal Justice Involvement

The relationship between CJ involvement and education is complicated and understanding the causal effect of CJ involvement has proven a challenging task. Research has shown that individuals who have been involved with the CJ system differ in various predispositions from those who have avoided the system. These differences include behavioral traits (Agnew 2001), demographic characteristics (Pettit and Western 2004), cumulative socioeconomic (dis)advantages (Wakefield and Uggen 2010), exposure to abuse and violence as both offenders and victims (Western 2015), and other characteristics. If heterogeneity among individuals is ignored or insufficiently considered in an analysis, the estimation of the causal effect of CJ involvement on a later-in-life outcome such as educational attainment might be biased (see, generally, Pearl 2009).

An important source of individual heterogeneity is genetic variation (Kendler 2017; Knopik *et al.* 2017). Since genetic variation is often not directly observed, genetic influences have been conventionally modeled as latent variables using twin, adoptee, or other family data. Recent developments in genomic science and technology have made it possible to collect

information from specific genetic variants located across the entire human genome. The genomic data revolution enables researchers to discover associations between specific genetic variants and outcomes of interest. Such studies are called genome-wide association (GWA) studies. Whereas most GWA studies have focused on physiological and health outcomes such as height (Wood *et al.* 2014), body mass index (Locke *et al.* 2015), cancer (Michailidou *et al.* 2013), and cardiovascular disease (Nikpay *et al.* 2015), there is a growing body of research on outcomes of interest to social scientists such as educational attainment (Rietveld *et al*. 2013a; Okbay *et al.* 2016b; Lee *et al.* 2018), subjective wellbeing (Rietveld *et al.* 2013b; Okbay et al. 2016a), and risk tolerance (Linnér *et al.* 2018).

Utilizing the findings of GWA studies, polygenic scores have been developed as compound measures that aggregate estimates of multiple genetic effects on an outcome (Purcell *et al.* 2009). These PGSs are particularly useful in investigations of human complex traits affected by a large number of genetic variants with moderate-to-small effects. As an example, Pappa *et al.* (2016) conducted a GWA study on aggressive behavior in childhood. Using polygenic scores constructed based on this study, Barnes and colleagues (2018) showed that polygenic scores for aggressive behavior significantly predict incarceration risk among males in the Health and Retirement Study.

Genetic factors are not only associated with crime and CJ involvement through intermediate individual traits such as aggressive behavior and delinquency, they may also contribute to the selection of social environments that raise one's risk for CJ involvement. Decisions on choice of schools, neighborhood, and peers, for example, may reflect individual characteristics (e.g., personality) that can be partially attributed to genetic factors (Plomin et al.

1977; Scarr and McCartney 1983; Wagner et al. 2013; Conley and Fletcher 2017). Thus, there are multiple pathways by which genetic factors could influence the risk of CJ involvement.

It is crucial to mention that genetic factors in and of themselves do not determine the process of selection into CJ involvement. Rather, genetic factors likely interact with social environments to influence one's risk of criminal behavior and CJ involvement. A large body of research now shows that adverse social environments—such as living in a socioeconomically disadvantaged family or neighborhood—are associated with higher genetic risk, whereas favorable social environments—such as living in a socioeconomically advantaged family or neighborhood—are associated with lower genetic risk (Guo *et al.* 2008; Simons *et al.* 2011; Liu *et al.* 2015; Li *et al.* 2015).

The purpose of considering genetics in research of CJ involvement effects is to enhance our understanding of the complex mechanisms that link CJ involvement with later-in-life socioeconomic inequalities. In so doing, incorporating genetic data in such research can help refine the estimation of socio-environmental influences on inequalities, thereby improving the basis on which socio-environmental interventions might reduce inequalities (Barnes et al. 2014; Liu and Guo 2015; Simons et al. 2011). Put a different way, considering genetic factors may help researchers gain a more thorough understanding of the social factors that—either in isolation or in conjunction with genetic risks—also play a role.

Genetic Correlation between Education and Criminal Behavior

Educational outcomes are complex traits influenced by both social environments and genetic inheritance. Research based on family data shows that genetic factors contribute to, on average, 40% of the variation in educational attainment across different populations (Branigan et al. 2013). Recent socio-genomic studies have identified more than a thousand specific genetic

variants that are significantly associated with educational attainment (Lee *et al*. 2018; Okbay *et al.* 2016b; Rietveld *et al.* 2013a), and researchers have investigated specific mechanisms that explain the genetic associations (Belsky et al. 2016; Domingue et al. 2018; Liu 2018; Rietveld *et al.* 2014). Accordingly, genetic variants may affect education through intermediate individual traits such as cognitive ability, self-control, and interpersonal skills, as well as the selection of educational environments.

What remains unclear, though, is whether criminal behavior and CJ involvement plays a role in explaining the genetic associations with educational outcomes. A recent study by Wertz and colleagues (2018) shows that the polygenic score for educational attainment significantly predicts the risk of criminal offending, suggesting that the relationship between criminal offending and educational outcomes may be more complicated than prior theory has anticipated. This study raises an important question: why is the genetic influence on educational attainment (as measured by an education PGS) predictive of criminal offending? We see at least three possibilities, each of which is shown graphically in Figure 1. First, the association between the education PGS and criminal behavior might be mediated by education (Scenario 1 in Figure 1). Specifically, lower PGSs are associated with poorer educational outcomes (e.g., leaving school with poor qualifications), which in turn, increase the risk of criminal offending.

The second possibility is shown in Scenario 2 in Figure 1. In this scenario, criminal offending that occurs in early life may mediate the genetic association with education. Young offenders with higher genetic risk may become ensnared by the negative consequences of their criminal behaviors, particularly involvement with the CJ system, and are therefore less likely to achieve the highest levels of education.

The third explanation, as shown by Scenario 3 in Figure 1, suggests that some genetic variants may contribute to variation in both criminal offending and education. If the genetic variants associated with the risk of criminal offending are not independent of the genetic variants associated with education[1], then the relationship between criminal offending and education is likely to be confounded by those shared genetic factors. In other words, causal analyses of the crime-education relationship will suffer from omitted variable bias if genetic measures are ignored (Conley et al. 2015; Liu and Guo 2016).

The aim of this study is to assess the mediating effect of CJ involvement during adolescence on genetic associations with educational outcomes. In so doing, we will also explore the extent to which the relationship between CJ involvement and educational outcomes is attributable to genetic confounding. We will, by virtue of our research design, rule out Scenario 1, and focus on Scenarios 2 and 3 as potential explanations for the associations among genetics, CJ involvement, and educational outcomes.

[Figure 1 about here]

CURRENT STUDY

Integrating the various strands of literature outlined above, we propose a genetically informed life-course model to disentangle the complex relationships among genetics, CJ involvement, and educational outcomes (see Figure 2). The Model leads to three hypotheses. *Hypothesis 1* concerns the genetic association with the risk of CJ involvement during adolescence:

[1] As an example, consider that cognitive ability is negatively correlated with offending propensity but it is positively correlated with educational attainment. In this case, genetic influences on offending risk might operate through cognitive ability, meaning that educational attainment and offending outcomes (e.g., CJ involvement) cannot be considered exogenous.

Hypothesis 1: Genetic risk of lower education predicts higher risk of CJ involvement during adolescence.

Following that, we consider factors that explain the genetic association with the risk of adolescent CJ involvement. A wealth of research has demonstrated that criminal behavior and CJ involvement are associated with both individual characteristics such as cognitive ability and delinquency (Hirschi and Hindelang 1977; Thornberry 2018), and a variety of social factors including family influences (Hirschi 1969; Hoeve *et al.* 2009; Sampson and Laub 1997), school experiences (Bernburg and Krohn 2003; Gottfredson 2001; Sampson and Laub 1993), and neighborhood effects (Shaw and McKay 1969; Wikström and Sampson 2003; Kling *et al.* 2005). Most—if not all—of these factors are likely subject to direct or indirect genetic influences. Accordingly, we hypothesize that the genetic association with adolescent CJ involvement may be attributable to those factors.

Hypothesis 2: The genetic association with the risk of adolescent CJ involvement can be attributed to individual and social factors in early life.

We then examine the role of adolescent CJ involvement in the genetic association with education. Research has shown that the influence of CJ involvement on education might occur at different stages of the educational trajectory (Kirk and Sampson 2013). While some individuals who become involved with the CJ system early in life are impeded from completing high school, others might not experience educational disruption until later (e.g., prior to/during tertiary education). We therefore develop three interrelated hypotheses based on three educational outcomes:

Hypothesis 3a: Adolescent CJ involvement mediates the genetic association with overall educational attainment in adulthood.

Hypothesis 3b: Adolescent CJ involvement mediates the genetic association with high school completion in adulthood.

Hypothesis 3c: Adolescent CJ involvement mediates the genetic association with participation in tertiary education in adulthood.

Pertaining to genetic confounding, as Figure 2 shows, adolescent CJ involvement and educational outcomes might be subject to the same genetic influences operating through multiple complex pathways, and such genetic correlation may confound the estimation of the causal effect of CJ involvement on educational outcomes. In our analysis, we will demonstrate how to assess genetic confounding effects using directly observed genetic measures.

[Figure 2 about here]

Before moving to the analysis, there are two additional points that should be considered. First, it is important to acknowledge the severe level of stratification across gender that is traditionally observed for criminal behavior and CJ involvement (Barnes *et al.* 2015; Brame *et al.* 2014; Moffitt 2001; Wilson and Herrnstein 1985). Because of that, we will perform all analyses separately for males and females. This process is in line with recent socio-genetic analyses informed by gender theory (Perry 2016). Second, the PGS used in this study is constructed based on the findings from a GWA study that relied on samples of European descent. It is uncertain whether these findings are replicable in other racial/ethnic populations (Martin *et al.* 2017). To minimize confounding effects of population stratification, the analysis in this study is restricted to non-Hispanic Whites.

DATA

Data for this study are drawn from the National Longitudinal Study of Adolescent to Adult Health (Add Health). Add Health is a longitudinal study of a nationally representative

sample of adolescents in grades 7-12 in the United States during the 1994-95 school year. Add Health participants were drawn from a probability sample of 132 middle and high schools and were representative of American adolescents in grades 7-12 in 1994-1995. Participants have been interviewed in home across four waves (Wave I: 1994-1995; Wave II: 1996; Wave III: 2001-2002; Wave IV: 2008) (Harris *et al.* 2013). At Wave IV, genotype data were collected from participants. After quality control procedures (Highland *et al.* 2018), genotype data were available for 9,974 individuals. The polygenic scores in this study were conducted by the Social Science Genetic Association Consortium (SSGAC) using 609,130 genotyped single-nucleotide polymorphisms (SNPs).

Add Health is well positioned to help us understand causal relationships among genetics, CJ involvement, and education for three reasons. First, Add Health is one of the few nationally representative surveys that include both whole-genome genotype data and a range of CJ involvement variables. Second, Add Health collects longitudinal data from adolescence to adulthood, which facilitates selecting measures with an appropriate temporal order in causal analyses. Moreover, Add Health tracks and interviews participants in jail or prison. This helps to minimize the influence of sample attrition on the results.

To preclude reverse causality, we paid special attention to timing of the key measures. Given that the majority of Add Health participants completed their education after age 18, we focused on CJ involvement that occurred by age 18. We removed participants who were older than 18 at Wave I as they might have already completed their education at that time. In addition, we also removed those whose reported ages of first CJ involvement were younger than their ages at Wave I. By doing so, we minimized the possibility that CJ involvement occurred or education was completed before the covariates (e.g., individual factors and social experiences) were

measured. These procedures resulted in a final analytic sample of 1,697 males and 2,094 females.

VARIABLE MEASUREMENT

Dependent and Independent Variables

Adolescent Criminal Justice Involvement. At Wave IV, Add Health participants were asked whether they have been arrested ("Have you ever been arrested?"), convicted ("Have you ever been convicted or pled guilty to any charges other than a minor traffic violation?"), or incarcerated ("Have you ever spent time in a jail, prison, juvenile detention center or other correctional facility?"). Follow-up questions were used to obtain their age at first arrest ("How old were you the first time you were arrested?"), conviction ("How old were you the first time you were convicted or pled guilty to something?"), and incarceration ("How old were you the first time you went to jail, prison, juvenile detention or other correctional facility?"). Importantly, the Add Health staff conducted interviews with imprisoned participants, asking them to report their age at first arrest/conviction/incarceration.[2] We constructed an "any adolescent CJ involvement" variable, in which participants who reported that they have been arrested/convicted/incarcerated and whose first CJ involvement occurred by age 18 were coded as 1, and 0 otherwise.

Education. We measured overall educational attainment as years of education completed by the time of interview at Wave IV, when participants had reached an average age of 28. All participants were asked, "What is the highest level of education that you have achieved to date?" Response options and their numeric values (in parentheses) included eighth grade or less (8), some high school (10), high school graduate (12), some vocational/technical training (13),

[2] At Wave IV, 73 interviews were conducted in a prison or jail.

completed vocational/technical training (14), some college (14), completed college (16), some graduate school (17), completed a master's degree (18), some post baccalaureate professional education (18), completed post-baccalaureate professional education (19), some graduate training beyond a master's degree (19), and completed a doctoral degree (20). To test *Hypotheses 3b* and *3c*, we created two additional outcome variables: high school completion (1 = completed high school, 0 = otherwise) and tertiary education participation (1 = continued education after high school, 0 = otherwise).

Covariates

We focused on two groups of covariates: individual factors and social experiences. The covariates representing social experiences are further categorized into three groups: family factors, school experiences, and neighborhood characteristics.

Individual Factors. We considered three individual variables: cognitive ability, delinquency, and age. Cognitive ability and delinquency have been shown to be correlated with each other and both contribute to educational outcomes (Farrington 1989; Guay *et al.* 2005; Hirschi and Hindelang 1977; Moffitt and Silva 1988; Sweeten et al. 2009). To measure cognitive ability, we used the Peabody Picture Vocabulary Standardized score (i.e., PVT score) at Wave I (Dunn and Dunn 2007). Self-reported delinquency was measured based on 11 items from the Add Health questionnaire at Wave I: (1) deliberately damaged others' property, (2) so badly hurt someone that medical treatment was needed, (3) used a weapon to get something from someone, (4) took part in group fights, (5) pulled a knife or gun on someone, (6) shot or stabbed someone, (7) took part in fights in which self was injured, (8) stole something worth more than $50, (9) broke into a house or building to steal, (10) sold drugs, and (11) stole something worth less than $5. Responses to the 11 items were summed to create a delinquency score ($\alpha = .79$), with higher

scores indicating higher levels of delinquency. In addition, we included age as a covariate in the analysis as distributions of some key variables may vary by age.

Family Factors. Three family factors were considered: parental education, parental attachment, and parental supervision. At Wave I, both parents and participants were asked about parents' education. The parental education variable was constructed based on parents' report of the highest degree they had completed by the time of interview. If information was available for both parents, the one with higher education was used. If the parent's report was missing, the participant's report was used instead. Response options and their numeric values (in parentheses) included never went to school (0), eighth grade or less (8), more than eighth grade, but did not graduate from high school (10), went to a business, trade, or vocational school instead of high school (10), high school graduate (12), completed a GED (12), went to a business, trade, or vocational school after high school (13), went to college, but did not graduate (14), graduated from a college or university (16), professional training beyond a four-year college or university (18). To assess parental attachment, we used the sum of responses to three Wave I questions asking how close the respondent felt to his or her mother and father and a question concerning the respondent's feelings about how much his or her parents cared about him or her ($\alpha = .72$). A higher score indicated greater parental attachment. Parental supervision score was constructed by summing responses to a Wave I question asking the respondent if his or her parents allowed him or her to make their own decisions about the following seven items: the time they must be home on weekend nights; the people they hang around with; what they wear; how much television they watch; which television programs they watch; what time they go to bed on weeknights; and what they eat. A higher score indicated higher levels of parental supervision.

School Experiences. Three measures at Wave I were used to assess school experiences: school attachment, repeated a grade, and received a suspension from school. To measure school attachment, we summed the responses to three questions (α = .77) asking whether a respondent (rated on a scale of 1 to 5) felt close to people at school, felt like being part of the school, or felt happy at school. In addition, Add Health participants provided information about whether they had ever repeated a grade and whether they had ever received an out-of-school suspension from school (1 = yes; 0 = no).

Neighborhood Characteristics. We assessed the neighborhood environment using three Wave I block group-level variables from the Add Health Public Contextual Database: proportion of aged 25+ individuals with college degree or more, unemployment rate, and proportion of own children under 18 years in families and subfamilies not living with both parents. Block groups are geographic areas defined by the US Bureau of the Census, which in 1990 averaged 452 housing units or 1,100 people. It is the lowest level of geography in sample data published by the Census Bureau, and therefore captures the most localized available contextual characteristics of the areas in which individuals live (Billy *et al.* 1998).

METHOD

Polygenic Scoring

Analyses in this study are based on genome-wide polygenic scores (PGSs). PGSs are calculated using the following equation:

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

where PGS_i is the PGS of individual i, b_j is the coefficient for SNP j estimated in GWA studies, G_{ij} is the number of effect alleles (i.e., the allele positively associated with the outcome) on SNP j that individual i possesses, and J is the total number of SNPs. PGSs in this study were

constructed using b-weights from the most powerful GWA study on education to date (Lee *et al.* 2018). PGSs were normally distributed in Add Health. Larger PGSs are associated with higher levels of educational attainment. To account for potential population stratification, PGSs were residualized on the first ten principal components computed from the genome-wide SNP data (Price *et al.* 2010; Price *et al.* 2006). Residualized PGSs were then standardized to have a mean of 0 (sd = 1) for regression analyses. Table 1 shows the distribution of PGSs for males and females in Add Health.

Hypothesis Testing

To test *Hypothesis 1*, we estimated and tested the association between the education PGS and any adolescent CJ involvement using logistic regression. A significant negative association would suggest that genetic risk of lower education predicts higher risk of CJ involvement during adolescence. We then entered into the logistic regression model the covariates to test *Hypothesis 2*. To test *Hypotheses 3a-3c*, we estimated associations between the PGS and three educational outcomes using regression models and then entered any adolescent CJ involvement into the models to test for mediating effects. The significance of the mediating effects was determined using the Sobel test (Sobel 1982).

We also replicated all the analyses using each of the three CJ involvement measures individually: arrest, conviction, and incarceration. All analyses were adjusted using the sampling weights and standard error estimates were corrected for the clustered design of Add Health.

RESULTS

Distribution of Key Variables

Table 1 displays the distributions of the variables for males and females in the analytic sample. As shown, compared to females, males were more likely to be involved with the CJ

system during adolescence (χ^2 = 237.26, p < .001). Specifically, the likelihood of any adolescent CJ involvement for males was over four times that for females in Add Health. Moreover, males were, on average, older (t = 3.73, p < .001), had higher PVT scores (t = 4.97, p < .001) and delinquency scores (t = 10.23, p < .001), were more likely to repeat a grade (χ^2 = 42.50, p < .001), more likely to receive a suspension (χ^2 = 131.27, p < .001), completed fewer years of education (t = -6.16, p < .001), were less likely to complete high school (χ^2 = 12.31, p < .001), and less likely to participate in tertiary education (χ^2 = 26,64, p < .001). These differences reveal the importance of stratifying the multivariate models by gender. There are no significant gender differences in parental education, parental supervision, school attachment, neighborhood variables, and the education PGS.

[Table 1 about here]

We then compared distributions of the variables for individuals who reported any CJ involvement during adolescence and those who did not. Table 2 shows the results. For both males and females, relative to those who had avoided CJ involvement during adolescence, those who had been involved with the CJ system during adolescence had lower levels of education at Wave IV (i.e., fewer years of schooling, lower high school completion rate, and lower tertiary education participation rate) and lower education PGSs. Also, the two groups significantly differed in delinquency scores and receiving suspension for both males and females. Distributions of PVT scores, parental education, attachment to parents, attachment to school, and repeating a grade significantly differed between the two groups for males but not for females. Additional analyses suggest that lower education PGSs are associated with lower PVT scores, lower parental education, higher delinquency, higher likelihood of repeating a grade, receiving a suspension, and living in neighborhoods with lower levels of education but higher

unemployment and single-parent household rates. Accordingly, the genetic association with the risk of adolescent CJ involvement may operate through multiple individual and social pathways.

[Table 2 about here]

Does the Education PGS Predict CJ Involvement during Adolescence? (Hypothesis 1)

Model 3.1 in Table 3 shows the results of the regression analysis for testing *Hypothesis 1*, which predicts that genetic risk of lower education predicts higher risk of CJ involvement during adolescence. Consistent with this hypothesis, the education PGS is negatively associated with any adolescent CJ involvement for both males and females. Specifically, a one standard deviation increase in the PGS is associated with respectively a .20 decrease in the odds of being arrested/convicted/incarcerated for males during adolescence (odds ratio = $e^{-.229}$, $p < .01$), and a .25 decrease in the odds for females (odds ratio = $e^{-.288}$, $p < .05$).

Is the Genetic Association with Adolescent CJ Involvement Attributable to Individual and Social Factors? (Hypothesis 2)

Models 3.2-3.6 displays the results for testing *Hypothesis 2*. As Model 3.2 shows, after controlling for the individual factors (i.e., age, PVT score, and delinquency score), the effect size of the education PGS on any adolescent CJ involvement risk is reduced by 19% for males and 7% for females. Results of the Sobel test suggest that the PVT score only significantly mediates the genetic association for males ($p < .05$), but not for females ($p > .1$). Delinquency significantly mediates the genetic association with the risk of any adolescent CJ involvement for females ($p < .05$). The mediating effect of delinquency is marginally significant for males ($p < .1$).

When the family variables (i.e., parental education, parental attachment, and parental supervision) are entered into the model, the effect size of the education PGS drops by 16% for

males and 15% for females (see Model 3.3). Results of additional analyses suggest that the reduction in the effect size is mainly attributable to parental education.

Model 3.4 shows the results for testing the mediating effects of school experiences (i.e., school attachment, repeated grade, and received suspension). When the school variables are included in the models, the effect size of the education PGS on any adolescent CJ involvement risk is reduced by 50% for males and 14% for females. These results suggest that school experiences play a key role in the genetic association with the risk of CJ involvement during adolescence. There is also evidence for gender differences in the school mediating effects. For males, the mediating effects of all three school variables are significant (Sobel test $p < .05$ for school attachment, $p < .01$ for repeated grade, and $p < .001$ for received suspension). Yet for females, school experiences are less influential. Among the three variables, only received suspension significantly mediates the genetic association (Sobel test $p < .001$).

Moreover, when the neighborhood variables are entered into the model, the effect size of the education PGS on any adolescent CJ involvement risk is not significantly changed (see Model 3.5). Additional analyses show that none of three neighborhood variables accounts for the genetic association with any adolescent CJ involvement risk for both males and females.

Finally, the effect size of the education PGS on any CJ involvement risk drops by 54% for males and 23% for females after controlling for all the covariates (see Model 3.6). The coefficient of the PGS is no longer significant, suggesting that the genetic association with any adolescent CJ involvement risk can be explained by the individual and social factors considered in the analysis.

[Table 3 about here]

Is the Genetic Association with Overall Educational Attainment Mediated by CJ Involvement During Adolescence? (Hypothesis 3a)

Table 4 displays results for testing *Hypothesis 3a*, namely adolescent CJ involvement mediates the genetic association with overall educational attainment in adulthood. As Model 4.1 shows, the education PGS is significantly associated with years of schooling at Wave IV. Specifically, a one standard deviation increase in the PGS is associated with about a .7 increase in years of schooling for both males and females ($p < .001$). Remarkably, the PGS alone explains about 14% of the observed variation in years of schooling in the analytic sample. The prediction power of the education PGS is comparable to that of cognitive ability and parental education, the most powerful predictors of educational attainment that have been identified so far (Lee *et al.* 2018).

According to results in Model 4.2, any adolescent CJ involvement is significantly associated with lower overall educational attainment. On average, males who had been involved with the CJ system had .9 years less schooling compared to those who had avoided the system during adolescence ($p < .001$); females who had been involved with the CJ system had 1.4 years less schooling compared to those who had avoided the system during adolescence ($p < .001$).

In line with *Hypothesis 3a*, any adolescent CJ involvement significantly mediates the association between the education PGS and overall educational attainment for both males and females (Sobel test $p < .01$ for males and $p < .05$ for females). Put a different way, early involvement with the CJ system appears to interrupt and reduce one's chances for achieving a high education. Also, as Model 4.3 shows, when both the education PGS and any adolescent CJ involvement are entered into the model, the effect size of any adolescent CJ involvement drops by 17% for males and 14% for females. This result suggests that the association between any

adolescent CJ involvement and years of schooling is partially confounded by genetic factors—highlighting the importance of accounting for genetic measures in causal analyses.

Model 4.4 demonstrates the regression results with all the covariates. As shown, after controlling for any adolescent CJ involvement and all the covariates, the genetic association with years of schooling drops by over 50% for both males and females. Accordingly, both individual factors and social experiences play significant roles in explaining the genetic association with overall educational attainment. In addition, the effect size of any adolescent CJ involvement is reduced by 63% for males and 34% for females (but remains statistically significant) after controlling for the education PGS and the covariates.

The results in Table 4 also provide evidence for gender differences in the relationships among delinquency, CJ involvement, and education. While adolescent delinquency and CJ involvement are strong predictors of overall educational attainment for both males and females, magnitudes of their effects on years of schooling are significantly larger for females than males (interaction $p < .05$). In particular, after controlling for the education PGS, any adolescent CJ involvement, and the covariates, delinquency no longer significantly predicts years of schooling among males ($p > .1$), but its effect remains significant for females ($p < .01$) (see Model 4.4). This suggests that whereas the effect of adolescent delinquency on education can be completely attributed to CJ involvement and other variables in the model for males, there are other unobserved pathways through which higher delinquency may result in lower overall educational attainment for females.

[Table 4 about here]

Is the Genetic Association with High School Completion Mediated by Adolescent CJ Involvement? (Hypothesis 3b)

Table 5 displays results for testing *Hypothesis 3b*. As Model 5.1 shows, a one standard deviation increase in the PGS is associated with respectively a 1.083 (odds ratio = $e^{.734}$, $p < .001$) and a .984 (odds ratio = $e^{.685}$, $p < .001$) increase in the odds of completing high school for males and females. For males who have been involved with the CJ system during adolescence, the odds of high school completion is reduced by .67 (odds ratio = $e^{-1.095}$, $p < .001$), and the reduction in the odds is .66 for females (odds ratio = $e^{-1.075}$, $p < .01$) (see Model 5.2). Consistent with *Hypothesis 3b*, any adolescent CJ involvement significantly mediates the association between the education PGS and high school completion for males (Sobel test $p < .05$). The mediating effect is marginally significant for females (Sobel test $p < .1$). In addition, when both the education PGS and any adolescent CJ involvement are included in the model, the coefficient of any adolescent CJ involvement is reduced by 8% for males and 9% for females (see Model 5.3). Model 5.4 shows that after controlling for all the covariates, the genetic association with high school completion drops by around 50%, and the coefficient of any adolescent CJ involvement is reduced by 43% for males and 47% for females (but remains statistically significant).

[Table 5 about here]

Is the Genetic Association with Tertiary Education Participation Mediated by Adolescent CJ Involvement? (Hypothesis 3c)

Table 6 displays results for testing *Hypothesis 3c*. As shown by Model 6.1, a one standard deviation increase in the PGS is associated with respectively 1.069 (odds ratio = $e^{.727}$, $p < .001$) and .966 (odds ratio = $e^{.676}$, $p < .001$) increase in the odds of participating in tertiary education for males and females. For males who had been involved with the CJ system during adolescence, the odds of tertiary education participation is reduced by .53 (odds ratio = $e^{-.765}$, $p < .001$), and for females, the reduction is .64 (odds ratio = $e^{-1.021}$, $p < .001$) (see Model 6.2). Consistent with

Hypothesis 3c, any adolescent CJ involvement significantly mediates the association between the education PGS and tertiary education participation for both males and females (Sobel test $p < .01$ for males and $p < .05$ for females). Also, when both the education PGS and any adolescent CJ involvement are included in the model, the effect size of any adolescent CJ involvement drops by 11% for males and 8% for females (see Model 6.3). Model 6.4 shows that when the covariates are entered into the model, the genetic association with tertiary education participation is reduced by around 50%, and the coefficient of any adolescent CJ involvement drops by 51% for males and 13% for females (but remains statistically significant). Adolescent CJ involvement has a larger effect on tertiary education participation for females than males after taking into account the confounding variables in the analysis (interaction $p < .05$).

[Table 6 about here]

DISCUSSION AND CONCLUSIONS

This study makes several important contributions to the sociological literature. First, we develop a theoretical model by integrating life-course theories and recent socio-genomic research to understand the complex relationships among genetic inheritance, CJ involvement, and educational outcomes (as elaborated in Figure 2). We test the model by leveraging the longitudinal design and genome-wide data from the Add Health study. We find that polygenic scores for education are significantly associated with the risk of CJ involvement during adolescence. Further, we show that this association is explained away by individual factors including delinquency and cognitive ability, and social factors including family socioeconomic status (e.g., parental education) and school experiences (e.g., attachment to school, ever repeated a grade, ever received a suspension from school). In particular, the school variables are the most important explanatory factors of the genetic association with the risk of adolescent CJ

involvement. Accordingly, it may be possible to suppress the genetic risk of criminal behavior and CJ involvement by enhancing children's experiences at school.

In addition, we provide evidence that involvement with the CJ system during adolescence mediates genetic associations with three educational measures: overall educational attainment, high school completion, and participation in tertiary education. This finding adds an important explanation to the genetic association with education (Belsky *et al.* 2016; Domingue *et al.* 2018; Lee *et al.* 2018; Liu 2018; Okbay *et al.* 2016b; Rietveld *et al.* 2013a). Also, it supports the prior literature that suggests that CJ involvement, especially incarceration, may block educational pathways, thereby reducing the realized educational outcomes of those who have contact with the CJ system (Kirk and Sampson 2013).

Secondly, we find evidence for gender differences in relationships among genetics, adolescent delinquency and CJ involvement, and educational outcomes. Our analysis shows that although adolescent delinquency and CJ involvement are much less frequent among females than males, their effects on educational outcomes—particularly tertiary education participation and overall educational attainment— are significantly *greater* compared to males. Moreover, our results suggest that the effect of adolescent delinquency on education operates mainly through CJ involvement for males, but not for females. In other words, for males, the association between higher levels of delinquency and reduced education is most likely the result of delinquency leading to involvement with the CJ system. In contrast, the delinquency-education association may operate through other pathways for females. Research suggests that relative to men, women's early involvement with delinquency and the CJ system is more likely to result in weakening pro-social interpersonal ties, lack of psychological adjustment, and isolation from social support systems (Lanctôt *et al.* 2007). All of these mechanisms may lead to detrimental

consequences in later life. These findings complicate the life-course model in Figure 2. More analyses can be conducted to understand the gender differences in future research.

Thirdly, selection bias is a long-standing issue in causal analyses with observational data. Statistical strategies have been developed to address selection bias, yet such strategies mostly suffer from "hidden bias" due to unobserved variables (Rubin 1978). Genetic factors are often theorized as an important source of the "hidden bias"(Pearl 2009; VanderWeele 2015). Recently, advancements in genomic science and technology have produced novel data and methods to account for genetic confounding. In this study, we demonstrate how PGSs can be used to assess genetic confounding in regression analyses. We find around one-sixth of the association between any CJ involvement during adolescence and overall educational attainment is explained away by the education PGS.

As the association between the education PGS and adolescent CJ involvement is shown to be mediated by the individual and social covariates in this study, one might expect that controlling for the covariates in estimating the effect of CJ involvement would be sufficient to address genetic confounding, and therefore it is unnecessary to use genetic measures. Yet we maintain our support for using genetic measures for several reasons. Most importantly, not all the mediating mechanisms of the genetic associations between CJ involvement and later life outcomes are well understood. For some complex outcomes, researchers are unlikely to obtain a comprehensive list of all mediating variables that account for the genetic associations. The results might still be biased if some important mediating variables are ignored. In contrast, results based on genetic measures are more robust to unobserved heterogeneity. Also, unlike the covariates, genotypes are exogenous in nature, and the results based on genetic measures are less susceptible to endogeneity and further concerns such as collider bias (Elwert and Winship 2014).

Moreover, genotypes are typically more accurately measured than other covariates and therefore suffer less from consequences of measurement error (Westfall and Yarkoni 2016).

This study can be extended in several ways. First, additional information is needed to achieve a better understanding of the collateral consequences of CJ involvement such as frequency (i.e., how often the individual was involved?) and type (i.e., why the individual was involved?) of CJ involvement. Due to reductions in sample size, we were unable to conduct analyses that would allow us to answer these types of questions with the Add Health data. Also, although genetic measures are available for some minorities (e.g., Blacks and Hispanics), their sample sizes are insufficient to achieve adequate statistical power for separate analyses. We hope more extensive analyses can be conducted in future studies when more data become available. Moreover, the conceptual model and empirical analyses in this study can be extended to other later life outcomes (e.g., occupational status, income, wealth) and intergenerationally (e.g., the influence of parents' involvement with the CJ system on their children's socioeconomic and health outcomes).

To summarize, this study demonstrates how sociology and socio-genomic research can be integrated to improve our understanding of the social world. On the one hand, socio-genomic research provides novel insights to enrich theoretical models and improve empirical analyses in sociological studies. On the other hand, sociology provides theoretical guidance to disentangle complex causal relationships between biological and social variables. Genomic data are increasingly available in large-scale social science datasets (e.g., the Fragile Families Study, Health and Retirement Study, and the Wisconsin Longitudinal Study). These resources provide us with unprecedented opportunities to advance scientific knowledge and innovation.

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Table 1. Univariate Distributions of Key Variables

	Males Mean/% (SD)	Females Mean/% (SD)
Key Dependent and Independent Variables		
Any Adolescent Criminal Justice Involvement	.138	.033
Arrest	.130	.032
Conviction	.053	.008
Incarceration	.057	.013
Years of Schooling (Wave IV)	13.966(1.967)	14.357(1.913)
High School Completion (Wave IV)	.913	.943
Tertiary Education Participation (Wave IV)	.738	.809
Education Polygenic Score	.044(1.010)	-.009(.999)
Covariates		
Individual Factors		
Age (Wave IV)	28.351(1.315)	28.188(1.347)
PVT Score (Wave I)	106.085(11.989)	104.143(11.949)
Delinquency (Wave I)	1.938(3.346)	.988(2.058)
Family Factors		
Parental Education (Wave I)	14.196(2.347)	14.117(2.394)
Parental Attachment (Wave I)	14.761(1.975)	14.347(2.299)
Parental Supervision (Wave I)	2.050(1.508)	1.975(1.479)
School Experiences		
School Attachment (Wave I)	8.349(2.521)	8.441(2.640)
Repeated Grade (Wave I)	.189	.113
Received Suspension (Wave I)	.266	.120
Neighborhood Characteristics		
Proportion of Aged 25+ with College Degree or More (Wave I)	.237(.148)	.237(.150)
Proportion of Unemployed (Wave I)	.062(.046)	.063(.046)
Proportion of Single Parent Household (Wave I)	.188(.134)	.187(.131)
N	1,697	2,094

Note: Education PGSs are residualized on the first ten principal components and standardized in the whole sample.

Table 2. Distributions of Key Variables of Individuals Who Had Any Criminal Justice Involvement during Adolescence and Others

	Males		Females	
	Any Adolescent CJ Involvement Mean/% (SD)	No Adolescent CJ Involvement Mean/% (SD)	Any Adolescent CJ Involvement Mean/% (SD)	No Adolescent CJ Involvement Mean/% (SD)
Key Dependent and Independent Variables				
Years of Schooling (Wave IV)	13.141***	14.098***	13.029***	14.403***
	(2.078)	(1.917)	(1.769)	(1.902)
High School Graduation (Wave IV)	.812***	.929***	.857**	.946**
Tertiary Education Participation (Wave IV)	.594***	.761***	.614***	.815***
Education Polygenic Score	-.156***	.076***	-.283**	.000**
	(.977)	(1.012)	(.789)	(1.004)
Covariates				
Age (Wave IV)	28.081***	28.394***	27.786**	28.202**
	(1.256)	(1.319)	(1.284)	(1.347)
PVT Score (Wave I)	103.816***	106.448***	103.029	104.181
	(11.091)	(12.091)	(11.041)	(11.980)
Delinquency (Wave I)	3.756***	1.647***	2.414**	.939**
	(4.836)	(2.941)	(3.622)	(1.965)
Parental Education (Wave I)	13.799**	14.259**	13.571	14.136
	(2.186)	(2.366)	(2.429)	(2.391)
Parental Attachment (Wave I)	14.500*	14.802*	14.214	14.352
	(2.107)	(1.950)	(1.918)	(2.312)
Parental Supervision (Wave I)	1.996	2.058	1.943	1.976
	(1.472)	(1.514)	(1.328)	(1.484)
School Attachment (Wave I)	7.679***	8.456***	8.014	8.456
	(2.557)	(2.500)	(3.048)	(2.624)
Repeated Grade (Wave I)	.278***	.174***	.086	.114
Received Suspension (Wave I)	.513***	.226***	.300***	.114***
Neigh Proportion of Aged 25+ with College Degree or More (Wave I)	.221	.239	.239	.237
	(.139)	(.150)	(.164)	(.149)
Neigh Proportion of Unemployed (Wave I)	.067	.062	.064	.063
	(.049)	(.045)	(.045)	(.046)
Neigh Proportion of Single Parent Household (Wave I)	.204	.186	.197	.187
	(.139)	(.133)	(.128)	(.132)
N	234	1,463	70	2,024

Note: Analyses use t tests for continuous variables and chi-square tests for dichotomous variables to compare those who had any CJ involvement and those who did not during adolescence. All tests were conducted for males and females separately.
*p< .05; **p< .01; ***p< .001 (two-tailed tests).

Table 3. Coefficients (Standard Error) in Logistic Regression Models Predicting Any Criminal Justice Involvement during Adolescence (*Hypotheses 1* and *2*)

	Model 3.1		Model 3.2		Model 3.3		Model 3.4		Model 3.5		Model 3.6	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
Education Polygenic Score	-.229**	-.288*	-.186*	-.268*	-.192**	-.244†	-.114	-.249*	-.209**	-.300*	-.106	-.221
	(.071)	(.123)	(.077)	(.130)	(.074)	(.130)	(.075)	(.126)	(.072)	(.126)	(.081)	(.136)
Covariates												
Individual Factors												
Age			-.188**	-.278**							-.284***	-.344***
			(.057)	(.094)							(.064)	(.104)
PVT Score			-.011†	.002							-.007	.004
			(.006)	(.011)							(.007)	(.012)
Delinquency			.133***	.178***							.088***	.149***
			(.018)	(.034)							(.019)	(.038)
Family Factors												
Parental Education					-.062†	-.072					-.004	-.081
					(.032)	(.055)					(.038)	(.063)
Parental Attachment					-.072*	-.020					-.025	.022
					(.032)	(.050)					(.036)	(.058)
Parental Supervision					-.038	-.037					-.074	-.099
					(.048)	(.084)					(.053)	(.092)
School Experiences												
School Attachment							-.061*	-.024			-.041	-.017
							(.028)	(.044)			(.030)	(.046)
Repeated Grade							.171	-.829†			.210	-.844†
							(.177)	(.452)			(.189)	(.479)
Received Suspension							1.129***	1.211***			.990***	1.089***
							(.154)	(.289)			(.166)	(.315)
Neighborhood Characteristics												
Proportion of Aged 25+ with College Degree or More									-.379	.532	-.214	.998
									(.566)	(.925)	(.630)	(1.019)
Proportion of Unemployed									-.027	-.627	-1.187	-.102
									(1.787)	(3.154)	(1.943)	(3.191)
Proportion of Single Parent Household									.722	.635	.201	.334
									(.576)	(1.003)	(.623)	(1.042)
Constant	-1.955***	-3.365***	1.809	.397	.038	-1.999†	-1.901***	-3.318***	-2.003***	-3.583***	3.524*	1.951
	(.107)	(.193)	(1.128)	(1.796)	(.668)	(1.049)	(.270)	(.432)	(.234)	(.426)	(1.541)	(2.314)
Log Likelihood	-674.333	-303.929	-638.042	-289.800	-669.836	-302.871	-636.813	-294.707	-672.764	-303.595	-612.838	-281.116

N	1,697	2,094	1,697	2,094	1,697	2,094	1,697	2,094	1,697	2,094	1,697	2,094

Note: All analyses were adjusted using the sampling weights and standard error estimates were corrected for the clustered design of Add Health.
$^{\dagger}p< .05$; $^{\dagger\dagger}p< .01$; $^{\dagger\dagger\dagger}p< .001$ (one-tailed tests).
$^{*}p< .05$; $^{**}p< .01$; $^{***}p< .001$ (two-tailed tests).

Table 4. Coefficients (Standard Error) in Linear Regression Models Predicting Overall Educational Attainment at Wave IV (*Hypothesis 3a*)

	Model 4.1		Model 4.2		Model 4.3		Model 4.4	
	Male	Female	Male	Female	Male	Female	Male	Female
Education Polygenic Score	.716***	.710***			.695***	.700***	.317***	.328***
	(.041)	(.038)			(.042)	(.038)	(.040)	(.036)
Any Adolescent Criminal Justice Involvement			-.946***	-1.377***	-.785***	-1.179***	-.351**	-.902***
			(.144)	(.214)	(.139)	(.206)	(.128)	(.191)
Covariates								
Age							.093**	.049†
							(.032)	(.027)
PVT Score							.023***	.021***
							(.004)	(.003)
Delinquency							.015	-.052**
							(.012)	(.019)
Parental Education							.188***	.228***
							(.020)	(.016)
Parental Attachment							.042*	.058***
							(.021)	(.016)
Parental Supervision							-.033	-.016
							(.028)	(.024)
School Attachment							.042*	.043**
							(.017)	(.013)
Repeated Grade							-.734***	-.929***
							(.112)	(.117)
Received Suspension							-.831***	-.693***
							(.103)	(.119)
Neigh Proportion of Aged 25+ with College Degree or More							1.269***	1.281***
							(.291)	(.244)
Neigh Proportion of Unemployed							.095	.166
							(1.048)	(.855)
Neigh Proportion of Single Parent Household							-.087	-.408
							(.327)	(.292)
Constant	14.068***	14.460***	14.223***	14.510***	14.166***	14.501***	5.430***	6.559***
	(.069)	(.061)	(.074)	(.066)	(.070)	(.061)	(1.157)	(.917)
Adjusted R^2	.138	.139	.030	.018	.156	.151	.371	.402
N	1,697	2,094	1,697	2,094	1,697	2,094	1,697	2,094

Note: All analyses were adjusted using the sampling weights and standard error estimates were corrected for the clustered design of Add Health.
†p< .05; ††p< .01; †††p< .001 (one-tailed tests).
*p< .05; **p< .01; ***p< .001 (two-tailed tests).

Table 5. Coefficients (Standard Error) in Logistic Regression Models Predicting High School Completion at Wave IV (*Hypothesis 3b*)

	Model 5.1		Model 5.2		Model 5.3		Model 5.4	
	Male	Female	Male	Female	Male	Female	Male	Female
Education Polygenic Score	.734***	.685***			.716***	.683***	.337**	.318**
	(.092)	(.100)			(.094)	(.100)	(.107)	(.117)
Any Adolescent Criminal Justice Involvement			-1.095***	-1.075**	-1.003***	-.981**	-.622**	-.565
			(.196)	(.356)	(.203)	(.362)	(.234)	(.435)
Covariates								
Age							.254**	.241**
							(.085)	(.091)
PVT Score							.047***	.040***
							(.010)	(.010)
Delinquency							.050†	-.074*
							(.026)	(.037)
Parental Education							.187***	.274***
							(.053)	(.059)
Parental Attachment							.008	.091*
							(.046)	(.041)
Parental Supervision							.046	.066
							(.066)	(.074)
School Attachment							.080*	.053
							(.038)	(.037)
Repeated Grade							-.986***	-1.147***
							(.212)	(.240)
Received Suspension							-1.182***	-1.205***
							(.215)	(.245)
Neigh Proportion of Aged 25+ with College Degree or More							1.816†	2.790*
							(1.040)	(1.191)
Neigh Proportion of Unemployed							2.320	1.438
							(2.323)	(2.363)
Neigh Proportion of Single Parent Household							-.885	-.903
							(.765)	(.803)
Constant	2.725***	3.231***	2.752***	3.090***	2.908***	3.287***	-12.254***	-12.747***
	(.142)	(.163)	(.142)	(.152)	(.152)	(.166)	(3.001)	(3.069)
Log Likelihood	-466.070	-432.440	-486.508	-453.871	-455.011	-429.415	-368.048	-331.633
N	1,697	2,094	1,697	2,094	1,697	2,094	1,697	2,094

Note: All analyses were adjusted using the sampling weights and standard error estimates were corrected for the clustered design of Add Health.
†p< .05; ††p< .01; †††p< .001 (one-tailed tests).
*p< .05; **p< .01; ***p< .001 (two-tailed tests).

Table 6. Coefficients (Standard Error) in Logistic Regression Models Predicting Tertiary Education Participation at Wave IV (*Hypothesis 3c*)

	Model 6.1		Model 6.2		Model 6.3		Model 6.4	
	Male	Female	Male	Female	Male	Female	Male	Female
Education Polygenic Score	.727***	.676***			.717***	.673***	.374***	.376***
	(.063)	(.062)			(.064)	(.063)	(.073)	(.071)
Any Adolescent Criminal Justice Involvement			-.765***	-1.021***	-.684***	-.935***	-.378*	-.890**
			(.147)	(.252)	(.155)	(.260)	(.180)	(.295)
Covariates								
Age							.116*	-.002
							(.055)	(.052)
PVT Score							.035***	.032***
							(.006)	(.006)
Delinquency							.046*	-.050†
							(.021)	(.028)
Parental Education							.261***	.257***
							(.035)	(.034)
Parental Attachment							.058†	.042
							(.033)	(.027)
Parental Supervision							-.088*	-.0004
							(.045)	(.045)
School Attachment							.034	.027
							(.027)	(.024)
Repeated Grade							-.754***	-.816***
							(.158)	(.169)
Received Suspension							-.928***	-.503**
							(.149)	(.174)
Neigh Proportion of Aged 25+ with College Degree or More							1.663**	2.141***
							(.633)	(.646)
Neigh Proportion of Unemployed							-.736	-.171
							(1.640)	(1.513)
Neigh Proportion of Single Parent Household							.345	-.382
							(.542)	(.508)
Constant	1.311***	1.635***	1.329***	1.552***	1.410***	1.678***	-10.156***	-5.894***
	(.091)	(.093)	(.089)	(.088)	(.095)	(.095)	(1.958)	(1.749)
Log Likelihood	-896.082	-956.498	-959.234	-1,014.786	-886.587	-950.538	-729.658	-811.322
N	1,697	2,094	1,697	2,094	1,697	2,094	1,697	2,094

Note: All analyses were adjusted using the sampling weights and standard error estimates were corrected for the clustered design of Add Health.
†p< .05; ††p< .01; †††p< .001 (one-tailed tests).
*p< .05; **p< .01; ***p< .001 (two-tailed tests).

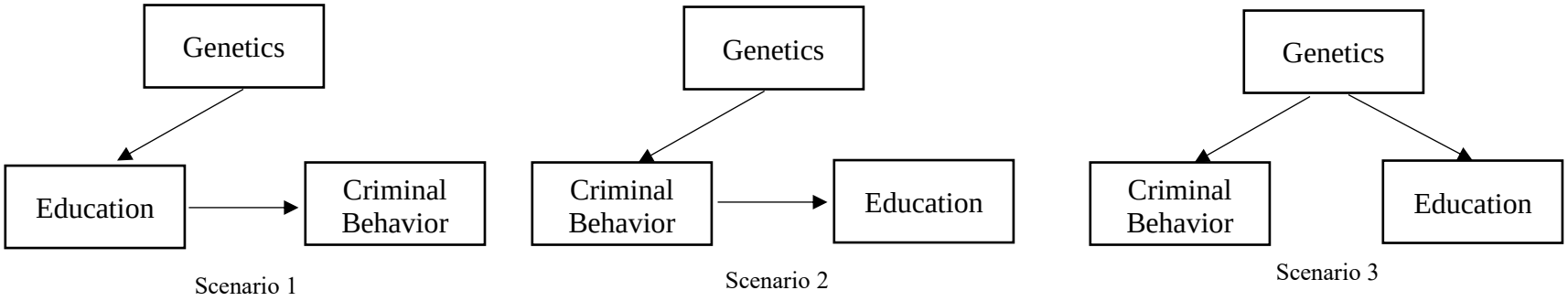

Figure 1. Genetic Correlation between Criminal Behavior and Education

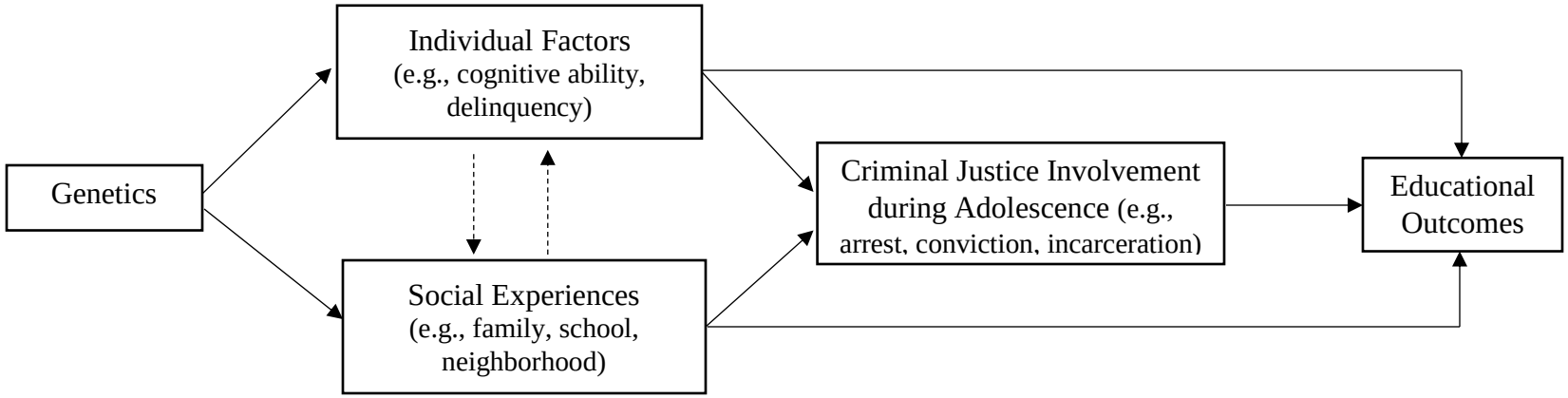

Figure 2: A Life-course Model of the Relationships among Genetics, Criminal Justice Involvement, and Education