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A GENETICALLY-INFORMED STUDY OF THE PREDICTORS AND THE DEVELOPMENT OF DELINQUENCY

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A GENETICALLY-INFORMED STUDY OF THE PREDICTORS AND THE
DEVELOPMENT OF DELINQUENCY

DISSERTATION

A dissertation submitted in partial fulfillment of the
requirements for the degree of Doctor of Philosophy in the
College of Agriculture, Food and Environment
at the University of Kentucky

By

Albert J. Ksinan

Lexington, Kentucky

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Lexington, Kentucky

2018

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ABSTRACT OF DISSERTATION

A GENETICALLY-INFORMED STUDY OF THE PREDICTORS AND THE DEVELOPMENT OF DELINQUENCY

Although the rates of delinquent behavior have been decreasing since the 1990s, adolescent delinquent behavior continues to take a great toll on society as well as on perpetrators themselves. In this way, it is essential to understand the process of delinquency development. The current dissertation is comprised of three studies that analyzed the predictors and the development of delinquency using genetically-informed designs. The sample used for all studies comes from the Add Health dataset, a nationally-representative data on adolescents followed across 14 years.

The first study modeled the longitudinal development of delinquency in three adolescent cohorts: early, middle, and late adolescence. The results showed significant heritability effects on delinquency, with varying estimates across cohorts. The longitudinal stability of delinquency was mostly driven by heritability, while changes were affected by nonshared environmental influences.

The second study tested the GxE interaction between two dopaminergic polymorphisms (DRD4 7-repeat allele and DRD2 A1 allele) and parenting, operationalized by child abuse on the one negative extreme and maternal closeness on the other, in longitudinally predicting delinquent behaviors. Main effects of maternal closeness and childhood abuse on later delinquency were found. On the other hand, no significant interaction of DRD2 or DRD4 polymorphisms with either maternal closeness or childhood abuse were observed.

The third study used a twin design to test whether neighborhood disadvantage has a genetic component and whether this might be explained by an individual's IQ and self-control. The results showed substantial heritability of the neighborhoods the individuals moved into as adults. This was partly explained by IQ, as adolescents' IQ predicted neighborhood disadvantage 14 years later.

KEYWORDS: delinquency, adolescence, behavior genetics, parenting, neighborhood, twins

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INTRODUCTION

Delinquent behavior among youth remains a serious social problem today. Rates of different delinquent behaviors spike during the adolescent years. These changes have been observed repeatedly, suggesting that intervention efforts trying to mitigate this issue have been largely unsuccessful. Despite the ubiquity of this problem, its existence is still deeply troubling. The most obvious issue is the detrimental effects on the society as adolescents commit a disproportionate amount of crime. In 2010, 11% of male arrests and 14% of female arrests involved a person younger than 18 years, despite the fact that youth comprise only 6% of the population (Johnson, Simons, & Conger, 2004; Snyder & Mulako-Wangota, 2014). It has been estimated that one specific cohort of 503 young males, ages 7-17 years, caused a total harm and cost to society ranging from \$89-\$110 million (Welsh et al., 2008).

In addition to tremendous costs to society, teenage delinquency negatively affects adolescents themselves through involvement with the juvenile justice system. In 2015, law enforcement made 921,600 arrests of persons under the age of 18 (Snyder & Sickmund, 2005). The juvenile justice system serves as an immediate response to and repression of antisocial behavior, but this entails substantial negative repercussions for the future. Youth involved in the juvenile justice system are less likely to graduate from high school (Hjalmarsson, 2008; Sweeten, 2006), or enroll in college (Kirk & Sampson, 2013). Most importantly, many youth who are involved in the juvenile justice system are then more likely to have higher rates of criminal behaviors as adults (Bernburg & Krohn, 2003; Dodge, Dishion, & Lansford, 2006; Gatti, Tremblay, & Vitaro, 2009). In this way, adolescent delinquency and the existing corrective systems are detrimental to the society

as well as to the individuals involved. Given that the highest levels of delinquency appear in adolescent years as well as its strong association with subsequent crime, understanding predictors of and the developmental changes in adolescent delinquency is paramount for improving the current juvenile justice system as well as for society as a whole.

Criminology, its origins dating back to the second half of the 19th century, is concerned with studying predictors, development, and associated outcomes of delinquent and criminal behaviors. As is the case with many if not all social sciences, different approaches for explaining criminal behaviors emerged, with an individualistic perspective on one end emphasizing innateness of human traits, including propensity to crime, and an environmental perspective, considering the source of criminal behaviors as a consequence of external (social) factors. These two perspectives are of course rather crude generalizations, which serve to illustrate the extremes of the nature-nurture dichotomy explaining human behaviors.

Criminological perspectives have been for a long time dominated by a position that emphasized the role of environmental factors in explaining criminal behaviors. Historically, however, the individual differences perspective has been prominent in criminological thought and research during its beginnings. In the second half of the 19th century, criminology and other social sciences were heavily influenced by a biological paradigm, explaining psychological phenomena by pointing to biological processes and characteristics of an individual. One of the most influential methods for explaining human behavior during this era was phrenology. This method was based on the following logic: the brain was the organ of the mind – the brain comprised different faculties responsible for all human traits – the more these faculties were active the bigger their size

– the size of these faculties can be inferred from a shape of the skull. The prevalent use of phrenology was to assess and predict psychopathology including criminal behaviors, and it was in criminology where the phrenological method was essential for determining would-be criminals by measuring their skull size, for instance (Rafter, 2004b, 2005).

Related to this, another important influence during this era was the theory of evolution, which emphasized that living organisms evolved historically through a process of natural selection, and knowledge about genetics and heredity, first proposed by Gregor Mendel. The findings from genetics and theory of evolution led to the development of eugenics, or a method of ‘improving’ human genetic quality. It was believed that human traits were purely biological and fully heritable in nature, including all psychological traits and behaviors. Within criminology, the influence of both phrenology and eugenics led to a biological determinism, according to which some humans are irredeemably born as criminals or delinquents (Rafter, 2004a). Based on these foundations, the proposed and executed interventions aimed at people carrying such traits included forced sterilization, imprisonment, and, in the case of Nazi Germany, even state-sponsored systematic genocide.

Of course, this was not only unethical and inhumane, but also scientifically unsound. As they could not withstand the scientific scrutiny, eugenics and phrenology began to be perceived as pseudo-science with dangerous implications. With it, though, the individualistic perspective in explaining criminal behavior fell out of favor, as the sociogenic perspective began to dominate. It was not until the 1980s when the individualistic perspective began to challenge the existing paradigm in criminology. Two theoretical frameworks were essential for this development: the general theory of crime

and new findings based on the behavior genetic method. The general theory of crime was proposed by Gottfredson and Hirschi in their 1990's seminal book. They considered the individual trait of self-control or the ability to restrain impulses and delay gratification as a key variable important in understanding individual deviant or criminal acts. Although these authors acknowledge that crime cannot happen without opportunities, they see self-control, or more precisely, low self-control, as a key cause of crime. This theory has had a tremendous impact on criminology as many studies found empirical support for these assertions (Grasmick, Tittle, Bursik, & Arneklev, 1993; Pratt & Cullen, 2000; Vazsonyi, Mikuška, & Kelley, 2017).

The next important influence on criminological thinking came from research applying the behavior genetic method. Employing samples of siblings with different degree of relatedness (i.e., twins, non-twins, adopted siblings), this research design is able to disentangle the genetic and environmental effects on variance in any measurable trait. The past three decades of behavior genetics research has shown that differences among individuals in all psychological traits are, to a significant extent, heritable, in addition to environmental in origin (Plomin, DeFries, Knopik, & Neiderhiser, 2016; Turkheimer, 2000). The emergence of these individual difference perspectives made the then-existing sociogenic position of criminology, seeing crime as stemming from environmental influences and, in effect, humans as blank slates, untenable (Pinker, 2003).

This of course does not mean that the research from the sociogenic perspective was wrong or that the pendulum of paradigm change is going to swing to a view dominated by individual traits. Rather, a more nuanced approach is needed, one that takes into account that human psychological propensities are largely heritable but that the way

they manifest themselves is enhanced or inhibited based on the environments they find themselves in, a position consistent with both behavior genetics as well as general theory of crime.

The philosophical framework of the present dissertation stems from this ‘modified’ individualistic perspective on the origins and development of delinquent and criminal behaviors. It acknowledges that both genes and the environment play an important role in determining behaviors, a fact that is oftentimes ignored in common social science research design, which disregards genetic links that make individuals in the family similar to each other (Barnes, Boutwell, Beaver, Gibson, & Wright, 2014). Through three interrelated studies, this dissertation focuses on assessing different aspects of the environment along with individual propensities in relation to delinquent behaviors and its correlates. From an ecological framework, the three studies reflect three different developmental contexts of delinquent behaviors: individual, family, and neighborhood. All three studies employ the Add Health, a nationally representative dataset of adolescents and young adults, collected over a 14-year period across four waves. Each study focuses on the period of adolescence as the crucial time for development of problem behaviors and delinquency with lifelong consequences, but Study 2 and 3 also take into account additional assessments (Waves 3 and 4), when most of the individuals were in their twenties, to provide a developmental treatment and study spanning two decades of life.

The first study traced the longitudinal of delinquent behaviors in adolescence one year apart, from Wave 1 to Wave 2. Given that the age of the study youth varied at each timepoint, the development of delinquency was estimated in three cohorts: early, middle,

and late adolescents. More importantly, this study employed the twin design to estimate both genetic and environmental effects on the level of as well as on the rate of change in delinquency.

The second study focused on the effect of parenting behaviors on adolescent delinquency. Parenting has traditionally been emphasized as a key predictor variable in determining problem behaviors and delinquency among children. However, the known heritability component in delinquent behaviors suggests that genes might also play into this link. Merging these two perspectives, the past decade has seen a number of studies that sought to test the interaction of genes x parenting in understanding predicting problem behaviors and delinquency. Following this, the second study tested the effects of two dopaminergic polymorphisms (DRD2 and DRD4) on subsequent delinquent behaviors and, most importantly, test for their potential interactive effects with different parental behaviors, ranging from severe maltreatment to supportive parenting.

Finally, the third study did not focus on delinquency as an outcome variable, but instead, focused on neighborhood disadvantage. According to a classic sociogenic and criminological view as well as evidence, neighborhoods with high concentrations of disadvantage are conducive to higher levels of crime and delinquency because these structural characteristics weaken informal social control, known to be essential for regulating antisocial behaviors in neighborhood (Morenoff, Sampson, & Raudenbush, 2001; Sampson, 1997). In this view, neighborhood disadvantage is expected to predict higher levels of delinquent behavior as well as plethora of other negative outcomes, particularly among adolescents (Brooks-Gunn, Duncan, Klebanov, & Sealand, 1993; Leventhal & Brooks-Gunn, 2000). Based on the process of self-selection, this study

sought to flip the presumed causality by asking whether individual differences in any way predispose individuals to reside in particular neighborhoods, and to what extent is there a genetic basis for this hypothesized self-selection.

STUDY I: A GENETICALLY-INFORMED STUDY OF DELINQUENCY
DEVELOPMENT IN ADOLESCENCE

Abstract

The rates of delinquent behavior increase substantially as individuals move into adolescence, only to be followed by a similarly sharp decline in adulthood. Traditionally, the increase in delinquent behavior has been explained from a sociogenic perspective, emphasizing the role of family, school, or peers in this development. However, the past two decades of research in behavior genetics showed that a substantial portion of variance in delinquency is genetic in origin. However, there exists a scarcity of research that considers both genetic and environmental effects in a longitudinal design. The current study uses a genetically-informed design to model longitudinal development of delinquency in three adolescent cohorts: early, middle, and late adolescence. Employing Wave 1 and Wave 2 of the Add Health dataset, a total of $N = 1,038$ same-sex sibling pairs provided data. Delinquent behaviors were assessed by a self-report measure. The results showed significant effects of heritability on delinquency, with varying estimates based on cohorts. While heritability of delinquency in early and late adolescents was $h^2 \sim .40$, it was much lower ($h^2 = .23$) in middle adolescence, when delinquency was at its peak. The effect of the shared environment was modest and mostly related to early adolescence. The longitudinal stability of delinquency was mostly driven by heritability, while the change was affected by nonshared environmental influences.

Introduction

Criminology, originating in the second half of the 19th century, has been focused on studying the predictors, the development, and associated outcomes of deviant, antisocial, and criminal behaviors. One of the most robust findings in this work has been the age-graded developmental patterns of rates of deviance and crime. Namely, its rate increases sharply when individuals enter adolescence, peaks in mid-adolescence, and then drops just as swiftly in the early twenties. The relative ubiquity of this finding, appearing across nations, generations, or different types of criminal behavior suggests that a process universal to human behavior might be taking place. Some authors have suggested that this development might be affected by biological factors, thus emphasizing that the rates of deviance and crime are contingent on individual differences. More and more evidence supporting this standpoint has emerged over the past three decades. This is especially so based on insights from behavior genetic studies. These have shown that differences in any individual trait, including delinquency, are to at least some extent heritable, i.e., affected by genes (Turkheimer, 2000). At the same time, this work has also shown that large portion of variance in these traits is environmental in nature (Johnson, Turkheimer, Gottesman, & Bouchard, 2010). Furthermore, when genes and the environment interact, this source of variance in the behavior genetic designs (see below for more information) is included in the heritability coefficient, confirming that the environmental factors play a major role in affecting individual traits, rendering the artificial dichotomy of nature vs nurture meaningless today.

Nevertheless, there remain significant gaps in the literature concerning the development of delinquent behaviors in particular, in how we understand the extent to

which heritability and environment are important. Furthermore, although a number of studies have estimated genetic and environmental effects on delinquency, very few behavior genetic studies tested this question in a developmental framework, the gap that the current study seeks to fill. Thus, the current study employed a nationally-representative data set to answer the question regarding the developmental changes of delinquent behaviors during adolescence and the genetic and environmental effects on the stability and change.

Behavior Genetics

Over the past decade, there has been a large increase in genetically-informed studies of human traits. This field of study, called behavior genetics, emphasizes the contribution of both genes and the environment on individual traits or behaviors (phenotype). This methodology is possible mainly through the use of specific research design, namely twin and adoption studies.

Twin studies employ samples of monozygotic (MZ) and dizygotic (DZ) twins. MZ twins (“identical”) share 100% of their genes, while DZ twins (“fraternal”) share approximately 50% of their genes. Using this difference (while acknowledging certain priors such as equal environments in DZ and MZ families), it is possible to decompose the variance of the phenotype into a heritability component, shared environmental component, and nonshared environmental component. Heritability (h^2) refers to the differences among MZ and DZ twins that is due to their genetic similarity. Shared environment (c^2) refers to differences due to environments the twins share, which is usually conceptualized as family environment. Nonshared environment (e^2) is part of the environment that is idiosyncratic to each sibling. This variance component also includes

measurement error. If the covariance of a certain phenotype score is greater among MZ twin pairs as compared to DZ twin pairs, it suggests that certain part of the variance in this phenotype is due to genetics.

Yet another approach of estimating the above-mentioned components is by using adoption studies. These either compare the phenotype of a child to a phenotype of adoptive parents (with whom they share environment but no genes) as well as to biological parents (with whom they share genes but no environment). Alternatively, it compares biological twins reared apart with adoptive twins reared together.

The results from behavior genetics studies have shown that there is a significant heritable component to most of human phenotypes, psychological traits notwithstanding. In fact, more than three decades of research consistently showed genetic influences on cognitive abilities, psychopathology, personality traits, attitudes, or problem behaviors (Plomin, DeFries, Knopik, & Neiderheiser, 2013). The results show that approximately 40-50% of variance in psychological traits is heritable. Moreover, the findings suggest that this heritability variance comprises many genes with small effects, thus there does not seem to be a “candidate gene” onto which the psychological traits might be mapped (Plomin, DeFries, Knopik, & Neiderhisier, 2016). Finally, it is important to emphasize that these heritability estimates also show that 50-60% of the remaining variance is due to shared and nonshared environmental effects. Behavior genetics, despite its name, is simply a research methodology that does not favor one over the other source of variance; it is, in effect, a complete treatment of all sources of variability in a trait or behavior of interest.

Even though the research in behavior genetics is ever-expanding and its conclusions regarding some genetic influence on psychological variables are currently widely accepted, this has not always been the case. A lot of controversy surrounded behavioral genetic findings, oftentimes stemming from misunderstanding of the methodology, its focus, and its implications (Pinker, 2003). However, not all social sciences have moved to accept evidence from behavioral genetic research at the same pace.

Criminology and Behavior Genetics

Until recently, social sciences tacitly assumed that any psychological phenomena (e.g., attitudes, intelligence, impulsivity) are learned solely through the process of socialization. In this view, the etiology of individual differences is only environmental in nature. The studies using this method thus completely disregarded potential genetic effects when studying family members. For example, finding significant positive correlations between harsh parenting and impulsivity in children was explained as a negative effect of parenting, which could have been prevented if the parenting was different (e.g., warmer and less punitive). Such view ignores the fact many human traits also include a heritable component, shared between parent and a child by definition. In this view then, it is quite possible that the correlation between parenting and child's behavior might be due to some genetic overlap between the parents and the child (Rhee & Waldman, 2003). It might be that a genetic propensity for impulsivity in parents makes them harsh in their parenting, and this was the trait inherited by the child (a passive gene x environment correlation; Moffitt, 2005). This does not mean that this is the only possible explanation for the phenotypical link found or that it completely negates the

effect of parenting on development of impulsivity; however, given what we know about genetic effects on human behavior so far, relying solely on phenotypic correlations without controlling for this source of variance leads to inflated and thus unreliable estimates of the true association between the variables, and importantly, a misunderstanding and partial misattribution of what explains the variance in any given trait or behavior.

Criminology has been very slow to apply the behavior genetic method in its research. Traditionally, given that the roots of criminology are in sociology, it has been dominated by sociological explanation for human behaviors, in part as a reaction to and response to heavily biological explanations used in the 19th century (Lombroso, phrenology). Thus, different social environments (family, school, neighborhood) were considered the key socializing agents in affecting behaviors in individuals. Individual differences were considered as either unimportant or as an outcome of social influences (Udry, 1995). In the case of delinquency, this was explained as stemming from harsh parenting, lack of role models, poor neighborhood, social class, peer influence, or mass media, just to name a few. However, the past two decades of criminological research have shown an increase in studies focusing on individual differences and applying the behavior genetic method (Barnes, Boutwell, et al., 2014; Tuvblad & Beaver, 2013). These studies have shown a presence of a considerable heritability component, in addition to an environmental one. Much of the pioneering work was carried by David Rowe and colleagues, whose research indicated support for genetic sources of variance in delinquency (e.g., Rowe, 2002; Rowe & Osgood, 1984).

Since then, a number of studies have done the same and have shown how genes also seems to play an important role in delinquent behaviors (Barnes, Wright, et al., 2014). The meta-analysis by Rhee and Waldman (2002), based on 51 twin and adoption studies, indicated that approx. 41% of variance in antisocial behavior was explained by genetic influences, 16% by shared environmental influences, and 43% by nonshared environmental influences. A remarkable similarity regarding the heritability estimate was found across the studies regardless of sex, age, operationalization, or zygosity (Viding, Larsson, & Jones, 2008)

However, as recently as 2015, the field continued to engage in heated exchanges on the utility of behavioral genetic research and its findings (Barnes, Wright, et al., 2014; C. Burt & Simons, 2014, 2015; Moffitt & Beckley, 2015; Wright et al., 2015).

Development of Delinquency

One of the most consistent findings in criminological research is the relationship between age and crime. Specifically, it has been repeatedly found that the criminal behavior suddenly increases as individuals move from childhood to adolescence when it peaks in mid-adolescence, only to drop as one moves to the early twenties. In this way, the theory called “age-crime curve” posits that the shape of criminal behavior resembles a bell-shaped curve with peak in mid-adolescence (Hirschi & Gottfredson, 1983). This finding was indicated by Quetelet (1842), a Belgian polymath who first observed this distribution in the 1830s. However, it was Hirschi and Gottfredson who first brought wider attention to this phenomenon when they provocatively posited several facts related to this distribution: 1) the age effect is invariant, meaning that when plotting the rates of deviance by age, regardless of the year of the data, place, demographic groups, or even

type of crime, the shape of the curve remains the same (this does not mean absolute invariance as the shape and the kurtosis of the shape might slightly differ, but the shape would still be characterized by a peak in adolescence, followed by sharp decline); 2) age has a direct effect on crime, meaning that age is simply not a proxy for hypothetical social characteristics but is in fact the main driving force behind the rise and fall of crime levels; 3) no longitudinal designs for studying the causes of crimes are necessary since the development of crime is known and thus cross-sectional studies might be equally valid as longitudinal ones for assessing predictors of crime (Gottfredson & Hirschi, 1986). In this way, Gottfredson and Hirschi might have argued that the age-crime relationship might be biologically determined.

Results supporting these insights on the age-crime curve have been shown by Sampson and Laub in their lifecourse analysis following the trajectory of criminal behavior of 1,000 boys from adolescence to old age. They found that regardless of the severity or frequency of crime behavior, all boys would eventually desist from crime, which peaked during adolescence years (Laub & Sampson, 2006; Sampson & Laub, 2003, 2005). However, Sampson and Laub emphasized that the desistance from crime, i.e., the decline in rates of crime in the twenties, is driven by social control, whereas adolescents come of age and become more connected to the social institutions (they especially emphasize the desisting effect of occupation and marriage), they slowly turn away from crime.

However, there has been a contention whether the age-crime curve is truly universal development of crime propensity and incidence or whether this overall pattern might mask distinctive trajectories. The main proponent of the latter approach is Terrie

Moffitt who followed the development of 512 boys from Dunedin, New Zealand, born between 1972-73, from age 3 to age 26. She postulated the existence of at least two distinctive trajectories of crime/antisocial behavior development: adolescence-limited and life-course-persistent trajectory. The adolescence-limited trajectory resembles the age-crime curve in which the levels of problem behaviors suddenly increase in early adolescence to fall sharply following late adolescence. The life-course-persistent trajectory is characterized by high and stable levels of delinquency across the lifespan, starting from childhood (Moffitt, 1993). This taxonomy was later expanded to include a total of four trajectories, adding groups called “low-level chronics” with persistent low rates of offending from childhood through adulthood, and “abstainers”, who almost never engaged in any delinquent behavior in childhood or adolescence (Moffitt, Caspi, Harrington, & Milne, 2002). Arguably the most important and novel finding of Moffitt’s theory was the life-course-persistent group as the individuals following the lifecourse-persistent trajectory comprise only a small portion of the population (estimated to be 5%), yet are considered to be responsible for the majority of crimes. In this way, this characteristic resembles older conceptualizations of “habitual offenders” or “career criminals” (Blumstein, Cohen, & Farrington, 1988a, 1988b), or the notion that majority of crimes are committed by a minority of individuals, who are characterized by a life-course pattern of repeated offending.

The Genetic Effects on Development of Delinquency

There have not been many studies that applied behavior genetics design to a longitudinal study of delinquency in adolescence. The study by Eley, Lichtenstein, and Moffitt (2003) analyzed the change in aggressive and non-aggressive antisocial behavior

(ASB) from time 1 (age 8-9 years) to time 2 (age 13-14 years). Their results showed that aggressive ASB was highly heritable ($h^2 = .60$) with little shared environment ($c^2 = .15$), and the development was mostly affected by genes; on the other hand, non-aggressive ASB had a substantial effect by both genes and shared environment ($h^2 = .49$, $c^2 = .35$), as was the stability between the waves. Hicks et al. (2007) compared delinquent behaviors in two waves, wave 1 at the age of 17, and wave 2 at the age of 24. They found evidence for increase in heritability in time for men, but no support for increase, and in some cases decrease for women. The study by Wichers et al. (2013) traced the development of externalizing behavior from age 8 to 20 in four timepoints. They found support for a large heritability component ($h^2 = .80$) that remained stable (although with new genetic sources) but no sex differences. Similarly, Kendler et al. (2015) estimated the heritability of criminal behavior in three male cohorts: ages 15-19, ages 20-24, and ages 25-29. They found that heritability was decreasing with age (from 59% to 41% at the last timepoint). Jacobson, Prescott, and Kendler (2002) traced the development of antisocial behavior in three age groups: prior to age 15 (childhood), 15-17 (adolescence), and 18 years and older (adult). They found that heritability increased significantly from childhood to adolescence but remained stable from adolescence to adulthood, with no sex differences observed.

Present Study

The current study sought to answer several important questions. Based on insights from the age-crime curve link, we know that delinquent behaviors peak in adolescence and then decline gradually. The current study took advantage of the first two waves of Add Health to assess the development of delinquency across adolescence. Next, the

genetic, shared, and non-shared variance (ACE) of the delinquent behaviors and changes from Wave 1 to Wave 2 were evaluated using Cholesky decomposition. Thus, longitudinal development was modelled separately in three cohorts, namely early, middle, and late adolescence.

Method

Sample

The data used in this study were drawn from two waves of the Add Health Project, a national longitudinal study of adolescents. The first timepoint (Wave 1) was collected between 1994-95, and consisted of 20,745 adolescents in grades 7-12. The second timepoint (Wave 2) was collected year later in 1996. Add Health data collection design also included a subdataset of siblings, including oversampling of twins. Specifically, there was a total of 3,139 sibling pairs at Wave 1, including 285 monozygotic (MZ) and 430 dizygotic (DZ) twin pairs. This subsample of twins does not deviate from demographic characteristics of the full sample (Jacobson & Rowe, 1999). Due to well-known sex differences in rates of delinquency as well as uncertainty in the literature regarding the existence of qualitative and quantitative sex effects on delinquency (Jacobson et al., 2002; Meier, Slutske, Heath, & Martin, 2011), we decided to reduce the potential complexity by focusing only on same-sex pairs. Because the Add Health dataset (see more below) includes a wide age range of participants at Wave 1 (11-21 years of age), it was necessary to divide the sample into cohorts given the well-known differences in delinquency with regards to age. The siblings were divided in the following way: early adolescents (age 12-14), middle adolescents (15-16), and late adolescents (17+). Given our cohort-based approach, the sample sizes using only twins would be

limited. For this reason, we decided to take advantage of the other types of sibling pairs available in the sibling subdataset, i.e., full siblings (FS), half-siblings (HS), cousins (CO), and non-related siblings (NR). Again, we selected only same-sex pairs of siblings. Given that non-twin siblings vary in age within the pair, we selected only those pairs that were less than 2 years apart in age. For those siblings where their age difference might make them fall into different cohorts, we randomly distributed the siblings so that either sibling one's age or sibling two's age had the same chance of affecting the resulting categorization. The full sample included $N = 1,038$ sibling¹ pairs, with $n = 244$ early adolescents, $n = 430$ middle adolescents, and $n = 364$ late adolescents. Detailed information on the sample demographics are provided in Table 1.

Measures

Control variables.

Age. The age of the siblings. The average age at Wave 1 was 14.9 years for early adolescents, 16.03 for middle adolescents, and 17.67 for late adolescents. At Wave 2, the average age was 15.12 for early adolescents, 16.99 for middle adolescents, and 18.60 for late adolescents.

Sex. The sex of the participants. There was 119 female pairs in the early adolescent cohort (48.8%), 215 pairs in middle adolescence (50.2%), and 204 female pairs (44%) in late adolescence.

Race. To control for potential race differences but given the limited sample size, we recoded race into a dummy-variable with 0 = *White* (reference group) and 1 = *non-White*.

¹ For the ease of understanding, we will refer to all the pairs in the subsample as 'siblings' even though they might not be siblings per se (in case of cousins and non-related pairs).

Delinquency. To create a delinquency measure, we have originally selected 23 items that pertained to different delinquent behaviors (theft, assault, fighting, using substances, vandalism) and that were shared across the two waves. Then, we removed items that had very low variability (e.g., more than 95% responses missing). A total of 15 items was selected (see Appendix I for a list of items). Items 1 and 2 were rated on a 3-point Likert-type scale (0 = *never*, 1 = *once*, 2 = *more than once*); item 8 was dichotomous item, with response categories yes-no; the rest of the items were rated on a 4-point Likert-type scale (0 = *never*, 1 = *one or two times*, 2 = *three or four times*, 4 = *five or more times*). All items were standardized and then averaged to provide a composite score of delinquency. The reliability of the delinquency measure was good in all cohorts and timepoints (Cronbach α ranging from .76 - .90).

Plan of Analysis

First, sibling pairs were compared on background characteristics, across pairs, and, in the case of age and delinquency, within pairs. In all sibling models, the delinquency variable was residualized by sex, age, and race to control for these individual differences (McGue & Bouchard, 1984). In the next step, intraclass correlations were computed in each wave and cohort and compared across sibling pairs. Then, a longitudinal ACE model was estimated using a Cholesky decomposition, where phenotypic variance is decomposed into genetic (A), shared environmental (C), and non-shared environmental (E) component. Moreover, these variance components are allowed to affect subsequent timepoints (in this case, Wave 1 ACE affecting Wave 2 delinquency variance). In this sense, whereas Wave 1 variance is affected by Wave 1 ACE components only, Wave 2 variance is affected by both Wave 2 ACE as well as Wave 1

ACE components, thus controlling for previous timepoint. This Cholesky model was fitted three times, for each cohort. Based on the percentage of DNA shared by siblings, the covariance between genetic sources of variance (A factors) was set to $r_a = 1.0$ for MZ twins, $r_a = .5$ for DZ twins and full siblings, $r_a = 0.25$ for half siblings, $r_a = .125$ for cousins, and $r_a = 0$ for non-related pairs. Since siblings shared the same environment, the covariance of shared environment was set to $r_c = 1.0$ for all pairs except for cousins, where it was set to $r_c = 0$. This model is shown in Figure 1.

The squared estimates of the standardized paths were then used to compute the effect of heritability (h^2), shared environment (c^2), and nonshared environment (e^2) on delinquency in each wave. Furthermore, besides focusing on total variance, it is also possible to decompose the explained variance, i.e., to decompose the stability coefficient or the correlation between Wave 1 and Wave 2. This is done by using the formula: $r = (a_{11} * a_{12}) + (c_{11} * c_{12}) + (e_{11} * e_{12})$, which multiplies the respective paths connecting delinquency at Wave 1 with delinquency at Wave 2 through Wave 1 A, C, E components (see Figure 1). Then, the proportion of correlation due to genetic effect is computed by dividing $(a_{11} * a_{12})$ to the total correlation, while the proportion of shared environment is obtained by dividing $(c_{11} * c_{12})$ by the total correlation, and non-shared environment by dividing $(e_{11} * e_{12})$ by the total correlation.

To estimate statistical significance of the relative contribution of h^2 , c^2 , e^2 in all sibling models, bootstrapping with 5,000 bias-corrected confidence intervals was used. Given that these new parameters do not follow a known distribution, bootstrapping was used to properly estimate their standard errors. All models were run in Mplus version 8

using full information maximum likelihood to estimate missing data (Muthén & Muthén, 1998-2017).

Results

First, siblings were compared on their demographic characteristics. The sibling groups did not significantly differ among themselves in the proportion of male/female pairs in any of the cohorts, early adolescents: $\chi^2(5) = 6.867, p = .231$, middle adolescents: $\chi^2(5) = 3.139, p = .679$, late adolescents: $\chi^2(5) = 6.003, p = .306$. The siblings did not significantly differ on the basis of age within the groups (one sibling older than the other) in any of the three cohorts. Similarly, no significant mean differences were found for sibling pairs in any of the three cohorts (results of pairwise *t*-tests not shown but available upon request). The siblings did differ on the basis of race in each cohort, early adolescents: $\chi^2(5) = 23.294, p < .001$, middle adolescents: $\chi^2(5) = 16.939, p = .005$, late adolescents: $\chi^2(5) = 16.882, p = .005$.

Next, we plotted the standardized delinquency measure based on age of all siblings within the waves to see the mean levels of delinquency based on age. The plot is shown in Figure 2 along with a trendline. It is important to realize that this does not reflect a longitudinal trajectory but rather mean levels of delinquency within the age categories. As is apparent, the delinquency levels in the current sample seem to follow the age-crime curve with middle adolescents showing the highest levels of delinquency.

In the next step, intraclass correlations were assessed for each sibling group within each wave and cohort. These results are presented in Table 2 along with 95% confidence intervals. These results show that MZ twins generally showed higher correlation estimates than other types of siblings, suggesting a genetic effect on

delinquency. However, this was not the case across all timepoints, as Wave 1 in early adolescence and Wave 2 in late adolescence did not show this difference. Furthermore, comparing the correlations between MZ and DZ twins showed that MZ estimates were not twice the size of DZ estimates, suggesting that genetic effect could not fully account for the differences among sibling groups.

Then, three ACE Cholesky longitudinal models were estimated, separately for each cohort. The results from these analyses along with 95% bias-corrected bootstrap confidence intervals are presented in Table 3. The genetic effect on variance in delinquency was substantial and statistically significant in all three cohorts. Comparing across cohorts, heritability explained 37% of variance in Wave 1 in early adolescence, 21% in middle adolescence, and 63% in late adolescence. Moreover, genetic effect on Wave 1 delinquency was also significantly related to explaining delinquency at Wave 2, as it explained 48% of variance in Wave 2 among early adolescents, 25% in middle adolescence, and 32% in late adolescence. On the other hand, little support was found for new genetic effects (change) on delinquency variance at Wave 2. Among early adolescents, about 5% of variance at Wave 2 could be explained by genetic effects different from Wave 1, but this effect was zero among middle and late adolescents. Shared environment explained a modest and non-significant portion of variance in all cohorts. Among early adolescents, it explained 12% of variance at Wave 1, while its effect dropped to 5% for middle and late adolescents at Wave 1. However, in early adolescence, up to 13% of Wave 2 variance was explained by shared environmental influence unrelated to Wave 1. This was not the case for middle and late adolescents, as no new sources of shared environmental influence emerged.

Next, non-shared environment at Wave 1 explained 52% of variance among early adolescents, 74% among middle adolescents, and 33% among late adolescents.

Moreover, non-shared environment was the strongest predictor of new variance at Wave 2, explaining 31% among early adolescents, 65% among middle adolescents, and 63% among late adolescents. The Table 3 results are visualized in Figure 3.

Given that in Cholesky decomposition, the stability between waves is directly modeled, the variance in Wave 2 represents change from Wave 1 to Wave 2. In this sense, genetic effects are responsible for 10% of change in early adolescence, while shared environmental effects are responsible for 27% of change, and non-shared environmental effects are responsible for 63% of change. On the other hand, in both middle and late adolescence, the non-shared environmental effects are responsible for 100% of change from Wave 1 to Wave 2.

Finally, the stability coefficients from Wave 1 to Wave 2 were also decomposed. The results from these analyses are shown in Table 4. The stability in delinquency was very similar in all three cohorts, i.e., $r = .51/.49/.55$, respectively. Regarding the decomposition of the stability, the results are strikingly similar for early and late adolescents, as 82% of stability in early adolescence was explained by genetics (82/80% in late adolescence), 11%/7% explained by shared environment, and 6%/12% explained by non-shared environment. The results are much different for middle adolescence, where heritability explained only 47% of the stability, followed by 9% of shared environment, and 44% of non-shared environment. These results are presented in Figure 4.

Discussion

The aim of the current study was to use a genetically-informed design to evaluate the longitudinal stability and change of delinquency in three adolescent cohorts: early adolescence, middle adolescence, and late adolescence. The present results corroborate as well as extend findings from previous longitudinal studies of delinquency in samples of twins.

First, the current results confirmed that there was a significant and substantial genetic effect on delinquency. This finding confirmed previous studies finding substantial heritability for this type of behavior (Ferguson, 2010; Rhee & Waldman, 2002). Related to this, the heritability of delinquency differed based on the cohort analyzed. Middle adolescence showed absolutely lowest values of heritability as compared to early and late adolescents, while showing the highest estimates of non-shared environmental effects. This might be due to the fact that delinquency in our study peaked in middle adolescence. Middle adolescence is also the period where peer influence on individual's delinquency culminates (Gardner & Steinberg, 2005; Steinberg & Monahan, 2007). For this reason, genetic effects on variance in delinquency in this period are smaller because delinquency becomes normative in the sense that "everyone is doing it." If a large number of adolescents become involved in some delinquent behavior, then the genetic effect on individual differences becomes muddled (Lahey, Waldman, & McBurnett, 1999). It is possible that the large effect of non-shared environment on total variance as well as on stability might reflect the increasingly stronger peer effects in middle adolescence, as engaging in unstructured socializing activities ("hanging out with friends") has been found to be associated with higher delinquency (Augustyn & McGloin, 2013; Osgood,

Wilson, O'Malley, Bachman, & Johnston, 1996). Among middle and late adolescents, new sources of non-shared environment emerged at Wave 2, lending support to the hypothesis that as individuals grow older, they tend to move away from family influences and thus are more affected by unique experiences away from home (Scarr & McCartney, 1983). Of course, one has to bear in mind that nonshared environmental variance also includes measurement error, thus inflating its total effect.

The genetic effects might be better observed in cohorts where delinquent behavior is not normative – when it is too early (early adolescence), or too late (late adolescence). This was the case in the current study, as we found that among early adolescents, heritability explained 37% of variance at Wave 1 and 53% at Wave 2, while in late adolescence, it explained 63% at Wave 1 and 32% at Wave 2, both much higher than heritability during middle adolescence. It is unclear to us what might have caused the large one-year drop in heritability among late adolescents. Looking back at estimates of intraclass correlations, they showed that the correlation for late adolescent MZ twins at Wave 2 was inexplicably low and statistically non-significant. Given that this estimate was in stark contrast to more predictable patterns of correlations found in other sibling pairs, it is possible that this might not be reflect a meaningful development as much as a methodological issue related to the data collection procedure.

These conjectures are further underlined when decomposing only the explained variance, i.e., the stability of delinquency from Wave 1 to Wave 2. Although the stability from Wave 1 to Wave 2 was very similar across the three cohorts (approximately $r \sim .50$), its decomposition into genetic and environmental effects further highlighted the unique role of middle adolescence. Whereas the decomposition of delinquency stability

among early and late adolescents is almost identical (~80% heritability, 6%/12% of non-shared environmental effect), in middle adolescence, a full 44% of the stability variance is due to non-shared environment (with 47% heritability). This again suggests that differences in delinquent behaviors among middle adolescents are less affected by genetic sources, and are less stable and predictable, depending more on non-shared environments.

The current results suggest that heritability is the main driving force behind stability of delinquency, confirming results from previous studies (Harden, Quinn, & Tucker-Drob, 2012; Wichers et al., 2013). On the other hand, longitudinal change was for the largest part driven by non-shared experiences. Although it might be tempting to consider the three cohorts as forming part of a prototypical delinquency development throughout adolescence, it is necessary to keep in mind that these cohorts comprise different individuals and thus, the potential for cohort effects cannot be ruled out. On the other hand, the heritability showed a very large effect on the stability of delinquency, especially among early and late adolescents. Except for a small effect in early adolescence (5%), no support was found for new genetic sources of delinquency emerging during adolescence. Although as mentioned above, the cohorts are not temporally associated, it seems unlikely that the heritability in delinquency might be associated with different sources than the one captured in younger cohort. As such, the results of the current study seem to point out in the conclusion of several previous studies, which found that the development of delinquency was largely affected by the same genetic source (Eley et al., 2003; Van Hulle et al., 2009).

Regarding the effects of shared environment, the relatively largest effect on variance was found in early adolescence, where it explained a total of 12% at Wave 1 and 16% at Wave 2 (13% from new source at Wave 2). However, in both middle and late adolescence, the estimates became smaller and unchanged (5% at Wave 1, 4% at Wave 2). This is in line with other studies in this area, which too found that the effect of shared environment decreased with age (Jacobson et al., 2002; Miles & Carey, 1997)

Limitations and Future Directions

There are several limitations of the current study worth mentioning. First, although the Add Health dataset includes a total of 3,139 sibling pairs, we had to limit the sample with regard to cohort, age difference, and sex. In this way, the standard errors for genetic and environmental decomposition might be too large (as indicated by wide confidence intervals), which leads to insufficient power for detecting statistical significance for some of the smaller estimates. However, as we acknowledge this limitation, we believe that our sample selection was necessary for an unbiased estimate of research questions at hand.

First, given the well-known associations of age and delinquency, it was necessary for us to split the Add Health sample into cohorts. Decades of research finding support for the age-crime curve have shown that adolescents vary greatly with regards to the mean levels of delinquency, which peaks in middle adolescence, only to decrease sharply from late adolescence onwards (Hirschi & Gottfredson, 1983). This age-crime curve was also confirmed in our current study. Further, the differences among cohorts were highlighted in the ACE models, as middle adolescents showed much different patterns of findings as compared to early and late adolescents.

Second, the literature seems to be ambiguous related to the effect of sex. Some studies found support for differences in estimates and sources of heritability and environment based on sex (Frisell, Pawitan, Långström, & Lichtenstein, 2012; Hicks et al., 2007; Van Hulle, Rodgers, D'Onofrio, Waldman, & Lahey, 2007), while other studies suggested there were no sex differences (Ferguson, 2010; Jacobson, Prescott, & Kendler, 2002; Van Hulle et al., 2007). For this study, we decided to focus simply on same-sex pairs to make the interpretations more straightforward. Furthermore, we controlled for the effects of sex in all analyses but given the limited sample size, we did not model males and females separately. Future studies with larger samples and more timepoints within adolescence period might be able to provide a robust test of the supposed sex differences in heritability of delinquency.

The measure of delinquency in our study was broad in its scope as it included several different types of antisocial behaviors. In this sense, the conceptualization of the delinquency measure did not distinguish between e.g., aggressive and non-aggressive antisocial behavior (Eley et al., 2003). This was an a priori decision on our part as we tried to capture a variety of delinquent behaviors one might be involved in during adolescence. Furthermore, informed by previous research, these delinquent behaviors are highly inter-related (Krueger, Markon, Patrick, Benning, & Kramer, 2007), and are presupposed to stem from the same genetic source (Dick, 2007; McGue, Iacono, & Krueger, 2006; Slutske et al., 1998). Finer refinement of these delinquency items might suggest different estimates of heritable and environmental effects, as some previous studies indicated that more serious behaviors might have higher heritability than less serious crimes that might be age-normative (DiLalla, 2002).

Finally, it is worth mentioning that the two waves of Add Health data used in this study were collected between 1994-1996. Although the dataset was nationally representative, it is now more than 20 years old. Given the historical changes in levels of adolescent crime and delinquency, with the early nineties actually being the period of highest rates in the US (OJJDP Statistical Briefing Book, 2017), the estimates of genetic and environmental effects on variance in delinquency might differ based on historical era. If there are periods where crime is much more prevalent, perhaps such antisocial acts are considered as more normative (in the sense of ‘more common’) than in times with lower crime rates. In this way, higher salience of crime might present itself as more opportunities for crime in a society. This might lower the threshold for engaging in crime and delinquency and thus lead to a lower genetic influence on crime and delinquency. This speculation reflects upon studies of gene-environment interaction, which found that heritability of delinquency increases with socioeconomic status. They suggest that individuals living in poor conditions are more likely to be pushed to antisocial behavior by social risk factors, whereas the effect of these factors is lacking for individuals in more affluent environments, thus genetic effects become more important for individual differences (Raine, 2002; Tuvblad, Grann, & Lichtenstein, 2006). Perhaps there would be a similar effect of historical era on heritability of delinquency and as such, future studies using more contemporary samples would be instrumental for comparing these results.

Tables and Figures

Table 1-1 *Descriptive Statistics of Sibling Age Groups*

		<i>n</i>	% female	% non- White	<i>M</i> age Wave 1	<i>M</i> age Wave 2	α Wave 1	α Wave 2
Early adolescence								
	MZ	64	59.4%	46.9%	14.15	15.09		
	DZ	62	43.5%	35.5%	13.91	14.88		
	FS	55	43.6%	37.1%	14.53	15.42		
	HS	20	65.0%	58.9%	14.44	15.33		
	CO	16	62.5%	87.5%	14.01	15.00		
	NR	27	48.2%	19.2%	14.17	15.08		
	Total	244	48.8%	43.1%	14.19	15.12	.87	.76
Middle adolescence								
	MZ	113	46.9%	39.8%	16.02	17.03		
	DZ	97	53.6%	48.4%	15.96	16.91		
	FS	123	47.1%	42.3%	16.14	17.09		
	HS	26	46.1%	65.4%	15.89	16.78		
	CO	22	63.6%	77.3%	15.98	16.94		
	NR	49	51.0%	36.7%	16.00	16.96		
	Total	430	50.2%	44.8%	16.03	16.99	.87	.80
Late adolescence								
	MZ	90	45.6%	40.0%	17.95	18.88		
	DZ	76	38.2%	44.7%	17.82	18.73		
	FS	126	46.1%	46.8%	17.41	18.33		
	HS	9	10.1%	77.8%	17.98	18.53		
	CO	16	50.0%	87.5%	17.58	18.59		
	NR	47	48.9%	55.3%	17.56	18.49		
	Total	364	44.0%	47.2%	17.67	18.60	.90	.89

Note. MZ = monozygotic twins, DZ = dizygotic twins, FS = full siblings, HS = half siblings, CO = cousins, NR = non-related siblings.

Table 1-2 *Intraclass Correlations for Sibling Groups*

		MZ	DZ	FS	HS	CO	NR
Early adolescence	Wave 1	.41 [.18, .59]	.43 [.21, .61]	.46 [.22, .64]	-.14 [-.54, .32]	.03 [-.45, .50]	-.01 [-.38, .37]
	Wave 2	.55 [.34, .70]	.41 [.17, .61]	.44 [.19, .64]	.06 [-.38, .49]	-.06 [-.52, .43]	.48 [.09, .74]
Middle adolescence	Wave 1	.32 [.15, .48]	.20 [.01, .39]	.08 [-.10, .25]	-.15 [-.50, .24]	.05 [-.37, .45]	.11 [-.18, .38]
	Wave 2	.30 [.11, .47]	.23 [.02, .42]	.22 [.04, .39]	-.05 [-.43, .35]	-.04 [-.49, .44]	-.12 [-.42, .22]
Late adolescence	Wave 1	.69 [.56, .78]	.39 [.18, .56]	.11 [-.07, .28]	-.09 [-.66, .57]	.00 [-.47, .48]	.08 [-.21, .36]
	Wave 2	.11 [-.11, .32]	.28 [.05, .49]	.33 [.15, .50]	-.49 [-.87, .33]	-.11 [-.56, .41]	-.04 [-.38, .31]

Note. The brackets refer to the 95% confidence intervals of the correlation estimates. MZ = monozygotic twins, DZ = dizygotic twins, FS = full siblings, HS = half siblings, CO = cousins, NR = non-related siblings.

Table 1-3 *Standardized, Squared Path Estimates from Longitudinal Genetic Analyses*

		Early adolescence		Middle adolescence		Late adolescence	
		Wave 1	Wave 2	Wave 1	Wave 2	Wave 1	Wave 2
Wave 1	A	.37 [.04, .83]		.21 [.01, .66]		.63 [.36, .83]	
	C	.12 [.00, .40]		.05 [.00, .18]		.05 [.00, .21]	
	E	.52 [.16, .77]		.74 [.39, .87]		.33 [.17, .59]	
Wave 2	A	.48 [.15, .87]	.05 [.00, .46]	.25 [.01, .54]	.00 [.00, .00]	.32 [.06, .74]	.00 [.00, .42]
	C	.03 [.00, .26]	.13 [.00, .34]	.04 [.00, .23]	.00 [.00, .11]	.04 [.00, .19]	.00 [.00, .07]
	E	.01 [.00, .02]	.31 [.11, .59]	.06 [.00, .25]	.65 [.50, .80]	.01 [.00, .22]	.63 [.44, .80]

Note. Numbers in brackets refer to the 95% bias-corrected bootstrapped confidence intervals. The sums of the estimates within a wave might differ slightly from 1 due to rounding.

Table 1-4 *Variance Decomposition of Stability Paths*

	Wave 1 – Wave 2 stability		
Early adolescence	$r = .52$	h^2	.82 [.34, .98]
		c^2	.12 [.00, .67]
		e^2	.06 [.01, .19]
Mid adolescence	$r = .48$	h^2	.47 [.05, .95]
		c^2	.09 [.00, .35]
		e^2	.44 [.03, .76]
Late adolescence	$r = .55$	h^2	.80 [.50, .99]
		c^2	.07 [.00, .35]
		e^2	.12 [.01, .45]

Note. h^2 = heritability, c^2 = shared environment, e^2 = non-shared environment. The brackets refer to 95% bias-corrected bootstrapped confidence intervals.

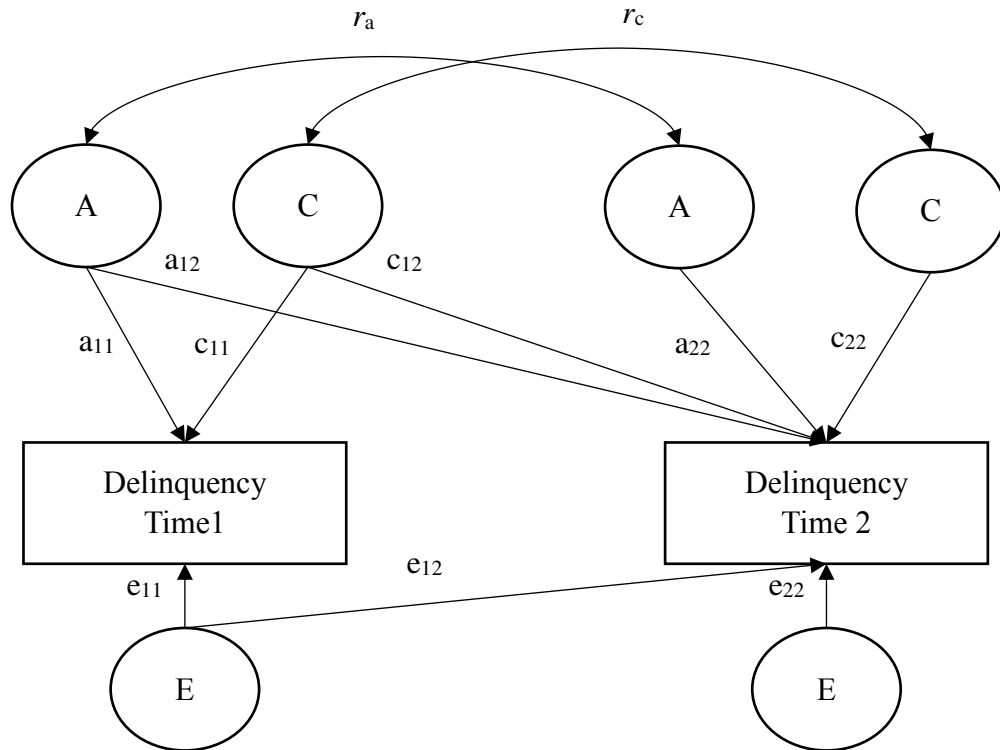


Figure 1-1 ACE model with Cholesky decomposition.

Note. Genetic covariance was set in the following way: $r_a = 1.0$ (MZ), $r_a = 0.5$ (DZ and FS), $r_a = .25$ (HS), $r_a = .125$ (CO), $r_a = .00$ (NR). Shared environment covariance (r_c) was set to 0 for CO and 1 for all other types of siblings. This model was assessed separately for early adolescents, middle adolescents, and late adolescents.

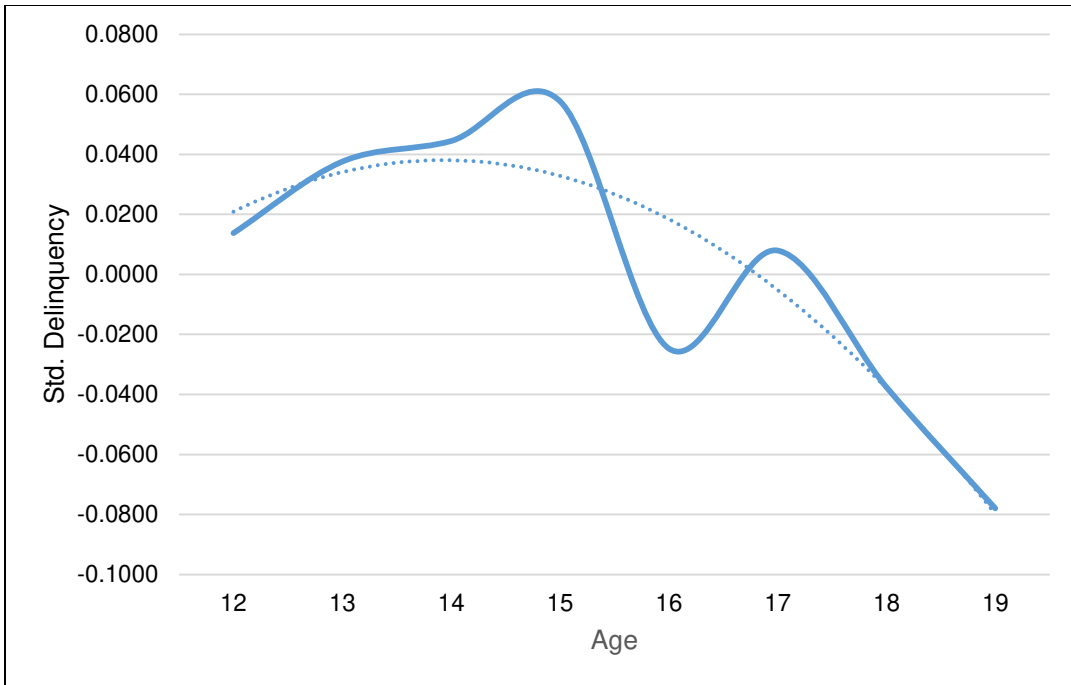


Figure 1-2 Standardized mean levels of delinquency across age groups.

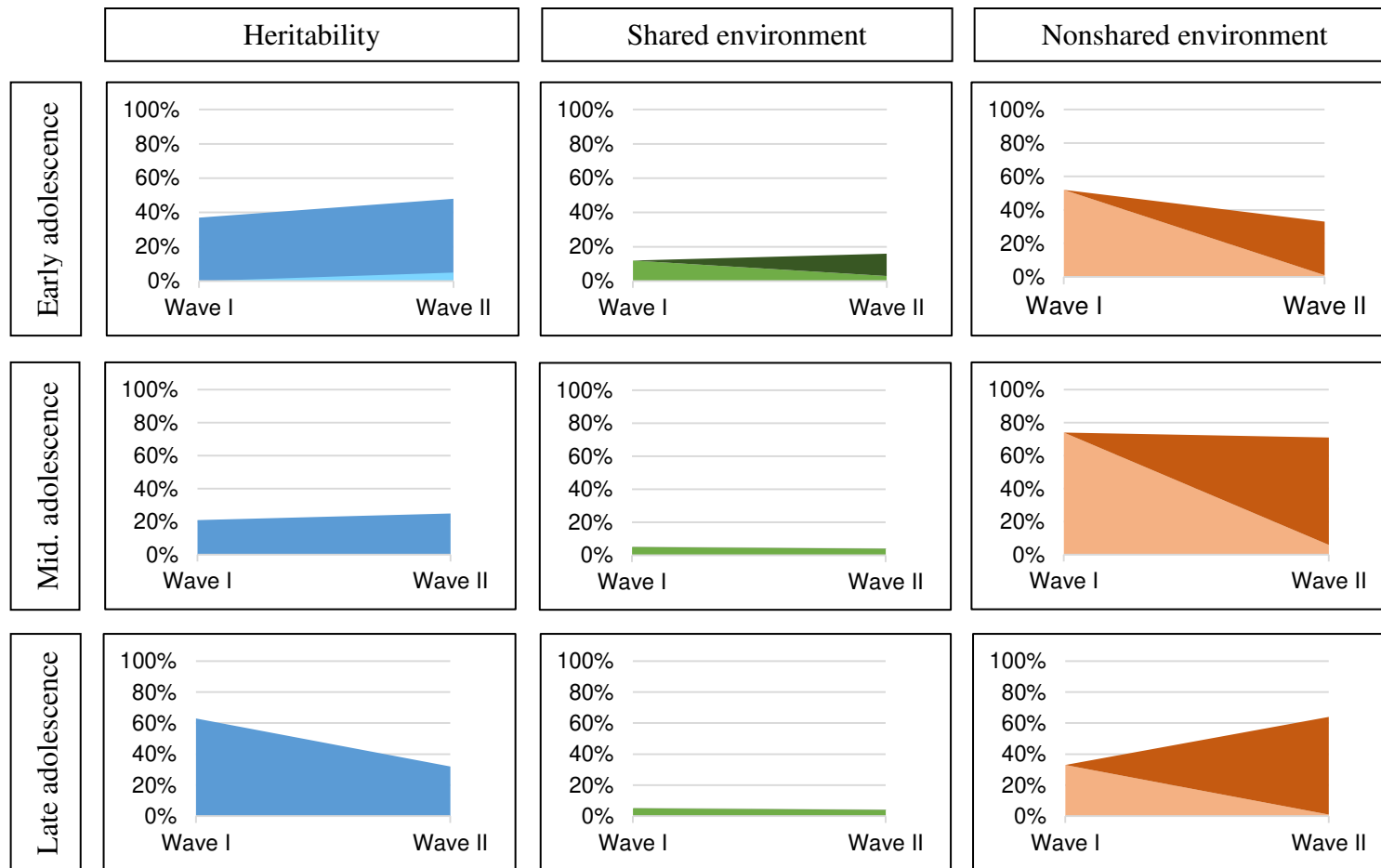


Figure 1-3 The relative genetic and environmental influences on stability and change in delinquency within each cohort. Different shade of color refers to new source of variance at Wave 2.

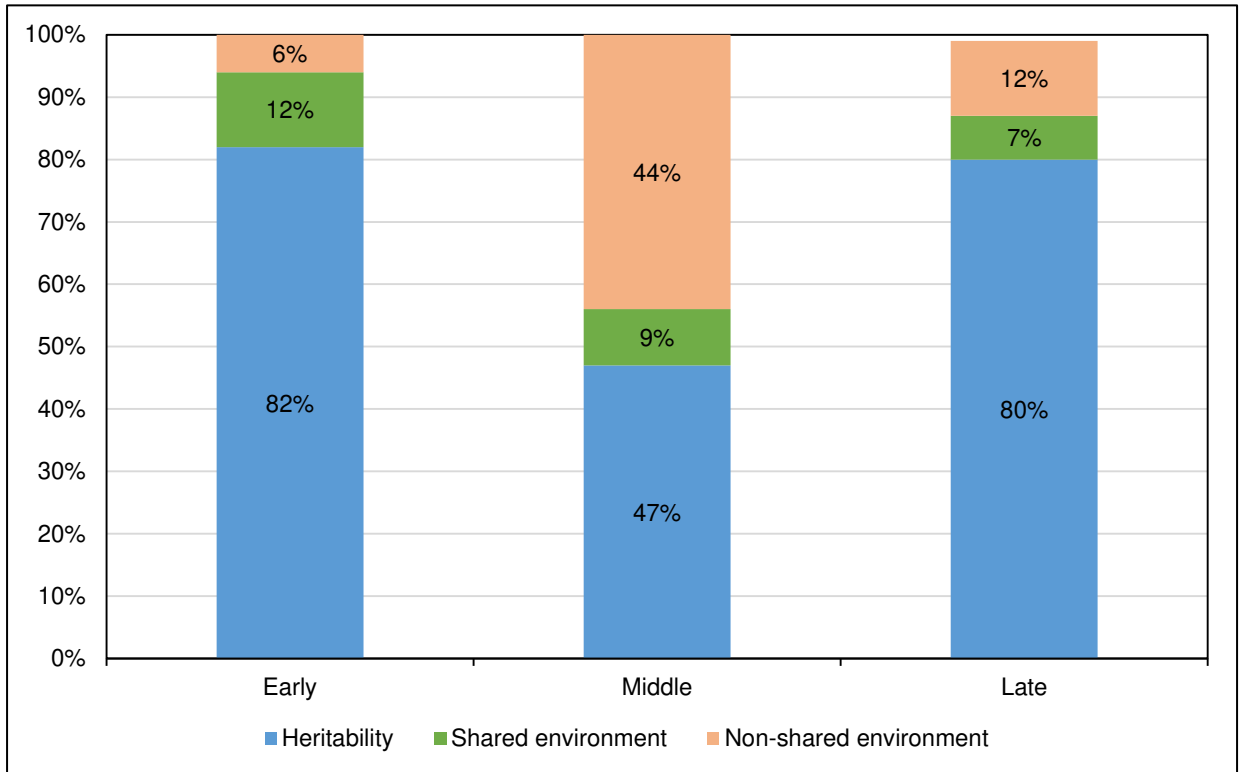


Figure 1-4 The proportion of variance in stability from Wave 1 and Wave 2 explained by genetic and environmental effects.

Note. Early = early adolescence, Middle = middle adolescence, Late = late adolescence.

STUDY II: THE ROLE OF DOPAMINERGIC GENES IN THE RELATIONSHIP
BETWEEN PARENTING AND ADOLESCENT DELINQUENCY

Abstract

Traditionally, parenting is considered as one of the most important protective factors for adolescent negative behavioral outcomes, including delinquency. In addition, behavior genetic research has shown that most psychological traits have a substantial genetic basis, suggesting that children might differ in their initial propensity for certain behaviors. Furthermore, recent advances in genetic research has enabled to test how and whether these genetic propensities interact with their environments, including parenting. The current study tested the GxE interaction between two dopaminergic polymorphisms (DRD4 7-repeat allele and DRD2 A1 allele) and parenting, operationalized by child abuse on the one negative extreme and maternal closeness on the other, in longitudinally predicting delinquent behaviors. Furthermore, given the existence of the hypothesized interactive effects, the study also tested whether the interactive effects were consistent with a diathesis-stress model or with a competing differential susceptibility model. The nationally representative sample of Add Health participants included $N = 8,932$ individuals. The results showed significant positive association of child abuse ($\beta = .201$, $p < .001$) with delinquent behaviors in adulthood as well as a protective effect of parental closeness ($\beta = -.072$, $p < .001$). Neither parental closeness nor child abuse showed a significant interaction with DRD2 or DRD4 in predicting later delinquency.

Introduction

One of the most common predictors of child and adolescent adjustment has been parental behavior. Decades of research have found parenting to affect children adjustment, both as risk factors (such as child maltreatment; Cicchetti & Toth, 2005; Kim & Cicchetti, 2010), as well as protective factors (such as close relationship; Lowe & Dotterer, 2013; Vieno, Nation, Pastore, & Santinello, 2009). However, vast majority of these studies have been driven by the standard social science method, which implicitly assumes that any associations between parents and their children are due to environmental factors (Barnes, Boutwell, Beaver, Gibson, & Wright, 2014). However, the past two decades of research have attested to the notion that human traits are heritable to a major extent (Plomin, DeFries, Knopik, & Neiderhiser, 2016; Turkheimer, 2000). This also means that a significant amount of the correlations found between parenting and children's outcomes could be due to genetic affiliation of parents and children.

A specific line of research in genetically-informed studies has been focused on the interaction between a child's specific genes and parenting behavior. Traditionally, this research has been framed by the diathesis-stress model, which focuses on the interaction of genetic and environmental risk factors for developing negative adjustment. An alternative to this view was proposed by Belsky and colleagues (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Belsky & Pluess, 2009a), who argued that carrying certain genes might not necessarily translate only to vulnerability to negative parenting, but also might be beneficial when found in a positive environment. In this way, genetic predispositions are considered to represent individual heightened sensitivity to an environment, which can lead to either negative or positive adjustment based on the

quality of the environment. The current study tested this proposition in a national sample of adolescents. Using two dopaminergic gene polymorphisms (DRD2 and DRD4), it tested whether their interactions with negative parenting (child abuse) as well as positive parenting (maternal closeness) longitudinally predicted delinquent behavior.

Parenting Research

Individuals learn about norms and functions of society in the process of socialization. The primary socializing agents include parents, who are the most proximal environment (microsystem), according to Bronfenbrenner (1977). Parents are essential because they provide children with norms and values of the society they live in through the process of modeling and learning. Research has shown that parental behavior and parenting affects children's development and their behaviors. Stemming from the socialization perspective emphasizing learning, the parent-child effect was perceived as environmental in origin. The underlying idea is that parents mold the children through their behavior via two paths – providing an example of appropriate behaviors (through the process of social learning) and regulating children's behaviors. In this way, through the process of socialization, children's characteristics are largely molded by the home environment.

Parental socialization was already emphasized in psychoanalysis as well as behaviorism, both grand theories that shaped psychological thinking, theorizing, and research during the first half of the 20th century (Darling & Steinberg, 1993). The conceptualization of parental behavior soon emerged. Several researchers proposed different dimensions of parenting behaviors; despite the fact that slightly different terms were used, they seemed to converge on main parenting dimensions: one related to the

quality of parental emotional relationship with a child and one related to the parental involvement and control of the child's behavior (Baldwin, 1948; Schaefer, 1965).

Influential work in this regard was done by Diana Baumrind, who combined low and high values on these two dimensions, which she termed responsiveness (referring to emotional relationship), and demandingness (referring to parental involvement and control), into parenting styles. According to Baumrind, there were three main styles of parenting: authoritarian (low responsiveness, high demandingness), authoritative (high responsiveness, high demandingness), and permissive (high responsiveness, low demandingness). Later on, Maccoby and Martin (1983) added a fourth parenting style called 'neglectful,' defined as low on both demandingness and responsiveness.

Subsequent studies have shown that the authoritative style was the optimal parenting style by its relation to positive outcomes in children (e.g., Baumrind, 1991; Lamborn, Mounts, Steinberg, & Dornbusch, 1991; Steinberg, Lamborn, Darling, Mounts, & Dornbusch, 1994).

Gray and Steinberg (1999) put forth the argument that researchers need to further "unpack" this parenting typology, namely define what precise dimensions are involved in parenting or family processes. Darling and Steinberg (1993), and later Steinberg and Silk (2002) argued for the distinction between parenting styles and parenting practices.

Parenting style, in their view, refers to an overall 'climate' of the parent-child relationship. On the other hand, parenting practices are actual parent-child interactions that happen and get their meaning from parenting styles. According to Steinberg and Silk (2002), parenting styles can be grouped into three main parenting dimensions: harmony (acceptance-involvement), autonomy (both psychological and behavioral control), and

parent-child conflict, a distinctive dimension that is often predicted by the other ones. For optimal functioning, children need to feel loved from their parents, need to have set clear boundaries, and they then need to be provided with autonomy within these boundaries.

Past research has consistently identified several parenting behaviors as important predictors of children's adjustment. One of the most salient and negative outcomes is externalizing behavior. Externalizing behavior comprises a broad set of behaviors that encompasses delinquent or deviant behavior, including use of violence, alcohol and substance use, school absenteeism, or overly criminal behaviors, such as stealing, vandalism, rape, or murder. Among specific parenting behaviors that were found to predict children's externalizing behaviors and delinquency, two are the most prominent. Child abuse and close parenting are two poles of a spectrum of parenting ranging from harmful parenting to a parenting dimension that has been found to be associated with best child outcomes. Maltreatment of a child in the form of abuse and/or neglect creates a pathogenic relational environment, with a potential for developing maladjustment among children (Cicchetti & Toth, 2005). This was supported by empirical studies, as child maltreatment has been consistently found to be associated with worse adjustment in children, including higher levels of delinquency (e.g., Keiley, Howe, Dodge, Bates, & Pettit, 2001; Kim & Cicchetti, 2010; Manly, Kim, Rogosch, & Cicchetti, 2001). Conversely, a close emotional bond between parent and child (referred to as parental warmth, closeness, connectedness, or attachment) has been identified as one of the key parenting dimensions for a healthy child development. Parental closeness has been found to be a protective factor against child's and adolescent's externalizing behavior in many

studies (e.g., Berkien, Louwse, Verhulst, & van der Ende, 2012, MacKenzie, Nicklas, Waldfogel, & Brooks-Gunn, 2012, Vieno, Nation, Pastore, & Santinello, 2009).

Taken together, a plethora of past research has emphasized the association of parental behavior and children's adjustment and behavioral outcomes. However, the vast majority of this research implicitly assumed that the effect of parenting on children is transmitted through socialization and social interaction only. In other words, the common assumption is that children's adjustment (broadly speaking) is determined by the social environment, mostly nurture, with the underlying assumption that children are blank slates and subsequently inscribed by experiences, principally parenting (Pinker, 2003).

However, such view is untenable given the development in our understanding of genetic effects. First, studies have shown that most of human characteristics are, to a varying extent, heritable, and psychological traits are no exception (Plomin et al., 2016; Turkheimer, 2000). This paradigm shift appeared during the 1980s prominently in the work of Sandra Scarr, Robert Plomin, David Rowe, and other researchers using the behavior genetic method. These authors argued that genetic affiliation between parents and their children plays an important role in the observed correlations between parenting and children. Children are genetic "products" of their parents in the sense that they share 50% of each parent's genetic material. Furthermore, parents also provide the environments for their children, so that children's experiences during a large part of their lives are carried out in contexts created by their parents. Scarr argued that the effects of actual parenting behavior on children outcomes are limited in range so that only extreme parenting (such as child neglect) might have a significant negative effect on the outcome while the remaining variation within the 'normal' range does not meaningfully affect

these outcomes above the effect of genes (Scarr, 1992). This later point (whether there is a variance in the effects of ‘normal’ parenting on child development) was and still remains a controversial issue among researchers with probably a majority of them actually emphasizing the substantial and key role of active parenting in children’s development (Baumrind, 1993; Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000).

Thus, to ignore potential effects by genes on the link between parenting and children’s adjustment would in fact ignore much of the existing evidence. Truly, past research has shown that much of the parenting effect, which might be superficially ascribed to ‘environment’ or nurture, can be in fact explained in terms of heritability (Rowe, 1994). This is shown in a concept called gene-environment correlation, referred to as r_{GE} .

Genetic Basis of Parenting

There are three types of r_{GE} : passive r_{GE} , evocative r_{GE} , and active r_{GE} (Plomin, DeFries, & Loehlin, 1977; Scarr & McCartney, 1983). Passive r_{GE} refers to parents passing their genes as well as the environment to their children. In the context of the association between parenting and child outcomes, the passive r_{GE} explanation might be used for cases when parenting and children’s behaviors might be explained by children inheriting a certain genetic propensity from their parents, which might be correlated with the environment they live in. For example, the well-known association between harsh parenting and a son’s externalizing behavior could be explained by a parent’s impulsivity trait that makes their parenting harsh, but this trait is also inherited by the boy himself. The impulsive trait (the genetic propensity) of the son makes him

then more likely to engage in externalizing behaviors, which might be further exacerbated by the environmental factors he experiences (actual harsh parenting).

The second type is evocative *r*GE, which refers to individuals eliciting responses from an environment on the basis of their genetic predispositions. For example, an infant with irritable temperament might elicit angry responses from parents, which further heightens the risk for developing externalizing or problem behaviors. In this case, the genetic propensity affects or even molds the environmental reaction (parenting behavior). The third type is active *r*GE, in which individuals actively seek environments that correspond to their genetic predispositions. This type of *r*GE is not applicable to parent-child relationships, as children do not actively select their parents. However, active *r*GE was found to play a role in the process of peer or mate selection.

Evidence from behavior genetic research has shown that the hypothesis of purely environmental effect of parenting on children's outcomes is untenable. In this way, any research design that does not take into account the genetic relatedness of parents and children might yield inflated or even spurious estimates for the parent-child associations. Studies using this genetically-informed design in assessing children's outcomes have consistently found that the variance affected shared environment was very modest, oftentimes resembling or close to zero, generally not larger than 20% (S. Burt, 2009). However, this does not mean that parenting does not affect children at all. Rather, it shows that an environmentally-transmitted effect of parenting on children's outcomes above and beyond the genetic affinity and its correlation with environment (which is accounted for in the heritability estimate) is limited and modest at best.

Besides genetic-environment correlation outlined above, a specific line of research in behavior genetics has focused on genotype-environment interactions (GxE). This research focuses on how genetic effects vary as a function of environmental measures (or vice versa). This is oftentimes carried out by employing specific genotypes that are known to be associated with certain outcomes. In a groundbreaking study in this area, Caspi et al. (2002) found that children who carried a monoamine oxidase A (MAOA) enzyme with low expression were more likely to develop antisocial behaviors in adulthood as a reaction to childhood maltreatment than children with high expression of this enzyme. In this way, it is hypothesized that a different genetic sensitivity to environmental influences might explain why some individuals show certain outcomes as a reaction to environmental predictors while for others, the association might be weaker or even null. A number of studies have tested the interactive effect of different genotypes and parenting behavior on children's adjustment.

Dopaminergic System

The dopaminergic system in brain is involved in motivational behavior and approach orientation, related to active exploration and approach towards novel stimuli (Propper, Willoughby, Halpern, Carbone, & Cox, 2007). The dopaminergic genes have been proposed to be associated with impulsive behavior, risk taking, attention deficit hyperactive disorder (ADHD), or substance abuse, suggesting that these traits may share a common congenital basis, all stemming from a certain neurobiological motivational mechanism (Munafò, Yalcin, Willis-Owen, & Flint, 2008). In the existing literature on the topic, there are two genes that were proposed as candidate genes for explaining the genetic basis of these problem behaviors: DRD4 and DRD2.

DRD4. The DRD4 is the dopamine D4 receptor gene, which is located on the short arm of chromosome 11. The existing research seems to suggest that the presence of the DRD4 allele with 7-repeat variant (“long”) might be a candidate gene for affecting variety of behaviors associated with problem behaviors. Specifically, the long alleles of DRD4 have been found to be associated with personality trait of novelty seeking (Strobel, Wehr, Michel, & Brocke, 1999), even among non-humans (Bailey, Breidenthal, Jorgensen, McCracken, & Fairbanks, 2007). A number of studies have also found the association of the DRD4 with ADHD (Faraone et al., 1999; Gizer, Ficks, & Waldman, 2009). However, several meta-analyses failed to produce significant estimates for the DRD4 long allele and the proposed outcomes, such as novelty seeking or externalizing behavior (Kluger, Siegfried, & Ebstein, 2002; Munafò et al., 2008; Schinka, Letsch, & Crawford, 2002).

Instead of main effects, most of the studies on the effect of DRD4 have focused on the interactive effect of DRD4 and environmental factors. Several studies found that this polymorphism significantly modified the association between parental quality and externalizing behavior so that the association was significant only for children who carried the 7-repeat allele (Bakermans-Kranenburg & van IJzendoorn, 2006a; Sheese, Voelker, Rothbart, & Posner, 2007; Windhorst et al., 2015). However, Propper et al. (2007) found that the interactive effect of DRD4 and warm-responsive parenting in predicting externalizing behavior was only significant for the short polymorphism of DRD4, not long, and this was found only among the African American subsample. On the other hand, Beach, Brody, Lei, and Philibert (2010) in the sample of African

American adolescents found that it was individuals with a long DRD4 allele that showed greater response to intervention program against substance use.

DRD2. Similarly, the dopamine D2 receptor gene has been also suggested as a candidate for explaining the genetic basis of externalizing behaviors, especially substance use. Specifically, the A1 allele, sometimes referred to as a reward gene, was found to be associated with higher novelty seeking (Suhara et al., 2001), and oftentimes found to be associated with substance use (Bowirrat & Oscar-Berman, 2005). Furthermore, a number of studies provide evidence that the presence of the DRD2 A1 polymorphism might moderate the effect of environmental predictors, particularly the effect of parenting behavior. A study by Zwaluw et al. (2010) found that adolescents with highly permissive parenting towards alcohol consumption and the DRD2 A1 allele used significantly more alcohol than individuals without these characteristics. The study by Guo, Roettger, and Shih (2007) found that among males in the Add Health sample, the A1/A2 heterozygotes showed the highest trajectories of delinquency as compared to the A2/A2 and A1/A1 genotypes. Similarly, Keltikangas-Järvinen et al. (2009) found that A1 genotype interacted with punitive maternal style in predicting higher levels of novelty seeking.

However, the study by Creemers et al. (2011) did not find a GxE interaction for either DRD2 or DRD4 with parenting on the development of alcohol or cannabis use. Similarly, Hiemstra, Engels, Barker, Schayck, and Otten (2013) tested the moderating effect of dopaminergic genes (DRD4, DRD2, DAT1) on the association between smoking-specific parenting and smoking onset in adolescence. They did not find any interactive effect for the DRD4, DRD2, or DAT1 genotypes with parental behavior on the onset of smoking. A study by Chhangur et al. (2015) assessed the interactive effect of

both DRD4 and DRD2 with parental support in predicting adolescent delinquency. The results showed no significant interaction for the DRD4 and parental support; a significant finding was found for DRD2, however this was found for the A2A2 genotype instead of the ones involving A1 allele. Thus, there is a mixed evidence regarding the interactive effect of DRD2 and parenting for predicting substance use.

Vulnerability versus Susceptibility

Traditionally, the theoretical framework for GxE studies was based on the diathesis-stress model. This model emphasizes that individuals carrying a certain “vulnerability” factor (temperamental, genetic) might be more at risk for developing negative outcomes when affected by an environmental stressor. Such a view considers higher susceptibility as relevant only to developing higher psychopathology. However, Belsky and Pluess (2009a) argued for a more inclusive view of the susceptibility, which emphasizes that the individual vulnerability to negative factors might in fact be more sensitive and thus more benefitting from positive environmental factors as well. In this way, the susceptibility (or, in Belsky and Pluess’ term, “plasticity”) of an individual is in fact “neutral” when it comes to outcomes and the same individuals with higher susceptibility might show worse outcomes in a risky environment as much as better outcomes in a supportive environment. The authors call this a “differential susceptibility” framework.

This is in fact an extension of the diathesis-stress model as it tests not only whether individuals with a susceptibility show more negative outcomes when exposed to an environmental stressor (such as neglectful parenting), but also whether they show better outcomes when the environmental factor is beneficial (such as supportive

parenting). These authors argue that many GxE studies modeling the interaction of genes and parental behavior have in fact been limited in their scope to the diathesis-stress model only as they mostly focused on negative effect of parenting on one pole and the absence of negative effect on the other side. In this way, by not including an interaction with a positive environmental factor (which is different than a simple absence of negative factor), it is impossible to determine whether the hypothesized GxE interaction is related to vulnerability to developing psychopathology only or whether it might refer to a higher susceptibility to both negative and positive environmental influences (Belsky & Pluess, 2009b; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & Ijzendoorn, 2011).

There have been several studies that have found support for the differential-susceptibility model. For example, study by Bakermans-Kranenburg and colleagues (2008) found that children with the 7-repeat DRD4 allele showed improved reaction (decreased externalizing behavior) to intervention focused on maternal sensitivity. Several studies have found support for the differential susceptibility model, where children's temperamental disposition was associated with more problematic behavior when family environment was adverse, and with lower levels when it was beneficial (Bakermans-Kranenburg & van IJzendoorn, 2015; Rioux, Castellanos-Ryan, Parent, & Séguin, 2016). Similarly, a recent study by Windhorst et al. (2015) on the association between maternal sensitivity and later externalizing behaviors in children found support for the GxE interaction of sensitivity with the 7-repeat DRD4 (see below) in the differential susceptibility model, where children with this polymorphism showed higher levels of externalizing behaviors when mothers used insensitive parenting and lower levels of externalizing behaviors when parenting was responsive.

Criticism of GxE research

The publication of the influential papers by Caspi and colleagues (Caspi et al., 2002, 2003) sparked a great interest in the study of GxE interactions, resulting in a vast number of studies published since then. However, it soon became clear that studies employing an interaction of a candidate gene with environmental measures were problematic. Most importantly, this type of research suffers from reproducibility problem. It was hard to accumulate enough evidence to unequivocally support the existence of almost any hypothesized GxE interaction, because for vast majority of candidate genotypes tested, the results were mixed at best, as novel studies with significant findings were oftentimes not replicated (Duncan L. & Keller, 2011). The early enthusiasm in adopting this research design was then soon followed by disappointment based on the lack of reproducibility, with many researchers arguing for the need to turn away from identifying candidate genes to larger and more robust genome-wide association studies (GWAS), for instance, as it has been argued that most of complex human behaviors are unlikely to be affected by a single gene (Thomas, 2010).

Nevertheless, the inadequate reproducibility of many candidate GxE studies was also related to methodological and statistical inadequacies and a lack of consistency across studies. For example, many studies suffered from non-representative samples, oftentimes very small, resulting in inadequate power (Duncan L. & Keller, 2011). The way the environmental measures were operationalized also differed widely, and they oftentimes lacked adequate psychometric properties. Finally, many studies suffered from inadequate statistical modeling of the interaction term, insufficient inclusion of covariates, or arbitrariness of the scales used (Dick et al., 2015; Salvatore & Dick, 2015).

Present Study

The current study focused on testing the gene-environment interaction between parental behavior in adolescence (defined as maternal closeness and child abuse) and dopaminergic genes (DRD4, DRD2) in predicting delinquency.

As shown previously, the literature on the effect of DRD4 and DRD2 and adolescent externalizing behavior is mixed. This is partially related to the uncertainty about the type of effect. Some studies showed support for the main effect of these polymorphisms, while some studies failed to do so. However, not finding a significant main effect does not preclude a significant interaction between genes and the environment. The previous GxE studies generally found support for the interaction of DRD4 and parenting in predicting externalizing behaviors in infants (Bakermans-Kranenburg et al., 2008; Bakermans-Kranenburg & van IJzendoorn, 2006b; Nikitopoulos et al., 2014; Propper et al., 2007; Sheese et al., 2007; Windhorst et al., 2015). Most of the studies done in adolescence focused on predicting substance use, with some studies finding a significant interactive effect (Vaske, Boisvert, Wright, & Beaver, 2013; Zwaluw et al., 2010), while some did not (Creemers et al., 2011, Hiemstra et al., 2013). Only a very small number of studies have focused on predicting externalizing behaviors or delinquency in adolescence. A recent study by Zandstra, Ormel, Hoekstra, and Hartman (2017) showed that adolescents with the DRD4 7R variant reported higher externalizing behaviors when affected by chronic stressors. The study by Chhangur et al. (2015) tested the DRD4 and DRD2 interaction with maladaptive parenting in predicting adolescent delinquency. They found no evidence for the DRD4 interaction with either

parental support or psychological control, but found a significant interaction of DRD2 with low parental support (unexpectedly for the A2A2 genotype).

In the current study, we wanted to test the interactive effect of the family environment and genetic susceptibility in predicting delinquency. Utilizing the Add Health dataset, this study will provide a robust test of the hypothesized interactive effect of gene x family environment in a nationally representative sample of US adolescents and young adults. Using both DRD2 and DRD4 genes that have been found to be associated with externalizing behaviors, it will provide a fuller picture of the role of dopaminergic genes in the development of delinquency.

The vast majority of GxE studies previously reviewed here have been framed by the diathesis-stress paradigm only. By including a fuller scope of parenting behaviors, ranging from adverse to lack of adverse (child abuse) and from lack of positive to positive (maternal closeness), the study seeks to test whether carrying a genetic susceptibility (defined as either DRD2 A1 allele, DRD4 7-repeat allele, or both) is associated with worse adjustment when affected by adverse parenting, as well as with better outcomes when affected by positive parenting.

An influential review by Dick et al. (2015) identified several strategies for improving the quality and reproducibility of candidate GxE studies. Informed by this review, the current study strives to provide robust test of the interactive effects of DRD4 and DRD2 and parenting in predicting delinquency in a longitudinal design.

The research questions are as follows:

1. Do the DRD4 and DRD2 polymorphisms interact with adverse or beneficial parenting in predicting rate and change of delinquent behavior in adulthood?

2. If there is a significant GxE interaction, does it follow the different susceptibility model?

Method

Sample

The data for this analysis come from National Longitudinal Study of Adolescent Health (Add Health), a nationally representative longitudinal sample of adolescents. Wave 1 was collected between 1994-95 and consisted of 20,745 adolescents in grades 7-12. The second wave was collected a year later in 1996, the third between 2001-2002 (Wave 3), and the fourth was collected in 2008 (Wave 4). In this way, the data span 14 years with four timepoints (with 1, 7, and 14 years apart from Wave 1). For the current study, we wanted to assess the association between genetic susceptibility and delinquent behavior and whether this relation might be affected by parental behavior during adolescence. For this reason, we employed parenting behavior assessed at Wave 1 (when adolescents were between 11-18 years of age at the baseline) and retrospectively at Wave 3 asking about childhood experiences (see Measures), and delinquent behavior assessed at Wave 3 (when adolescents were between 18-25 years of age).

For the DNA analyses, the biological specimens were collected during Wave 4 of Add Health data collection. From this, DNA was extracted and several candidate genes identified for the dataset. The total sample was 14,687 individuals (96% consented to participate). Due to a subsample of monozygotic twins included in the data, only one twin from the pair was selected randomly so that all genes in the sample are represented by a single copy. Given that Add Health uses weights to provide estimates that would be nationally-representative, we used the weight for Wave 3 ($N = 12,800$). However, since

the DNA collection was part of Wave 4, a number of individuals who are part of the weighted sample were not part of the DNA collection and vice versa, leading to a sample of $N = 9,086$. Finally, since the dataset includes a subsample of monozygotic twins, only one twin from the pair was selected randomly so that all genes in the sample are represented by a single copy, creating a final sample of $N = 8,932$. The weights were scaled using SUBPOPULATION option in Mplus was used to scale the weights so that they are still applicable within the selected subsample.

Measures

Age. The age of respondents. The average age at Wave 1 was 16.2 years ($SD = 1.55$).

Sex. The sex of the participants, coded as 0 = *male*, 1 = *female*.

Race. To control for potential race differences, we coded race as 0 = *White*, 1 = *Asian*, 2 = *Black/African American*, 3 = *Biracial*, 4 = *Latino/Hispanic*, and 5 = *Other*, and this was then recoded into five dummy variables.

Maternal closeness. Assessed at Wave 1 with four questions asking adolescents about the relationships with their mothers. This was rated on a 1-5 Likert-type scale, ranging from 1 = *strongly agree* to 5 = *strongly disagree*. For the purposes of the current study, this scale was reverse-coded so that higher values indicated more sensitivity. Sample item: “[Do you agree or disagree with the following statement?] Most of the time, your mother is warm and loving toward you. The full list of items is in the Appendix.

Child abuse. Assessed at Wave 3 asking participants to answer 4 retrospective items related to neglect (2 items; rated as frequency times parents left the respondent

alone home and Social Security involvement), physical abuse (1 item; rated as frequency of how many times parents slapped, hit, or kicked the respondent), and sexual abuse (1 item; rated as frequency of how many times the respondent was sexually touched or forced to engage in a sexual behavior or relation) that happened to them before the sixth grade (age of 12). The full list of items is in the Appendix.

Delinquency. Delinquency was measured at Wave 2 and Wave 3 using 10-item self-report measure asking about the frequency of certain delinquent behaviors in the past 12 months. It ranges from 0 (never) to 2 (more than once) and includes items asking about the frequency of using a weapon on someone, using hard drugs, stealing, damaging property, or hurting someone. Sample item: “In the past 12 months, how often did you deliberately damage property that didn't belong to you?” The 10-item measure was tested in a confirmatory factor analysis (CFA) and further changes were made (see Results), resulting in a final 8-item measure. See Appendix for list of items used.

Genotypes. For the DRD2, the presence of the A1 allele is of interest. For this reason, a variable was created that reflected the number of A1 variants present at either allele A or B. A total of 14,687 individuals' genotype were correctly identified for presence of either A1 or A2 variant. From this, 54.7% of individuals did not carry a single A1 allele, 37.9% carried a single A1 allele, and 7.4% carried two A1 variants on their alleles. The DRD4 is a highly polymorphic gene containing a 48-bp Variable Number Tandem Repeat (VNTR) polymorphism that can be repeated 2 to 11 times where the four repeat (4R) allele is the most common (Ptáček, Kuželová, & Stefano, 2011). For the current study, the 7R allele is of the most interest. In the current sample, the individuals were classified into three groups based on how many 7R alleles they

possessed. A total of 64.2% of the sample did not carry a single 7R allele, 31.5% of the sample possessed one 7R allele, and 4.3% of the sample possessed two 7R alleles.

Plan of Analysis

In the first step, all scales in the current study were tested in a confirmatory factor analysis to determine their psychometric validity. Then, descriptive statistics and correlations were computed to assess the relationships between the variables of interest. Then, the effects of the two types of parenting, DRD4, DRD2, and their interactions in predicting delinquency at Wave 3 were estimated in a structural model. To assess whether predictors of interest also predict change in delinquency, delinquency assessed at Wave 2 was added to the model as a predictor of the outcome. To assess whether the presence of the GxE interaction provided support for differential susceptibility rather than a diathesis-stress model, several steps were carried out, as described in Belsky, Bakermans-Kranenburg, and van IJzendoorn (2007): first, conventional statistical test of moderation was applied. Then, the susceptibility factor and the predictor should be independent, i.e., not show significant correlations because significant correlation between genetic propensity and parenting might reflect evocative rGE . Similarly, the susceptibility factor should not be correlated with the outcome; this might suggest support for diathesis-stress model only. Support for differential susceptibility would be obtained when plotting this moderation shows a cross-over interaction significantly different for the susceptibility group as opposed to a non-susceptibility group for both negative and positive environment.

To assess the interactive effect, the three-level DRD2 and DRD4 was recoded into two dummy variables each, with individuals carrying 0 candidate alleles (no A1 for

DRD2 and no 7-R or higher for DRD4) used as reference group. Such approach is an advantage over using the original three-level ordinal variable, which constrains the differences between the slopes of the levels to be equal, as well as makes all slopes cross at the same point (Swann et al., 2014). Furthermore, the analysis does not only control for main effect of background variables, but also for all interactions of DRD2, DRD4, closeness, and abuse with ethnicity and age (Dick et al., 2015; Keller, 2014).

All models used Taylor series linearization to adjust standard errors for clustering in schools and stratification by regions in the Add Health data collection. Thus, all structural models used group mean-centering. In addition, the predictor variables that were part of interactions were mean-centered. All structural models used sample weights to provide nationally-representative estimates. All models were assessed in Mplus 8 (Muthén & Muthén, 1998-2017).

Results

First, since most of the items in the Add Health dataset are not related to an existing measure, it was necessary to test the psychometric properties of the measures in the current study in the confirmatory factor analysis framework. First, parental closeness was modeled as a single factor indicated by six items. The fit of the one-factor model was not adequate, $\chi^2(9) = 1975.916, p < .001, CFI = .918, RMSEA = .106, RMSEA\ 90\%CI [.102, .110], RMSEA\ p\ close < .001$. Because items 5 and 6 are coded so that higher numbers indicate more closeness while items 1 through 4 are coded in a reverse way, a covariance was added between items 5 and 6. This led to an improved fit, $\chi^2(8) = 770.698, p < .001, CFI = .968, RMSEA = .070, RMSEA\ 90\%CI [.066, .074], RMSEA\ p\ close < .001$. Finally, a covariance was added between items 3 and 4 as they both directly

evaluate the relationship with mother ('good relationship with mother', 'good communication with mother'). The fit of this model was much improved, $\chi^2 (7) = 312.789, p < .001, CFI = .987, RMSEA = .047, RMSEA\ 90\%CI [.043, .052], RMSEA\ p\ close = .827.$

Next, the fit was tested for the supposed measure of child abuse. The fit of the 6-item model was poor, $\chi^2 (9) = 120.166, p < .001, CFI = .760, RMSEA = .029, RMSEA\ 90\%CI [.024, .033], RMSEA\ p\ close = 1.00.$ Two items related to intervention by Social Services, item 5 ('How often had Social Services investigated how you were taken care of or tried to take you out of your living situation?'), and item 6 ('how often had you actually been taken out of your living situation by Social Services') were removed because they showed very low loadings on the factor, item 5: $\lambda = .280, p < .001,$ item 6: $\lambda = .033, p = .560.$ Further, item 6 was answered only by 550 respondents.

Removing these items improved the model fit according to the additional fit indices, $\chi^2 (2) = 154.484, p < .001, CFI = .903, RMSEA = .072, RMSEA\ 90\%CI [.062, .081], RMSEA\ p\ close < .001.$ When a covariance was added based on modification indices between item 1 ('How often had your parents or other adult care-givers left you home alone when an adult should have been with you?') and item 4 ('How often had one of your parents or other adult care-givers touched you in a sexual way, forced you to touch him or her in a sexual way, or forced you to have sexual relations?'), this led to a largely improved model fit, $\chi^2 (1) = 11.096, p < .001, CFI = .994, RMSEA = .026, RMSEA\ 90\%CI [.014, .041], RMSEA\ p\ close = 1.00.$

Lastly, the measure of delinquency was tested in the CFA framework. As mentioned in the Measures section, we selected items that were common in both Wave 2

and Wave 3. This led to a selection of 10 items shared across these waves. In the first step, CFA was run to test the fit of a unidimensional model of delinquency across both Waves. Given the high skewness of the items as majority of answers to the delinquency items was *never*, we used a WLSMV estimator to treat the items as categorical indicators. The fit of the model was first tested in Wave 2 and 3 separately. The 10-item model showed an adequate fit in both waves. However, item 10 ('In the past 12 months, how often did you hurt someone badly enough to need bandages or care from a doctor or nurse?'), showed a very low loading in both waves but especially in Wave 3, $\lambda = .350$ and $\lambda = .110$, $p < .001$. Similarly, item 2 showed a very low loading at Wave 3, $\lambda = .126$, $p < .001$, suggesting that this item was no longer relevant for assessing delinquency at Wave 3. In order to control for previous levels of delinquency, it is necessary to use comparable constructs. This is why we decided to remove items 2 and 10 and continue with an 8-item scale.

This was then fitted in both waves as a configural model. Results showed a good fit across the waves, $\chi^2(95) = 1137.800$, $p < .001$, CFI = .953, RMSEA = .028, RMSEA 90%CI [.027, .030], RMSEA p close = 1.00. In the next step, we tested metric invariance across the waves by constraining the item loadings to equality. The metric model showed a significantly worse fit when compared to the configural model, $\Delta\chi^2(8) = 64.374$, $p < .001$, suggesting that the loadings significantly differed across waves. In the final step of the model fitting, we iteratively relaxed each loading to arrive at partial invariance. Partial invariance was reached when items 1 ('Pulled a knife or a gun on someone'), 5 ('Steal less than \$50'), and 9 ('Used weapon in a fight') were freed from equality constraints. This partial metric invariance model showed the following fit, $\chi^2(100) =$

1079.939, $p < .001$, CFI = .956, RMSEA = .027, RMSEA 90%CI [.025, .028], RMSEA p close = 1.00.

Next, bivariate correlation and descriptive statistics of the newly derived scales were assessed, and these are shown in Table 2. The correlations reported are polychoric correlations using latent variables whenever available, accounted for nesting and weighted. Closeness was negatively associated with abuse ($r = -.17$), delinquency at Wave 2 ($r = -.15$), and Wave 3 ($r = -.05$, all $p < .001$). Opposite pattern of associations was found for abuse, as it was positively associated with delinquency at Wave 2 ($r = .15$), and Wave 3 ($r = .23$, both $p < .001$). Delinquency at Wave 2 was significantly associated with delinquency measured 6 years later at Wave 3, $r = .33$, $p < .001$. No significant correlation was found for either DRD2 ($r = -.01$, $p = .853$), or DRD4 with delinquency ($r = -.02$, $p = .658$). Similarly, no significant correlation was found for DRD4 with either closeness ($r = -.01$, $p = .698$), or abuse ($r = -.01$, $p = .712$), and no significant association was found for DRD2 and abuse ($r = .02$, $p = .544$). However, a small negative correlation was found for DRD2 and closeness, $r = -.03$, $p = .032$. Boys were significantly more likely to be delinquent at both waves ($r = .18$ and $r = .28$, respectively), but were also more likely to report higher levels of both closeness ($r = .10$, $p < .001$) and abuse ($r = .05$, all $p < .05$). Living in a two-parent family was associated with higher closeness ($r = .07$), and less abuse ($r = -.15$, both $p < .001$) as opposed to a family with one parent. All scales showed good reliability (closeness: $\alpha = .85$, delinquency Wave 2: $\alpha = .78$, delinquency Wave 3: $\alpha = .67$) with the exception of abuse, showing an adequate reliability ($\alpha = .51$).

In the next step, these variables were used in a full structural model. Due to problems with convergence of a model using interactions with latent variables (a total of

six interactions) and outcome with categorical indicators, we decided to model the parenting behaviors (closeness, abuse) as observed variables, using their mean levels to represent them. First, delinquency at Wave 3 was regressed on delinquency at Wave 2, closeness, abuse, DRD2, DRD4, and control variables (sex, age at Wave 3, family structure, SES, and race). The fit of the structural model was adequate, $\chi^2(980) = 1355.467, p < .001$, CFI = .931, RMSEA = .007, RMSEA 90%CI [.006, .007], RMSEA p close = 1.00. All the standardized effects are shown in Table 3. Significant main effect was found for abuse, as it predicted delinquency at Wave 3 after controlling for delinquency at Wave 2 and above and beyond demographic variables, $\beta = .20, p < .001$, as well as for closeness, $\beta = -.07, p < .001$. No significant main effect was found for DRD4, either for 1 7R allele, $\beta = -.02, p = .367$, or 2 7R alleles, $\beta = -.05, p = .066$. Similarly, no effect was observed for individuals carrying one A1 DRD2 allele, $\beta = .02, p = .346$. On the other hand, a significant negative main effect was found for individuals who carried two A1 alleles, $\beta = -.05, p = .018$.

Finally, the interaction terms (i.e., DRD2/DRD4*closeness/abuse) were tested in the model. No significant interactions were found any combinations of DRD2 or DRD4 candidate alleles and closeness or abuse. The full model explained 31.7% in delinquency variance at Wave 3.

Discussion

The current study sought to assess the interaction of two genotypes (DRD2 and DRD4) and two types of parenting on predicting delinquency longitudinally in a nationally representative sample. The study provided support for the long-term effect of parenting on later delinquency found in other studies as well as brought about novel

findings related to the gene x environment interaction. The specific goals of the study were: 1) to test whether the presence of DRD2 A1 allele or long repeat DRD4 alleles interacts with maternal closeness and abuse in predicting delinquency, and 2) whether the interaction follows the diathesis-stress model when exposed to negative parenting only, or whether the genetic factors might lead to more positive outcomes when exposed to positive parenting (differential susceptibility);

First, the results showed the importance of parenting behaviors in predicting delinquency. In our study, we focused on two poles of supposed parenting spectrum – parental abuse and closeness, hypothesizing that parental abuse would serve as a risk factor for later delinquency while being close to parents should serve as protective factor. The current results confirmed these hypotheses, as parental abuse in adolescence was not only associated with adolescent delinquency, but it was even more strongly related to delinquency in adulthood seven years later. Similarly, adolescents who felt closer to their parents showed lower levels of delinquency in adolescence and in adulthood. Importantly, these main effects of parenting variables remained significant even after controlling for adolescent delinquency. In this way, negative and positive parenting were also predictive of change in delinquency from Wave 2 to Wave 3. These results support the importance of parenting closeness as a protective factor against adolescent delinquency (MacKenzie, Nicklas, Waldfogel, & Brooks-Gunn, 2012; Vieno, Nation, Pastore, & Santinello, 2009). Furthermore, they provide new evidence for its long impact beyond adolescence, as it was found to predict changes in adulthood delinquency.

On the other hand, experiencing abuse in childhood was related to more delinquent behavior in adolescence, and, more importantly, was even a stronger predictor

of later adult delinquency (Kim & Cicchetti, 2010; Manly, Kim, Rogosch, & Cicchetti, 2001). These results confirmed the previous findings of a long-term effect of victimization on later delinquent behavior (Lansford et al., 2007; Smith & Thornberry, 1995), and suggest that effects of abuse might be more detrimental to individual adjustment in the long run rather than concurrently.

Next, no main effects on delinquency was found for individuals carrying one or more 7R DRD4 polymorphisms. This is in line with previous studies that did not find main effects of these polymorphisms (Creemers et al., 2011; Hiemstra, Engels, Barker, Schayck, & Otten, 2013). On the other hand, the results of the current study showed main effect of DRD2 where individuals carrying two A1 alleles (but not one) showed lower levels of delinquency in adulthood as compared to individuals with no A1 allele. Such an effect is unexpected given the previous literature that found DRD2 to be a correlate of higher externalizing behaviors. Although this result remained significant in a full model including all covariates, it is possible that this finding might be a statistical artifact unlikely to be replicated in another study.

Regarding the association of candidate genotypes and environmental measures, DRD4 was not associated with either abuse or closeness, and no significant association was found for DRD2 and abuse. A small negative correlation was observed for DRD2 and closeness, but the interaction term was non-significant, rendering the distinction between GxE interaction and gene-environment correlation irrelevant in this case (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007).

Related to the main study hypotheses, no significant interactions were found for either DRD2 or DRD4 with either closeness or abuse. The current analysis provided a

more robust test of the potential GxE interaction by modeling the slopes of the candidate polymorphisms separately, as well as including the interactions of sex and age with GxE in the model. Furthermore, these results were obtained within a large, nationally-representative sample. These findings provide evidence of no interactive effect of either DRD2 or DRD4 and parenting, confirming some previous null findings (Chhangur et al., 2015; Creemers et al., 2011; Hiemstra et al., 2013), but in contrast to other studies that did find this effect (Bakermans-Kranenburg, IJzendoorn, Pijlman, Mesman, & Juffer, 2008; Bakermans-Kranenburg & van IJzendoorn, 2006; Guo, Roettger, & Shih, 2007; Zwaluw et al., 2010) There are several possible explanations for this discrepancy. First, most of the previous studies finding significant gene x parenting interactions were done on children, while the current study focused on adolescents. Second, although all of the cited studies used indicators of environment pertaining to parenting, their actual operationalization varied widely from study to study. Third, the dependent variable varied across the cited studies as well, with some defining it as externalizing behavior, substance use, or delinquency. For example, previous studies reporting positive interaction of DRD2 and parenting interaction used as their dependent variable novelty seeking (Keltikangas-Järvinen et al., 2009), or alcohol use (Zwaluw et al., 2010). Although oftentimes highly correlated with delinquency, it is possible that this DRD2*parenting interaction might be limited to these specific outcomes.

Finally, some of the previous studies finding a significant were mostly done with smaller convenience samples, increasing the chance that the observed interaction was due to a type I error (Dick et al., 2015; Keller, 2014). In this way, the null findings of the GxE interaction in the current study needs to be understood within the context of sample age

and variables used. It provides robust support for the conclusion that neither maternal closeness experienced in adolescence or experience with childhood abuse interacted with DRD2 A1 and DRD4 7R polymorphisms in predicting adult levels of delinquency.

Limitations and Future Directions

Given that our measure reflects a cumulative nature of child abuse (physical, sexual, neglect), it is possible that different types of abuse might be more salient or detrimental in predicting delinquency (e.g., experiencing sexual abuse might be a more prominent predictor). However, we decided to use a multiple-symptom measure of abuse, as it has been found that different types of abuse often co-occur (Arata, Langhinrichsen-Rohling, Bowers, & O’Farrill-Swails, 2005; Leitenberg, Gibson, & Novy, 2004). Future studies employing the GxE paradigm might focus on whether different types of parental abuse show different patterns or magnitudes of associations with problem behaviors, and/or whether they might be differentially conditioned by a presence of a certain candidate genetic polymorphism.

Related to this, the current study used a retrospective measure of childhood abuse, where respondents at Wave 3 were asked to report on experiences that happened to them before they were 12. Although using a retrospective measure of childhood abuse is a common method (Higgins & McCabe, 2001; MacMillan et al., 2001), it might introduce a potential bias as it relies on participants remembering experiences from more than 10 years ago for most of them (mean age at Wave 3 = 22.22 years). As suggested by Widom, Weiler, and Cottler (1999), respondents might be reluctant to disclose experiences with abuse to the researchers and those who are willing to do so might more likely to report other socially undesirable behaviors, thus potentially inflating the abuse-delinquency

link. Furthermore, this report asked respondents to assess childhood abuse before they were 12, whereas the average age at Wave 1 was 16 years. As such, the childhood abuse measure supposedly precedes Wave 1 closeness. However, we believe that experience with abuse in family would shape individuals' experience with parents in a long term and as such would serve as a conceptual antithesis to parental closeness, which was confirmed by their negative relationship in the current study.

Finally, all of the parenting and outcome measures used in the study were based on self-reports. Self-reports might introduce a common method bias to the analyses, especially in cross-sectional designs (Lindell & Whitney, 2001). Although using self-reports when reporting delinquency might be more precise than using other sources (official statistics might not capture non-criminal types of delinquency, other reporters might severely underestimate the delinquency of target participants; Thornberry & Krohn, 2000), employing e.g. parental reports of parenting in addition to child-reported ones would provide a more complex picture of child-parent relationship.

Tables and Figures

Table 2-1 *Fit Indices for the Final Versions of All Tested Models*

Type		χ^2	<i>df</i>	<i>p</i>	CFI	RMSEA	90% CI RMSEA	RMSEA <i>p</i> close
CFA	Closeness	312.789	7	<.001	.987	.047	[.043, .052]	.827
	Abuse	11.096	1	<.001	.994	.026	[.014, .041]	1.000
	Delinquency Wave 2	943.641	20	<.001	.951	.056	[.053, .059]	.001
	Delinquency Wave 3	637.060	20	<.001	.954	.045	[.042, .048]	.996
	Delinquency configural	1137.800	95	<.001	.953	.028	[.027, .030]	1.000
	Delinquency metric	1084.091	103	<.001	.956	.026	[.025, .028]	1.000
	Delinquency partial metric invariance	1072.939	100	<.001	.956	.027	[.025, .028]	1.000
SEM	Full structural model	910.021	403	<.001	.903	.012	[.011, .013]	1.000

Table 2-2 *Correlation Matrix and Descriptives of Study Variables*

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Closeness W1	1														
2. Abuse W0	-.17***	1													
3. Delinquency W2	-.15***	.15***	1												
4. Delinquency W3	-.06**	.23***	.33***	1											
5. Male ^a	.10***	.05*	.18***	.28***	1										
6. Age W3	-.08***	.02	-.04*	-.12***	.09***	1									
7. Asian ^b	-.02	.02	-.01	.01	-.01	-.02	1								
8. Biracial ^b	-.03	.05*	.02	.01	-.01	-.01	-	1							
9. Black ^b	.01	-.01	.01	-.02	.01	-.02	-	-	1						
10. Hispanic ^b	.01	.00	-.01	.00	-.01	.02	-	-	-	1					
11. Other race ^b	.02	-.01	.01	.02	-.01	.01	-	-	-	-	1				
12. SES W1	.04*	-.04	.00	.05**	.03	-.08***	-.01	-.03	.01	-.01	.01	1			
13. Two-parent family ^c	.07***	-.15***	-.07***	-.02	.02	-.02	-.01	.01	.01	-.01	-.02	.04*	1		
14. DRD2	-.03*	.02	-.01	-.01	-.01	.01	-.01	-.01	-.01	-.03	-.02	-.02	-.02	1	
15. DRD4	-.01	-.01	.01	-.02	-.01	-.01	.01	.01	.01	-.01	-.01	.01	-.03	-.01	1
<i>M / %</i>	4.38	0.54	0.11	0.06	47%	22.22	5%	13%	22%	6%	2%	5.89	63%	0.49	0.43
<i>SD</i>	0.63	0.73	0.27	0.18		1.81						1.79		0.61	0.59
<i>α</i>	.85	.51	.78	.67											

Note. W1 = measured at Wave 1; W2 = measured at Wave 2, W3 = measured at W3, W0 = retrospectively asking about experience before the age of 12
^a = reference group is female; ^b = reference group is non-Hispanic White; ^c = reference group is single-parent family.

Table 2-3 *Standardized Effects of Predictors of Delinquency at Wave 3*

Variable	β	<i>S.E.</i>	<i>p</i>
Closeness	-0.072	0.020	<.001
Abuse	0.201	0.020	<.001
Delinquency Wave 2	0.309	0.023	<.001
DRD2 A1/A2 ^a	0.015	0.016	.346
DRD2 A1/A1 ^a	-0.048	0.020	.018
DRD4 7R ^b	-0.015	0.016	.366
DRD4 7R/7R ^b	-0.048	0.026	.066
Closeness*DRD2 A1/A2	0.004	0.019	.838
Closeness*DRD2 A1/A1	0.003	0.021	.899
Abuse*DRD2 A1/A2	0.003	0.016	.837
Abuse*DRD2 A1/A1	-0.003	0.022	.894
Closeness*DRD4 7R	0.028	0.017	.098
Closeness*DRD4 7R/7R	0.030	0.021	.151
Abuse*DRD4 7R	0.010	0.017	.557
Abuse*DRD4 7R/7R	0.014	0.019	.465
Male ^c	0.336	0.016	<.001
Age Wave 3	-0.145	0.022	<.001
Asian ^d	-0.041	0.022	.060
Biracial ^d	0.003	0.024	.908
Black ^d	-0.028	0.020	.167
Hispanic ^d	-0.010	0.020	.618
Other ^d	-0.058	0.044	.193
SES	0.056	0.017	.001
Two-parent family ^e	-0.001	0.018	.968
<i>R</i> ²	0.318	0.029	<.001

Note. ^a=A1/A2 = carrying one A1 allele; A1/A1 = carrying two A1 alleles (reference group = no A1 allele); ^b = 7R = carrying one 7R allele; 7R/7R = carrying two 7R alleles (reference group = no 7R allele)

^c = reference group is female; ^d = reference group is non-Hispanic White; ^e = reference group is single-parent family. The analysis controlled for all interactions of age and ethnicity with closeness, abuse, DRD2, and DRD4 (not shown).

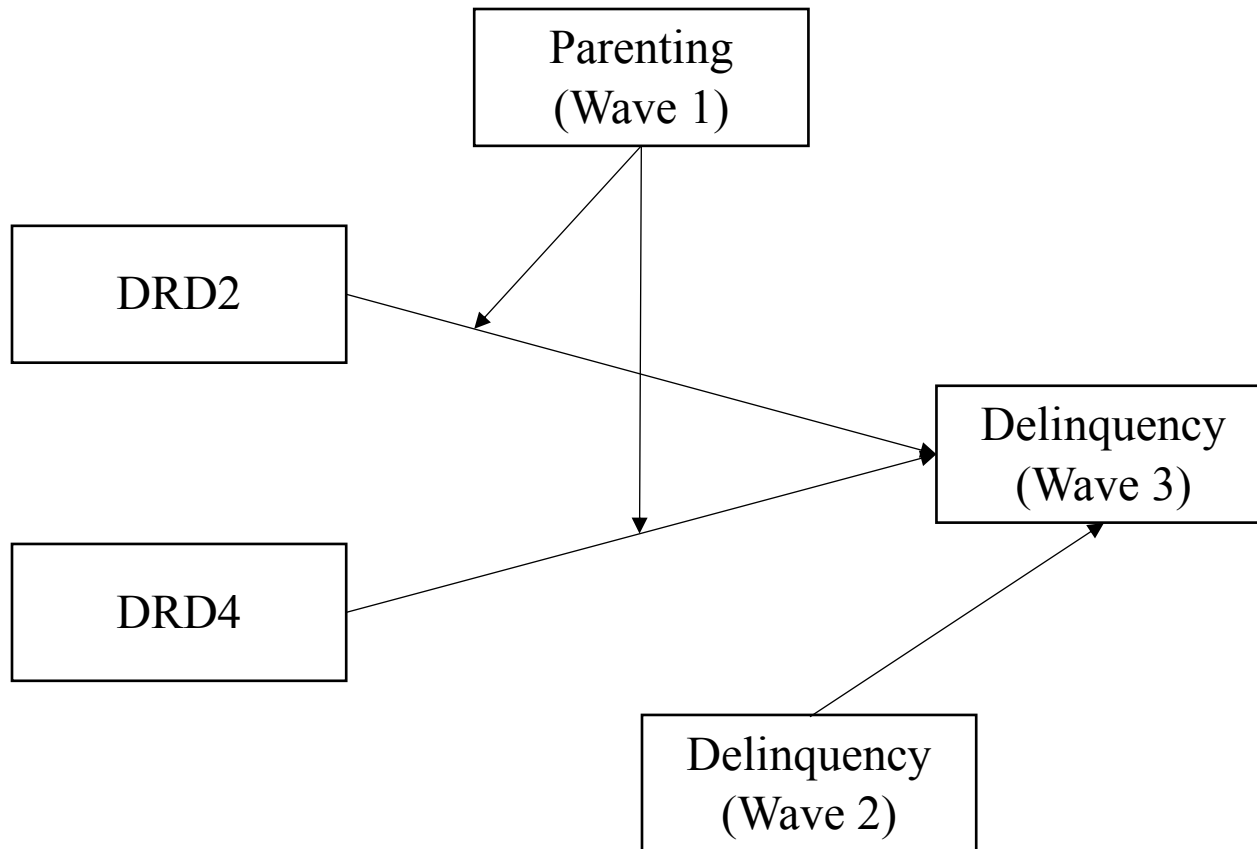


Figure 2-1 The conceptual model of the study hypotheses.

STUDY III: DO INDIVIDUAL CHARACTERISTICS PREDICT LIVING IN A
DISADVANTAGED NEIGHBORHOOD?

Abstract

A number of studies showed that neighborhoods characterized by concentrated disadvantage, i.e., neighborhoods with high residential mobility, high number of single-parent families, and high poverty have higher levels of crime and delinquent behaviors. In part, this has been explained by a process called collective efficacy, which is the ability to perform informal social control of neighborhood to prevent antisocial behavior. However, this does not explain the processes through which individuals become neighborhood residents, as individuals are not randomly allocated to neighborhoods; rather, they self-select to neighborhoods based on their individual preferences and characteristics. The current study employed a genetically-informed design to assess whether neighborhood disadvantage has a genetic component and whether this might be explained by an individual's IQ and self-control. Using a subsample of $N = 1,292$ Add Health siblings living away from parents, it found that heritability explained 31% of variance in neighborhood disadvantage, with the shared environment explaining 23% and the non-shared environment explaining 47%. The structural model found a significant negative effect of adolescent IQ on neighborhood disadvantage 14 years later ($\beta = -.05, p = .002$). This effect remained significant even when stability in neighborhood was accounted for. No significant effects were found for self-control. Subsequent analyses found that IQ explained part of the genetic variance in neighborhood disadvantage.

Introduction

Social scientists have been interested in the effects of residence on individuals since the first half of the 20th century. The Chicago school of sociology has been influential in analyzing environmental effects on individuals. The idea was that the ecology of urban spaces, i.e., the layout of the neighborhood and access to different institutions as well as opportunities might substantially affect the daily lives of its inhabitants, beyond simple aesthetics or convenience (Shaw & McKay, 1942). This paradigm soon became an important part of research in criminology. Research has provided evidence that neighborhoods with undesirable structural characteristics, such as high levels of mobility, high poverty, or high levels of single-parent families, were associated with high levels of criminal behavior in the neighborhood (Bursik & Grasmick, 1999; Sampson, 1985). Several studies postulated and found support for the idea that this association was mediated through the process of collective efficacy, which is the ability of neighborhood inhabitants to perform effective informal social control and to prevent the emergence of delinquent behaviors (Morenoff, Sampson, & Raudenbush, 2001; Sampson, Raudenbush, & Earls, 1997). When a certain neighborhood suffers from these negative characteristics, which most of the time co-occur (Wilson, 1987), the collective efficacy becomes inadequate or non-existent, and delinquent and criminal behavior can run rampant. Research on neighborhood effects has also more recently found its way to psychology, albeit the effects of neighborhood variables on individual outcomes have been found to be rather modest (Leventhal & Brooks-Gunn, 2000; Leventhal, Dupéré, & Brooks-Gunn, 2009).

The neighborhood effects have generally been considered to flow in one direction, i.e., from neighborhoods to individuals. However, there are very few studies that have hypothesized and tested the opposite, namely that individuals select their neighborhoods. Given that neighborhood variables reflect the aggregation of individual inhabitants, it seems likely that certain individual traits might predict neighborhood characteristics. Previous studies have found that certain social characteristics of individuals led to this self-selection effect (Hedman & van Ham, 2012); however, no study has assessed whether personality traits might predict neighborhood characteristics, for instance. If individual traits do in fact predict neighborhood characteristics and all psychological traits are to a certain extent heritable (Turkheimer, 2000), then it stands to reason that neighborhood characteristics will show some heritability effect as well. The current study used a genetically-informed design to assess the genetic and environmental effects on selecting neighborhoods and test whether two individual characteristics (self-control, IQ) have a longitudinal effects on this selection.

Neighborhood Effects

Criminology focuses on understanding and predicting criminal and delinquent behaviors. Rooted in sociology, it has used sociological paradigms to guide research endeavors. This traditional sociological approach has been one that emphasizes social determination of human behavior. In this view, criminal behavior is given a sociogenic explanation, which emphasizes the role of social agents (school, family, neighborhood, institutions) and social reality in general while ignoring or downplaying the importance of individual differences (Udry, 1995). A distinct tradition of criminological research was concerned with the effect of neighborhood and its characteristics on delinquency and as

such, represents a typical example of environmental explanation of human behavior (Bursik & Grasmick, 1999; Sampson, 1985).

This approach grew out of the Chicago school of sociology, which was a paradigm originating at the University of Chicago during the 1920s. Its main focus was on urban environment and its effect on individual psychology. It has been argued that the ecological effects are crucial for understanding sociological variables, especially negative outcomes, such as alcoholism, homicides, poverty, unemployment, etc. In this view, neighborhoods were the primary units of analysis and it has been argued that certain characteristics of neighborhood make individual more prone to criminal behaviors (Shaw & McKay, 1942). What is a neighborhood? A neighborhood is a subsection of a larger community. Typically, neighborhoods are operationalized using geographic boundaries defined by an administrative agency (such as Census Bureau), which equates neighborhoods with tracts or blocks (Sampson, Morenoff, & Gannon-Rowley, 2002).

When studying neighborhood effects, there are two distinct areas of research focus. The first one stems from a sociological perspective and analyzes the neighborhood effects on macro-level contextual variables, i.e., on a rate of violence within a certain location, rate of theft, rate of unemployment etc. The other, much younger research area, employs neighborhood effects as predictors of individual-level variables, focusing on outcomes such as youth externalizing and internalizing behavior, school achievement, or parental adjustment. Neighborhood research in psychology dovetailed nicely with the well-known and influential ecological theory by Bronfenbrenner (1977), which sees individuals as embedded in a series of nested ecological systems, starting from family, through school, neighborhood, city, state. According to the theory, the study of human

behavior must acknowledge that it exists within broader contexts, which affect individual's behavior as much as it affects them. However, as Bronfenbrenner's theory provides a general framework for understanding the embeddedness of individuals in their environments, it was the social disorganization theory that was the chief paradigm for understanding the process of neighborhood effects.

Social Disorganization Theory

Social disorganization theory stems from a broader criminological theory of social control. According to this theory, social groups (and society as a whole) are invested in preventing its members from committing criminal behavior, as it hurts the group as a whole. For this reason, the social group devised methods for controlling this unwanted behavior in its members. These methods are applied by socialization figures, i.e., family, community, and other authority figures.

These society effects on individuals can be direct, as in the case of punishment for infringement by socializing figures, as well as indirect, when individuals internalized these behavior restrictions. According to the theory, every individual is prone to engage in deviant behavior. It is only through bonds to society that might be severed which makes the criminal acts too costly and thus prevents crime from happening. The neighborhood process through which it controls the behavior of its members is termed collective efficacy (Morenoff et al., 2001). Collective efficacy refers to the ability of individuals sharing a neighborhood to work together to solve issues related to their neighborhood. In this way, individuals perform effective indirect social control in order to prevent neighborhoods from deteriorating. A typical example of indirect social control is when adults monitor youth loitering in the neighborhood, and are willing to confront

them when they disturb public space (Sampson et al., 1997). Well-functioning neighborhood is defined as a complex and cohesive system of social networks, rooted in the family and community (Sampson, Morenoff, & Earls, 1999).

As such, the social disorganization theory emphasized that neighborhood structural factors such as high poverty, single-parent families, residential instability, high unemployment, high number of minority inhabitants, are predictive of lower levels of neighborhood organization or the inability of the community to maintain effective social control (Sampson, 1997b; Sampson & Groves, 1989). The effect of these structural factors might then lead to alienation of the neighborhood inhabitants and low levels of investment in the community, which leads to higher social disorder and thus higher proneness to crime (Leventhal & Brooks-Gunn, 2000; Leventhal et al., 2009; Sampson & Groves, 1989).

Usually, these structural factors are highly correlated, i.e., they occur jointly for a neighborhood. In this way, neighborhoods that are poor also tend to show high African-American composition, more single-parent families, and high residential mobility. As these are all variables that have been found to be negatively predictive of individual outcomes, neighborhoods with multiple negative characteristics were referred to as neighborhoods with concentrated disadvantage. This was elaborated by Wilson (1987), who documented the increasing concentrated poverty in certain neighborhoods of American inner cities.

The support for the theory of collective efficacy in predicting delinquency was found in many studies. For example, a multilevel study by Sampson et al. (1997) found that concentrated disadvantage, immigration concentration, and residential stability

significantly predicted collective efficacy, which in turn mediated the effect of disadvantage and residential stability on several measures of violence. Similarly, Sampson and Raudenbush (1999) found that collective efficacy of a neighborhood predicted lower levels of crime and observed disorder (see also Molnar, Miller, Azrael, & Buka, 2004; Sampson, 1997b; Valasik & Barton, 2017).

On the other hand, a study by Vazsonyi, Cleveland, and Wiebe (2006) tested competing hypotheses in predicting deviant behavior. According to social disorganization theory, the level of neighborhood informal social control of individual's behavior, termed collective efficacy, is negatively associated with deviance. In other words, if the collective efficacy is low or even non-existent when neighborhood shows higher levels of social disorganization, individuals would be untethered in their delinquent tendencies, which would manifest in higher rates of deviance. A personality-based approach to predictors of deviance emphasizes individual characteristics (such as impulsivity) as instrumental for predicting deviant behaviors. The authors found that impulsivity and deviance were significantly associated, and that these results did not vary by neighborhood disadvantage, suggesting that the relationship between impulsivity and deviance was not conditioned by levels of collective efficacy.

Based on Leventhal and Brooks-Gunn's review (2000), neighborhood effects affect a plethora of individual's adjustment measures. Among them, neighborhood SES was found to positively predict educational attainment, mental health, as well as negatively predict delinquency and criminal behavior. However, neighborhood effects on individual outcomes have been found to be of a modest size in the past literature. Leventhal and Brooks-Gunn estimate that about 5% of variance in individual outcomes

can be explained by a neighborhood-level effects. However, it has been argued by some authors (Duncan G., Yeung, Brooks-Gunn, & Smith, 1998) that the negative neighborhood effect is likely to accumulate in time, and that it might be more salient for adolescents, as they spend more time away from home. Moreover, according to Leventhal and Brooks-Gunn (2000), most of the effects of neighborhoods on individuals are indirect and carried out through the following mechanisms: institutional resources (availability of schools, child cares, recreational activities, as well as employment opportunities in the community), relationships (parental characteristics and home environment, support networks available to parents), and norms/collective efficacy (how community supervises behavior of residents and the potential risks; see also Ainsworth, 2002).

Self-selection and Neighborhood

A number of studies briefly reviewed here show that neighborhoods individuals live in significantly affect their lives. However, how do individuals end up in a neighborhood? It is given that individuals are not randomly placed into neighborhoods but that they actively select their neighborhoods. If neighborhoods consist of individual inhabitants and the likelihood of individuals to live in a place is to a certain extent affected by their characteristics, then we might assume that neighborhood characteristics are affected by individual differences as well. This is referred to as 'self-selection'. The idea that a self-selection process might be taking place related to correlation between individuals' (or family's) and neighborhood characteristics is not new. In fact, the issue with non-independence of neighborhood sorting and individual's characteristics has been mentioned by several authors (Sampson & Sharkey, 2008). Mostly, however, the

individual's characteristics that were deemed to influence self-sorting into neighborhoods were of a social nature, such as being a renter vs. a homeowner, being single, or being an immigrant, to name a few (Hedman & van Ham, 2012). There is not, however, a clear understanding to the effect of self-selection on neighborhood effects. Some authors argued that self-selection leads to overestimation of neighborhood effects, while some argued that the opposite might be the case, as the individuals most affected by negative neighborhood effects are the ones most likely to move away. Some authors did not find support for neighborhood effects once the self-selection was accounted for (Oreopoulos, 2003) while others found the neighborhood effects above and beyond (Aaronson, 1998; Dawkins, Shen, & Sanchez, 2005; Galster, Marcotte, Mandell, Wolman, & Augustine, 2007).

Generally, previous studies have tried to address this problem by controlling for individual-level variables that might explain the selection process. A better yet rare method is by employing experimental design. The few experimental studies in this area found significant neighborhood effects for families that were randomly selected to move to a neighborhood with more desirable characteristics. For example, Leventhal and Brooks-Gunn (2003) reported that parents who moved to low-poverty neighborhoods reported lower distress, and their sons were reported to show lower internalizing problems. Similarly, Katz, Kling, and Liebman (2001) found that among families living in high-poverty Boston neighborhoods who were randomly selected for relocation, a significant increase was found for child health and behavior problems, as well as adult economic self-sufficiency and health (but see Kling, Liebman, & Katz, 2007 for limitations based on outcome and sex). However, existing studies have not generally

focused on psychological characteristics that might affect individual's choice of neighborhood.

Possible Genetic Influences on Neighborhood

Behavior genetic studies partition the variance in a phenotype into three sources of variance: heritability, shared environmental variance, and nonshared environmental variance. Typically, this method was used for estimating the effects of heritability and environment on psychological traits. In the past three decades, studies have consistently shown both environmental and genetic influence on cognitive abilities, psychopathology, personality traits, attitudes, or problem behaviors (Plomin et al., 2013). However, genetic effects are not only limited to individual characteristics. In fact, some supposedly environmental effects have also been found to be affected by genetics in the gene x environment (GxE) process. There are three ways these interactions can be tested: passive *rGE*, evocative *rGE*, and active *rGE* (Plomin, DeFries, & Loehlin, 1977). The passive *rGE* refers to correlations between individual characteristics and environmental measure (such as parenting), which is caused by individuals (children) sharing genes and environments with their parents. Evocative *rGE* refers to individual genetic propensities, which mold their environments. An example might be a child who shows inherent proclivity for music. This might lead to parents investing in obtaining musical instruments and even paying for music lessons, thus further enhancing this congenital fortune. Finally, active *rGE* refers to individuals actively selecting environments based on their inherent preferences (Moffitt, 2005).

The existence of genetic effects on environment indicated that our perception of genetic effects can not be confined to personality traits, but might in fact affect

environmental measures as well. This is because individuals are not randomly selected for certain environments as much as they are active agents in selecting, modifying, and adapting to the environments. This process is affected by their individual characteristics, which are themselves substantially affected by genetics. In this way, the environmental measures have been shown to indicate a significant heritability as well. A review of 55 studies by Kendler and Baker (2007) has shown that there are substantial genetic effects (average $h^2 = .27$) on measures of environment, such as parenting behaviors, stressful life events, social support, or peer interactions.

Nevertheless, there has not been a study that tested the heritability of neighborhood characteristics. Most of the genetically-informed studies on more distal environmental effects (such as schools or neighborhoods) focused on their moderating effects only. For example, Rowe, Almeida and Jacobson (1999) evaluated genetic and environmental estimates of individual aggression in different types of school. These authors found that in schools with lower levels of aggregated family warmth, the heritability of aggression was lower and shared environmental influences were higher than in schools with higher levels of family warmth. Related directly to neighborhoods, Cleveland (2003) estimated the moderating effect of neighborhood context on the heritability of aggression among adolescents. Using the Add Health data, the results showed that effects of shared environment was significant in disadvantaged neighborhoods (while non-significant in non-disadvantaged neighborhoods). This was explained as a support for the role of family processes, which should buffer the negative effect of disadvantaged neighborhoods on adolescent's aggression.

The key to understanding the hypothesis of genetic effects on neighborhood lies in the process of active *rGE*, according to which individuals ‘select’ their environments. In the case of neighborhoods, the selection process is both selection of certain neighborhood to live in as well as the variety of options determined to a certain extent by individual traits.

Consider the following example: neighborhood socioeconomic status is defined as socioeconomic status of the individual houses or their inhabitants. In other words – living in a “rich” neighborhood means living in a place with expensive houses, which can be afforded by people with significant fortune. In the context of USA, socioeconomic status is substantially correlated with level of education. Level of education is positively correlated with intelligence (L. Gottfredson, 1997a; Neisser et al., 1996; Strenze, 2007). Differences in intelligence have a large genetic component that has been found to increase with age, from 41% in childhood to 66% in older adolescence (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990; Devlin & Daniels, 1997; Haworth et al., 2010). Moreover, a more direct link between intelligence and career success, as well as intelligence and more positive outcomes in general, was also established in many studies (Caspi, Wright, Moffitt, & Silva, 1998; L. Gottfredson, 2004; Judge, Higgins, Thoresen, & Barrick, 1999; Schmidt & Hunter, 2004). Thus, it stands to reason that neighborhood socioeconomic status should have a genetic component, and individual IQ might partly explain this variance.

Another candidate personality trait, which might play a role in affecting neighborhood characteristics, is self-control. Self-control is the ability to exercise restraint in delaying immediate gratification and subduing our impulses. Probably the

most prominent theory emphasizing the role of self-control is criminological theory called General Theory of Crime (or self-control theory) by Gottfredson and Hirschi (1990). According to Gottfredson and Hirschi, all deviant and criminal behavior can be reconceptualized as a lack of self-control. A great number of studies has confirmed self-control as one of the strongest single predictors of deviant and criminal behavior (Wright, Caspi, Moffitt, & Silva, 1999; Hay, 2001; Vazsonyi, Mikuška, & Kelley, 2017). Moreover, self-control has also been found to predict many other important adjustment outcomes, such as better health, better career prospects, or less substance use (Casey et al., 2011; Mischel et al., 2011; Moffitt et al., 2011).

The paradigm of self-control theory focuses on individual differences and rejects the sociogenic theories, including social disorganization theory. In this view, the association between neighborhood disorganization and self-control would see self-control as the cause rather than the outcome, as individuals with low self-control would self-select into these neighborhoods (Evans, Cullen, Burton, & Dunaway, 1997). Given the plethora of studies finding self-control as one of key components of positive life outcomes, self-control theory would argue that divergent pathways causing individuals to end up in different neighborhoods might be due to their differences in ability to exercise restraint and delay gratifications. An identical argument was made by Caspi, Taylor, Moffitt, and Plomin (2000) with regards to lack of self-control among parents and the correlation between children's negative outcomes and neighborhood characteristics. They mentioned that "if parents' problem behaviors are passed genetically to their children, and if parents' problem behaviors interfere with their capacity to earn sufficiently to secure housing in a desirable neighborhood, this would create a correlation between

neighborhood conditions and children's behavior in the absence of any causal influence from neighborhoods” (p. 338).

The self-selection paradigm as well as the vast literature on the far-reaching effects of intelligence and self-control on life outcomes suggests that there exists strong evidence to hypothesize that individual differences might affect neighborhood membership. Given that human psychological traits are heritable to a large extent, it might be hypothesized that genetic effects affect neighborhoods as well. However, no research has taken up this challenge and tested whether neighborhoods might be heritable and what individual traits might account for this variance.

Current Study

The aim of the current study was to determine whether there is a genetic basis to the neighborhoods individuals live in. Specifically, is there a genetic basis for sorting into disadvantaged neighborhoods? Although previous studies in this area focused on predictors of neighborhood sorting, these were limited to individual choices and social characteristics. There has been no study so far that would evaluate whether individual differences can play a role in determining one’s neighborhood, a gap in the literature that this study fills. Furthermore, taking a converse perspective on the relationship between neighborhood characteristics and individual outcomes, this study also considered what individual characteristics might predict one’s future neighborhood. The estimation of genetic influences on neighborhood characteristics is possible by using a sample of siblings, including monozygotic twins, embedded in the Add Health dataset, a nationally representative longitudinal dataset. Moreover, using several waves of data, the current study considered individual predictors of subsequent neighborhood association.

The main research questions include:

1. Is there a genetic basis to neighborhood disadvantage?
2. If there is a genetic basis, which individual variables significantly predict the neighborhood disadvantage?

Method

Sample

The data for this analysis come from National Longitudinal Study of Adolescent Health (Add Health), a nationally representative longitudinal sample of adolescents. Wave 1 was collected between 1994-95 and consisted of 20,745 adolescents in grades 7-12. Add Health data collection design also included a subdataset of siblings, including oversampling of twins. Specifically, there was a total of 3,139 sibling pairs at Wave 1. This subsample of siblings does not deviate from demographic characteristics of the full sample (Jacobson & Rowe, 1999). Given that behavior genetic studies employ correlations between siblings, for the analyses of neighborhood, we will be using data from Wave 4 of the sample, as this is when most of the siblings lived apart from their parents. The analytic sample in the current study was $N = 1,292$ siblings², including 154 MZ twins, 233 DZ twins, 548 full siblings (FS), 169 half siblings (HS), 58 cousins (CO), and 130 non-related (NR) siblings.

Measures

Control variables.

Age. Given that participants show a wide range of age at each wave of data (Wave 1: age range 11-19), age was included in the predictive model as a covariate.

² For the ease of understanding, we will refer to all the pairs in the subsample as ‘siblings’ even though they might not be siblings per se (in case of cousins and non-related pairs).

Sex. The sex of the participants coded as 0 = *male*, 1 = *female*.

Race. Given the limited sample size for the biometric analyses, we recoded race into a dummy variable with 0 = *White* (reference group), 1 = *non-White*. For full structural models, we used more refined distinction with the following groups coded as dummy variables: *Asian*, *Black/African American*, *Biracial*, *Latino/Hispanic*, and *Other*.

SES. Conceptualized as the highest attained education of both parents, ranging from 1 = *8th grade or less*, to 9 = *professional training beyond 4-year college*.

Neighborhood disadvantage. Assessed at Wave 1 and 4. Measured by employing several indicators: a) the proportion of households with children, who are single-parent families, b) the proportion of households with less than \$15,000 of annual income, c) unemployment rate in bloc groups, d) proportion of households receiving public assistance, e) proportion of Black households. These indicators were provided in the U.S. Census Bureau dataset as part of the extended Add Health data. These are the indicators that were used in previous studies to measure neighborhood disadvantage (Sampson, 1997b; Sampson et al., 1999).

IQ. Assessed at Wave 1. Measured by the Peabody Vocabulary Test (PVT), an abbreviated version of the Peabody Picture Vocabulary Test – Revised. The PVT was used as a measure of IQ in previous studies (Beaver et al., 2013; Beaver & Wright, 2011; Rowe, Jacobson, & Van, 1999).

Self-control. Assessed at Wave 1. Since there was no established scale of self-control used in the Add Health data, previous researchers (Perrone, Sullivan, Pratt, & Margaryan, 2004; Wolfe & Hoffmann, 2016) proposed several items that should resemble the self-control facets as defined by Gottfredson and Hirschi (1990). However,

based on face validity, many of these previously proposed items seemed to be related to more broader adjustment problems (self-esteem, problems with friends) than to self-control per se. For this reason, we decided to conceptualize self-control as a lack of impulsivity, using the following four items: 1) When you have a problem to solve, one of the first things you do is get as many facts about the problem as possible; 2) When you are attempting to find a solution to a problem, you usually try to think of as many different ways to approach the problem as possible; 3) When making decisions, you generally use a systematic method for judging and comparing alternatives; 4) After carrying out a solution to a problem, you usually try to analyze what went right and what went wrong. All items were answered on a Likert-type scale ranging from 1 (strongly agree) to 5 (strongly disagree). The validity of this scale within the Add Health dataset was confirmed previously (Vazsonyi et al., 2006).

Plan of Analysis

First, descriptive statistics and reliabilities of study variables were computed and correlations among the variables of interest were estimated. In the next step, the twin models were estimated for the neighborhood disadvantage. The twin model estimates the relative contribution of additive genetic (A), shared environmental (C), and nonshared environmental (E) variance components using scores from siblings. The covariance between genetic sources of variance (A) was set to $r = 1.0$ for MZ twins, $r = 0.5$ for DZ twins and regular siblings, $r = .25$ for half siblings, $r = 0.125$ for cousins, and $r = 0$ for unrelated siblings. These estimates refer to the percentage of DNA that the different types of siblings share – MZ twins share 100% of their genes, DZ twins and siblings generally share 50%, and half siblings share approximately 25%. The covariance for the shared

environment (C) was set to 1.0 for siblings that were raised in the same household, and 0 for cousins. This model is shown in Figure 1.

Given that there is a significant genetic effect for neighborhood disadvantage, in the next step, candidate individual characteristics from Wave 1 were used as predictors. These include IQ, self-control, sex, age, and race. This model is shown in Figure 2. For this model, we used a sample of all individuals who indicated that they were not living with their parents at Wave 4, for an analytic sample of $N = 8,499$. In this model, neighborhood disadvantage was modeled as a latent variable using the five tract-level proportions as indicators. Moreover, low self-control was modeled as a latent variable with four items as indicators. This model also controlled for non-independence of cases due to nesting of participants in school clusters from which they were drafted, and stratification by region, using TYPE = COMPLEX option in Mplus. Neighborhood disadvantage was regressed on IQ, low self-control, parental education, age, sex, race, and two-parent family. The WLSMV estimator was used to deal with non-normal distribution of the variables. Moreover, this analysis used sample weights to provide nationally-representative estimates.

Lastly, for variables with significant predictive effect on neighborhood disadvantage, the genetic and environmental overlap between them was estimated using Cholesky decomposition. All the analyses were run in Mplus 8 (Muthén & Muthén, 1998-2017).

Results

First, neighborhood disadvantage at Wave 4 was computed as a standardized average of the following measures related to tract: proportion of Blacks, proportion of

single mothers, unemployment rate, proportion of households with income lower than \$15,000/year, and proportion of households receiving public assistance. Then, we selected only those twins where both members of the pair indicated that they were not living with their parents, creating an analytic sample of $N = 1,292$ pairs. The sibling pairs did not statistically differ based on their average age at Wave 1, sibling 1: $F(5, 1299) = 1.872, p = .096$, sibling 2: $F(5, 1300) = 1.432, p = .245$. The siblings did show significant differences in sex distribution for sibling 2 (not for sibling 1), as there were significantly more girls among cousin 2 as compared to DZ twin 2 ($\chi^2(1) = 6.680, p = .010$, and more when compared cousin 2 to first sibling 2, $\chi^2(1) = 3.990, p = .045$). There were also significant differences in proportions of non-white and white siblings, as there were significantly more non-white cousins, and half-siblings, sibling 1: $\chi^2(5) = 86.712, p < .001$, sibling 2: $\chi^2(5) = 80.018, p < .001$. There were no significant differences in age within pairs. Expectedly, there were no significant differences within pairs for race. Regarding sex differences within pairs, there were significantly more girls coded as sibling 1 among first siblings, $\chi^2(1) = 27.045, p < .001$. Thus, it was necessary to account for these demographic characteristics in all subsequent analyses. The neighborhood disadvantage index showed a good reliability for both sibling 1, $\alpha = .85$, as well as for sibling 2, $\alpha = .84$. Controlling for these characteristics in a multigroup analysis, no differences were found for neighborhood disadvantage at Wave 4 for either of the siblings, sibling 1: $\Delta\chi^2(5) = 4.894, p = .429$, sibling 2: $\Delta\chi^2(5) = 2.166, p = .826$.

The sibling intercorrelations for neighborhood disadvantage adjusted by demographic variables, are $r = .51, p < .001$ for MZ, $r = .38, p < .001$ for DZ, $r = .37, p < .001$ for FS, $r = .24, p < .001$ for HS, $r = .42, p < .001$ for CO, and $r = .33, p < .001$ for

NR. The stronger correlation for MZ twins as opposed to other types of twins suggests a genetic effect on neighborhood disadvantage; however, the differences cannot be simply explained by genetic effect, as the strength of correlation coefficients did not decline linearly with the declining genetic associations (especially the relatively high correlation for cousins). These correlations are shown in Table 1, along with basic demographics.

In the next step, the neighborhood disadvantage was assessed in a twin ACE model. The intercepts were set to equality within the pairs, but not across them. The model controlled for sex, race, and age of sibling by regressing it on the neighborhood disadvantage. The effect of sex was set to equality within sibling pairs and across sibling groups. No significant difference in model fit was found, S-B $\Delta\chi^2(12) = 10.134, p = .604$, suggesting that the association of sex and neighborhood disadvantage did not vary as a function sibling type or sibling order. The significance of the variance components h^2 , c^2 , and e^2 was assessed using bias-corrected bootstrapped confidence intervals with 5,000 bootstrapped resamplings. The results showed a significant and substantial contribution of heritability, $h^2 = .309$, BcCI [.019, .609], shared environment, $c^2 = .225$, BcCI [.064, .369], and nonshared environment $e^2 = .465$, BcCI [.297, .640] on differences in neighborhood disadvantage.³

Having established genetic effect on neighborhood disadvantage, we turned to testing individual predictors of neighborhood disadvantage in a full structural model. The model showed an adequate fit, $\chi^2(68) = 352.603, p < .001$, CFI = .985, RMSEA = .018,

³ Since it was unclear whether the cousins in the sample were growing up in different households (as might be expected) or whether they might have been living within the same household, we have also tested a model where the shared covariance for cousins was set to equal 1 (instead of 0). The variance decomposition showed relatively small change, $h^2 = .280$, BcCI [.001, .573], $c^2 = .240$ BcCI [.090, .366], $e^2 = .480$, BcCI [.304, .653].

90% CI [.016, .020], RMSEA p close = 1.00. African American ($\beta = 0.23, p < .001$), biracial participants ($\beta = 0.05, p = .002$), and Native American participants ($\beta = 0.14, p < .001$) were more likely to live in a disadvantaged neighborhood as opposed to White participants. In addition, lower neighborhood disadvantage was found for participants who grew up in a two-parent family, $\beta = -0.05, p < .001$, and who were older $\beta = -0.03, p = .045$. SES, conceptualized as highest attained parental education, was also related to lower neighborhood disadvantage, $\beta = -0.05, p < .001$. Adolescent low self-control did not emerge as a significant predictor of neighborhood disadvantage, $\beta = -0.014, p = .412$. IQ measured at Wave 1 was a significant predictor of neighborhood disadvantage 14 years later, above and beyond other predictors, $\beta = -0.05, p = .002$, suggesting that individuals with higher IQ tended to live in less disadvantaged neighborhoods.

Although the analysis already controlled for a variety of background structural variables, we decided to include neighborhood disadvantage measured at Wave 1 to assess whether there would be similarities between the neighborhood individuals grew up in and the one they moved into in adulthood. To further make sure individuals were not staying in the same house even when they indicated that they were not living with their parents, we used variable which indicates number of kilometers individuals moved from Wave 1 to Wave 4 and filtered out those respondents with zero kilometers moved. The neighborhood disadvantage at Wave 1 was again represented by a latent variable with five indicators, allowed to covary with other predictors, and predicted neighborhood disadvantage at Wave 4. The residual variances of neighborhood disadvantage indicators were allowed to covary across the two timepoints.

The direction and statistical significance of the other predictors remained unchanged (except for the effect of biracial identity, which became statistically non-significant), yet the strengths of the associations decreased due to correlation of neighborhood disadvantage at Wave 1. Neighborhood disadvantage at Wave 1 emerged as the strongest predictor, $\beta = .39, p < .001$, showing a substantial stability in the neighborhood of origin and the neighborhood one lives in in adulthood. For our predictors of interest, LSC did not predict neighborhood disadvantage, $\beta = .03, p = .167$; however, Wave 1 IQ still remained a significant predictor, $\beta = -0.05, p = .001$. All the Wave 1 variables explained 18.2% of variance in neighborhood disadvantage at Wave 4; from this, neighborhood disadvantage at Wave 1 accounted for 9.9%.

Lastly, to assess the genetic overlap between IQ and neighborhood disadvantage at Wave 4, a bivariate ACE model with Cholesky decomposition was estimated. Only c^2 emerged as a significant variance component, explaining 92.7% of variance; however, the upper bound of the CI exceeded 1, and this was the case for h^2 as well, with point estimate of 32.3% but with a BcCI of [.006, 1.168]. This is because of negative non-shared environmental covariance between IQ and neighborhood disadvantage, indicated by the standardized e path = $-.031, p = .237$. However, testing the nested submodels (CE, AE, AC) did not bring about a clearer picture of the actual decomposition. This is probably related to the modest correlation of IQ and neighborhood disadvantage, $r = -.12, p < .001$, which might be too small to be reliably decomposed with the current sample size. An alternative approach was tested where IQ was included as a predictor in the basic ACE model of neighborhood disadvantage. The variance decomposition from the original ACE model ($h^2 = .309, c^2 = .225, e^2 = .465$) changed in the following way when IQ was

added: $h^2 = .178$, $c^2 = .269$, $e^2 = .553$, showing that the heritability of neighborhood disadvantage decreased substantially when IQ was accounted for in the model. To test whether the change was statistically significant, we fixed the predictive paths of IQ to neighborhood disadvantage to zero and compared the change in model fit. The results showed that the difference was significant, S-B $\Delta\chi^2(1) = 10.203$, $p = .001$, suggesting that IQ plays a significant role in explaining heritability for neighborhood disadvantage.

Discussion

The current study aimed to assess the genetic effects on neighborhood selection in adulthood and to test candidate individual characteristics that might explain this. The following study was informed by existing studies of neighborhood effects, most prominently framed in the social disorganization theory (Leventhal & Brooks-Gunn, 2000; Sampson, 1985). However, its rationale uses a converse causality – instead of asking how disadvantaged neighborhood affect individual outcomes, it tested which individual characteristics would predict neighborhood disadvantage. The current results found support for genetic effects on neighborhood disadvantage and, using a nationally-representative sample, it also indicated that intelligence might be partially responsible for explaining this variance.

First, the study employed a classic ACE sibling model to decompose variance in neighborhood disadvantage among those siblings that were no longer living with their parents. The results showed a substantial genetic effect, as it explained 31% of variance in neighborhood disadvantage. However, the results also showed a significant effect of shared environment, explaining 23% of variance. Thus, the current results suggest that neighborhood one lives in, characterized by its level of disadvantage, is partially affected

by differences among individuals as well as by the rearing environment they grew up in. In this way, the current results corroborate and extend the existing findings on the genetic effects on environment (Kendler & Baker, 2007). The heritability estimate in the current study ($h^2 = .31$) is very similar to the average heritability ($h^2 = .27$) reported by Kendler and Baker in their review of *rGE* studies. However, this is the first study to focus on genetic influences on neighborhood.

In the next step, finding genetic effects on neighborhood disadvantage, we tested two candidate individual traits that we hypothesized to be affecting neighborhood disadvantage – IQ and self-control. In a rather conservative test, we used Wave 1 measures from adolescence to predict Wave 4 neighborhood disadvantage 14 years later. Based on the previous research finding substantial heritability for both self-control (Beaver et al., 2009), and IQ (Bouchard et al., 1990; Devlin & Daniels, 1997; Haworth et al., 2010), it was argued that individual differences in these variables would be stable and related to later life outcomes, such as neighborhood one lives in adulthood.

Regarding self-control, no significant findings emerged in the current study. Although existing literature indicates that self-control is considered a stable trait (M. Gottfredson & Hirschi, 1990; Vazsonyi & Jiskrova, 2017), which is crucial for predicting many positive life outcomes, including studies finding effects of childhood self-control on adulthood outcomes (Mischel et al., 2011; Moffitt et al., 2011). However, we found no effect of adolescent self-control on later neighborhood disadvantage. This might be due to a limited nature of self-control items available in the current study, which only referred to (a lack of) impulsivity, thus not tapping into other facets of self-control previously defined in the literature (M. Gottfredson & Hirschi, 1990).

On the other hand, our findings found support for an important role of IQ in predicting neighborhood disadvantage, showing that individuals with higher IQ lived in less disadvantaged neighborhoods. The effect of adolescent IQ on neighborhood disadvantage was significant above and beyond other factors, including parental SES, indicated as highest attained education of parents. Moreover, the effect of IQ remained unchanged even when neighborhood disadvantage at Wave 1 was added to the model. Subsequently, IQ was confirmed to be one of the phenotypic mediators of the genetic effect on neighborhood disadvantage, as controlling for IQ in the ACE models led to a significant decrease in the heritability of neighborhood disadvantage.

These results confirm the relative importance of IQ in predicting life success, a finding that has been confirmed in a plethora of other studies (L. Gottfredson, 1997b, 2004). Such an association is hardly surprising especially in the context of the United States, where intelligence is associated with level of education, which is associated with higher socioeconomic status (L. Gottfredson, 1997a; Neisser et al., 1996; Strenze, 2007). Given that intelligence is the single best predictor of educational outcomes (L. Gottfredson, 2002), individuals with higher intelligence would be more likely to attain higher education, which is then associated with higher income. Since neighborhood disadvantage is indicated by high levels of poverty, it follows that individuals with higher income will be less likely to live in these neighborhoods. Such idea reflects the cognitive sorting hypothesis, where society becomes stratified on the basis of intelligence as individuals with higher intelligence move into positions requiring high education. There, they meet other highly intelligent individuals with whom they marry and reproduce.

Given that intelligence is highly heritable, this leads to a self-reproducing caste of cognitive elite (Herrnstein & Murray, 1994).

Of course, although study findings generally align with these propositions, it is necessary to emphasize the fact that although significant, IQ explained only a very limited proportion of variance in neighborhood disadvantage. This suggests that IQ is one predictor of neighborhood disadvantage, but neither the sole nor the strongest one. Even when controlling for stability in neighborhood disadvantage, a large portion of its variance remained to be explained by other factors, be it individual or environmental ones. A substantial portion of variance (23%) in neighborhood disadvantage was due to the environment the siblings shared. This might refer to the parenting practices, parental education, family structure, but also the neighborhood in which they lived or the school they attended. These environmental effects shared by adolescent siblings growing up together were also important experiences that partially affected the neighborhood they lived in as adults.

The current results do not suggest that there are no neighborhood effects on individuals. Rather, they emphasize that the person-environment relationship is likely bidirectional (Scarr & McCartney, 1983) and that individuals play an active role in selecting as well as modifying their environments. This is not surprising given that individuals are certainly not randomly selected to occupy various neighborhoods but rather a process of self-selection takes place. However, it is necessary to mention that self-selection in this sense refers to a broader concept than simply ‘individuals making deliberate choices when deciding where to live.’ Such view would be imprecise and potentially harmful, as it might put too much emphasis on personal responsibility for

detrimental living conditions. Thus, self-selection refers to a more impersonal process where individuals with different life histories occupy different life trajectories that lead them to different places, and, in many cases, living in a certain neighborhood is not a volitional act as much as a situation that one cannot easily change. Living in a disadvantaged neighborhood is a great disadvantage to its inhabitants, but this predicament results from a nexus of complex influences, ranging from individual differences, via family influences, city and state policies, to the historical context.

Using terminology of *r*GE theory, the findings provide evidence that neighborhood self-selection encompasses not only the active *r*GE, where individuals actively seek environments that align with their individual characteristics, but also passive *r*GE as well, whereby it might be much more difficult to move into a better neighborhood for individuals with genetic risk factors already residing in a disadvantaged neighborhood. Truly, the current results attest to substantial stability in neighborhood quality, as adolescent neighborhood disadvantage at Wave 1 predicted the neighborhood in which they resided 14 years later. The neighborhood of origin explained almost 10% of variance in neighborhood individuals moved to at Wave 4, and this was after accounting for individual factors. The relative stability in individual's neighborhood characteristics might attest to the existence of an intergenerational cycle of poverty, where living in disadvantaged neighborhood provides very little opportunities for social mobility, particularly for ethnic/racial minority populations (Mayer & Jencks, 1989; South, Crowder, & Chavez, 2005).

Limitations and Future Directions

The current study includes several limitations worth mentioning. First, although all our sibling analyses controlled for the effect of sex, due to the limited sample size, we did not test for qualitative or quantitative sex differences in neighborhood disadvantage. Given that there is no comparable study of genetic effects on neighborhood, we did not hypothesize the existence of sex effects on the magnitude and type of genetic and environmental factors affecting neighborhood disadvantage. Further, in this study, the sex variable showed a uniform effect on neighborhood disadvantage, regardless of same-sex or different-sex pairs. However, future studies might benefit from explicitly testing the qualitative and quantitative sex differences for neighborhood disadvantage.

This study focused on the genetic basis of neighborhood disadvantage in the vain of the *r*GE paradigm. However, the effects of heritability might not be the same across different environments. Given that the current results show that part of the genetic basis of neighborhood is attributable to differences in IQ, some of the existing studies showed that heritability of IQ differed based on family SES, where in impoverished families, the heritability of IQ was diminished and shared environment explained the largest portion of variance, while in affluent families, IQ was mostly explained by genetic effects with little to no effect of shared environment (Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003; but see Hanscombe et al., 2012 for no moderating effect of SES on heritability). Since the sibling subsample of the Add Health data demographically matches the nationally-representative full sample, our biometric results should reflect average estimates across different SES strata. Nevertheless, future studies might explore

whether the genetic effect on neighborhood is moderated by environment individuals find themselves in.

Tables and Figures

Table 3-1 *Descriptive Statistics for Siblings Moved Away from Parents' Home*

Twin pair	<i>n</i>	<i>M</i> age at Wave 1	% female	% non-White	Correlation of neighborhood disadvantage at Wave 4	Neighborhood disadvantage reliability
MZ	154	16.23	58.4%	35.7%	.51	.89
DZ	234	16.03	49.4%	39.8%	.38	.82
FS	552	16.15	54.7%	30.0%	.37	.84
HS	171	15.90	59.4%	45.6%	.24	.84
CO	60	15.72	64.2%	88.0%	.42	.86
NR	135	15.97	52.6%	34.5%	.33	.85

Note. MZ = monozygotic twins, DZ = dizygotic twins, FS = full siblings, HS = half siblings, CO = cousins, NR = non-related pairs.
All correlations significant at $p < .001$.

Table 3-2 *Standardized Effects of Predictors of Neigh. Disadvantage (Wave 4)*

Variable	Model 1			Model 2		
	β	S.E.	<i>p</i>	β	S.E.	<i>p</i>
Male ^a	-0.015	0.014	.289	-0.009	0.014	.514
African American ^b	0.229	0.015	<.001	0.123	0.024	<.001
Asian American ^b	-0.018	0.016	.256	-0.020	0.013	.130
Biracial ^b	0.051	0.016	.002	0.031	0.016	.052
Hispanic ^b	0.016	0.014	.256	0.011	0.012	.377
Native American ^b	0.135	0.034	<.001	0.082	0.032	.011
Other ^b	-0.027	0.025	.270	-0.024	0.016	.136
Age	-0.028	0.014	.045	-0.034	0.014	.019
Two-parent family ^c W1	-0.046	0.013	<.001	-0.030	0.014	.031
SES W1	-0.054	0.013	<.001	-0.036	0.012	.004
LSC W1	-0.014	0.017	.412	0.026	0.019	.167
IQ W1	-0.048	0.015	.002	-0.053	0.017	.001
Neigh. Disadv. W1				0.390	0.030	<.001
<i>R</i> ²	.083	0.008	<.001	.182	0.021	<.001

Note. LSC = low self-control; Neigh. Disadv. W1 = Neighborhood disadvantage; W1= variable measured at Wave 1.

^a reference group = female

^b reference group = non-Hispanic White

^c reference group = single-parent family.

$r_a = 1.0$ (MZ), $r_a = 0.5$ (DZ and full sibs), $r_a = .25$ (half sibs), $r_a = .125$ (cousins), $r_a = 0$ (non-related siblings)

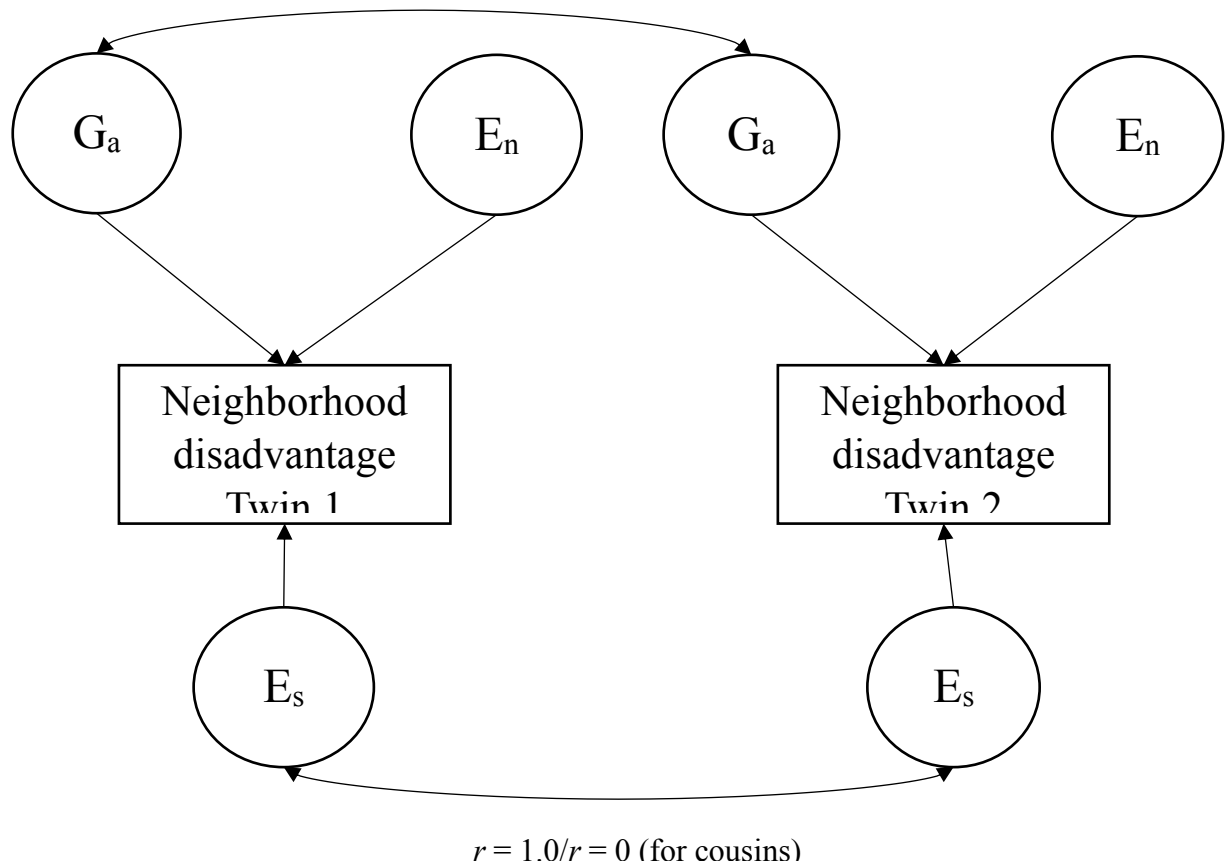


Figure 3-1 The ACE model of neighborhood disadvantage.

G_a = additive genetic variance, E_n = nonshared environmental variance, E_s = shared environmental variance. The covariance between latent variables representing genetic variance was set to $r_a = 1.0$ for MZ twins, $r_a = .5$ for DZ twins and full siblings, $r_a = 0.25$ for half siblings, $r_a = .125$ for cousins, and $r_a = 0$ for non-related pairs.

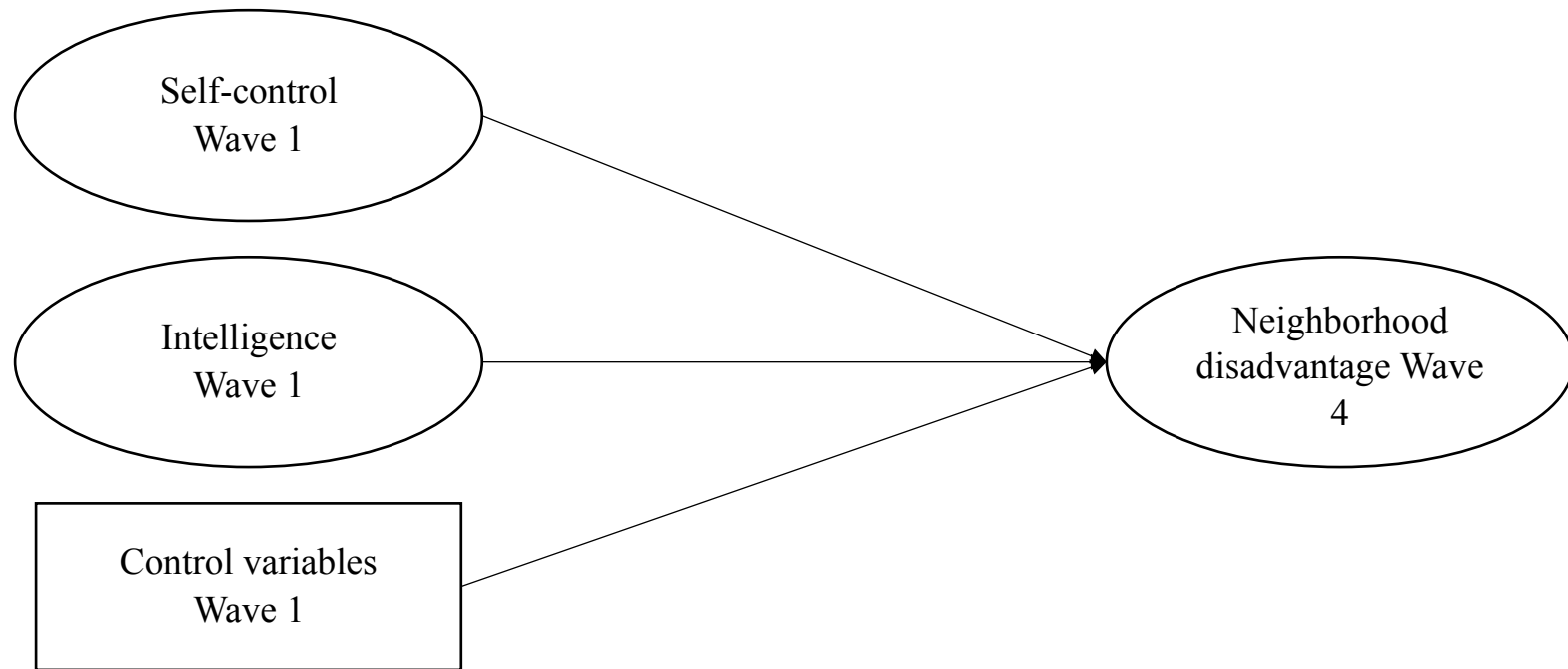


Figure 3-2 A schematic representation of the predictive model.

DISCUSSION AND CONCLUSION

The aim of the current dissertation was to employ genetically-informed designs to analyze the development of delinquency in adolescence and its correlates. The dissertation is comprised of three studies, each based on the Add Health dataset, a nationally-representative sample of US adolescents. Each study differs from the other two by its design as well as its framing within the ecological theory (Bronfenbrenner, 1977), with moving from more proximal to more distal developmental contexts from Study 1 to Study 3.

Study 1 analyzed the longitudinal development of delinquency in adolescence in a genetically-informed design. Using a sample of siblings, it looked at developmental change and stability in delinquency from Wave 1 to Wave 2 of Add Health data within three cohorts: early adolescents, middle adolescents, and late adolescents. By employing ACE models, it was possible to decompose the variance of delinquency in both waves into genetic, shared environmental, and nonshared environmental effects, as well as capture and decompose the longitudinal stability within each cohort. In this way, it was possible to compare the effect of heritability and environment on delinquency development in different adolescent cohorts.

Study 2 employs the gene x environment (GxE) paradigm and focuses on the interaction of two dopaminergic genotypes and parenting in predicting delinquency longitudinally. Specifically, the study asked whether individuals carrying the DRD4 7-repeat allele or DRD2 A1 allele would show higher rates of delinquency in adulthood (Wave 3) if they were exposed to abusive parenting in childhood, and whether they might show lower rates of delinquency if they experienced close relationships with their

mothers in adolescence (Wave 1). Informed by differential susceptibility hypothesis, this study tested whether carrying these specific polymorphisms put individuals at higher risk for negative outcomes or whether these polymorphisms might be equally related to good or bad outcomes depending on the type of environment individuals were exposed to. The study also tested whether an interaction of these candidate genes might be associated with delinquency.

Lastly, the focus of Study 3 was on neighborhood disadvantage analyzed from a gene-environment correlation (*r*GE) perspective. Living in a disadvantaged neighborhood has been found to be associated with high levels of crime and delinquency. This study focused on individual differences that might predict the type of neighborhoods individuals live in. Using a sample of adult siblings who are not living with their parents (Wave 4), the study assessed genetic and environmental effects on individual differences in neighborhood disadvantage in an ACE model. Then, it tested whether two candidate individual characteristics assessed in adolescence (Wave 1), IQ and self-control, would predict neighborhood disadvantage 14 years later (Wave 4).

The results of these studies provided novel insight into the interplay of genetic and environmental effects on delinquency and its correlates, as well as corroborated findings from previous studies. The structural models in Study 2 and Study 3 are adjusted for nesting of individuals within schools and regions and employ appropriate sample weights. In this way, these results might be considered nationally-representative.

Study 1 results showed that delinquency in adolescence had a substantial heritable component. This confirmed previous studies focusing on heritability of delinquency/externalizing behavior/antisocial behavior in adolescence (Ferguson, 2010;

Rhee & Waldman, 2002). Our cohort-based approach proved to be beneficial in identifying different patterns of genetic and environmental effects based on the cohort. Specifically, middle adolescence cohort showed a much lower heritability estimate as compared to early and late adolescence. In our sample, middle adolescence was the period when delinquency peaked, following the age-crime curve of development, with early adolescents characterized by increasing trend in delinquency, and late adolescents showing decrease in delinquency. It is possible that the high prevalence of delinquent behavior in this age might make it more salient for adolescents and, in this way, normative to a large extent. Seeing that 'everyone is doing it' lowers the threshold for engaging in delinquent or criminal acts, which, to a certain extent, lowers the genetic effect on individual differences in delinquency. The more normalized perception of delinquency in this age group is further substantiated by affiliating with delinquent peers. A plethora of studies showed that peer delinquency is one of the strongest predictors of individual delinquent behavior (Beaver et al., 2009; Haynie & Osgood, 2005). The impact of peers is the strongest in middle adolescence (Gardner & Steinberg, 2005), suggesting that the emergence of large nonshared environmental effect on variance in middle adolescent might be due to spending time with peers.

Regarding the stability of delinquency, the results showed that delinquency was moderately stable from Wave 1 to Wave 2 ($r \sim .50$ in all cohorts), and that large portion of the stability was driven by genetic influence. This was especially the case for early and late adolescents, where heritability explained 80% of the variance in stability whereas in middle adolescence, it was only 47%, again emphasizing the diminished role of heritability in explaining delinquency in this cohort. In general, the results do not lend

support for new genetic sources of delinquency emerging during adolescence (Eley et al., 2003; Van Hulle et al., 2009). Although this was a cohort-based design, it seemed that genetic effects on delinquency were stable and possibly stemming from the same source. On the other hand, the changes in delinquency (from Wave 1 to Wave 2) seemed to be entirely driven by nonshared environmental effects, especially in later age cohorts.

The results from Study 2 confirmed parenting as one of the most important environmental predictors of delinquency. Experience with parental abuse in childhood was related to higher levels of delinquency in adulthood (Keiley, Howe, Dodge, Bates, & Pettit, 2001; Kim & Cicchetti, 2010; Manly, Kim, Rogosch, & Cicchetti, 2001). Conversely, having a close relationship with mother during adolescence served as a protective factor, as it was related to lower levels of delinquency 7 years later (Berkien, Louwse, Verhulst, & van der Ende, 2012, MacKenzie, Nicklas, Waldfogel, & Brooks-Gunn, 2012, Vieno, Nation, Pastore, & Santinello, 2009). These longitudinal results are remarkable given the long span between parenting and delinquency as well as the fact that the study controlled for delinquency at Wave 2, thus essentially predicting change in delinquency from Wave 2 to Wave 3.

No interaction was found for either DRD4 or DRD2 polymorphisms with abuse. Given that abuse in childhood was hypothesized to represent negative environmental predictor of delinquency, which was confirmed in the current study, no support was found for a diathesis-stress model involving these two genotypes. Similarly, no significant interaction of DRD4 or DRD2 polymorphisms and closeness was found. Thus, the current results provide a robust and nationally-representative findings of null effects

for the proposed interaction of maternal closeness or childhood abuse with the two candidate genes.

Finally, Study 3 evaluated genetic effects on neighborhood disadvantage in a sample of siblings living away from home. Informed by existing line of research finding substantial heritability in measures of environment (Kendler & Baker, 2007), this study was the first one to focus on heritability of neighborhood. The results showed that 31% of variance in neighborhood disadvantage was due to genetic effects, with 23% due to shared environment, and 47% due to nonshared environment. In the next step, the study tested IQ and self-control as two individual characteristics, measured at Wave 1, which could account for the individual differences in neighborhood disadvantage at Wave 4. The results showed no significant effect of self-control on neighborhood disadvantage. However, a significant negative effect of adolescent IQ on neighborhood disadvantage was found, above and beyond background variables including parental education. Furthermore, the effect of IQ remained unchanged even when neighborhood disadvantage at Wave 1 was included in the model. When IQ was entered into ACE model, the heritability of delinquency significantly decreased, suggesting that heritability of neighborhood disadvantage is partly explained by IQ.

This study was one of the first to emphasize the role of individual differences in neighborhood self-selection. By finding substantial heritability of neighborhood disadvantage, it showed that individuals are not randomly selected into neighborhoods but might possess certain individual characteristics that would be predictive of the neighborhoods they would occupy in the adulthood. One of such characteristics found to be a significant longitudinal predictor was IQ. These results emphasized the relative

importance of IQ for later life success, as previous studies have found IQ to predict a plethora of outcomes (Deary, Whiteman, Starr, Whalley, & Fox, 2004; Hanscombe et al., 2012; Strenze, 2007). However, it is necessary to emphasize that the effect of IQ on neighborhood disadvantage was quite modest ($\beta = -.05$). On the other hand, the effect was still noteworthy given that it predicted neighborhood disadvantage 14 years later, and above and beyond other variables, including neighborhood of origin.

Implications

There are several important implications that can be gleaned from results of these three studies. First, there are significant genetic effects on delinquency and its development. To a large extent, these explain why people differ in their levels of delinquency as well as condition the effects of parenting on delinquency. However, the genetic effects differ with regards to age, especially in adolescence. It is also important to realize that rates of delinquency and genetic and environmental effects on differences in delinquency are not necessarily related. In fact, it has been hypothesized that genetic effect on delinquency increases as individuals age, while getting older is associated with sharp decline in delinquent behavior (Hirschi & M. Gottfredson, 1983). Moreover, the rates of crime and delinquency differ based on historical era (M. Gottfredson & Hirschi, 1986). One might actually argue that the proportion of genetic effects and rates of delinquency might be inversely related, as it has been found that shared environmental effects explain more differences in delinquency among individuals from low SES environments, which are associated with higher rates (Tuvblad, Grann, & Lichtenstein, 2006).

The current dissertation has demonstrated the importance of genes on development of delinquency in adolescence, on the association between parenting and delinquency, as well as on the neighborhood one lives in. Undoubtedly, differences in delinquent behaviors among humans are affected by genes to a certain extent. Certain individual characteristics inherited from parents provide wide boundaries for their development as well as condition their experiences. In this sense, some individuals are more prone to delinquent or criminal behavior. However, such statement is a far cry from a genetic determinism, which is oftentimes wrongly assumed when speaking about genetic influences. Complex psychological phenomena such as delinquency are not 'set in stone' but are highly malleable even within the context of genetic influence, as this dissertation attested to. The finding of genetic effects on delinquency does not in any way invalidate efforts, be it parental or intervention-based, focusing on reducing delinquent behaviors. Given that heritability estimates include genetic-environmental correlations (r_{GE}), it is essential to focus these efforts especially on individuals who might be genetically at risk for developing delinquency. Understanding the additive as well interactive effects of genetic and environmental influences on delinquency provides us with un-biased estimates of environmental effects on this behavior, which is vital for planning truly effective intervention efforts. Incorporating findings from behavior genetics enables practitioners to recognize early signs of inherent risk propensities for delinquency. Knowing that a substantial portion of delinquency development is affected by the environment, including parental practices, intervention efforts need to take into account the environmental risks, which can activate or exacerbate the inherited liabilities.

APPENDIX

Delinquency Items

During the past 12 months, how often did the following happen?

1. *You pulled a knife or a gun on someone (never-once-more than once)*
2. *You shot or stabbed someone (never-once-more than once)*
3. Have you ever used weapon in a fight? (no-yes)

Response categories for the following items:

never – 1 or 2 times – 3 or 4 times -5 or more times

4. How often did you drive a car without the owner's permission?
5. *How often did you go into a house or building to steal something?*
6. *How often did you sell marijuana or other drugs?*
7. *How often did you steal something worth less than \$50?*
8. How often did you take something from a store without paying for it?
9. *How often did you use or threaten to use a weapon to get something from someone?*
10. How often were you loud, rowdy, or unruly in a public place?
11. *How often did you deliberately damage property that didn't belong to you?*
12. How often did you hurt someone badly enough to need bandages or care from a doctor or nurse?
13. How often did you paint graffiti or signs on someone else's property or in a public place?
14. *How often did you steal something worth more than \$50?*
15. How often did you take part in a fight where a group of your friends was against another group?

Note. Italicized items were used for measuring delinquency in Study 2.

Closeness Items

Do you agree or disagree with the following statements?

Response categories: Strongly agree – agree – neither agree nor disagree – disagree – strongly disagree

1. Most of the time, your mother is warm and loving toward you.
2. When you do something wrong that is important, your mother talks about it with you and helps you to understand why it is wrong.
3. You are satisfied with the way your mother and you communicate with each other.
4. Overall, you are satisfied with your relationship with your mother.

Response categories: Not at all – very little – somewhat – quite a bit – very much

5. How close do you feel to your mother?
6. How much do you think she [resident mother] cares about you?

Child Abuse Items

Response categories: One time – two times – three to five times – six to ten times – more than ten times

1. By the time you started 6th grade, how often had your parents or other adult care-givers left you home alone when an adult should have been with you?
2. How often had your parents or other adult care-givers not taken care of your basic needs, such as keeping you clean or providing food or clothing?
3. How often had your parents or other adult care-givers slapped, hit, or kicked you?
4. How often had one of your parents or other adult care-givers touched you in a sexual way, forced you to touch him or her in a sexual way, or forced you to have sexual relations?
5. How often had Social Services investigated how you were taken care of or tried to take you out of your living situation? (count)
6. How often had you actually been taken out of your living situation by Social Services? (count)

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- Zandstra, A. R. E., Ormel, J., Hoekstra, P. J., & Hartman, C. A. (2017). Chronic stressors and adolescents' externalizing problems: Genetic moderation by dopamine receptor D4. The TRAILS study. *Journal of Abnormal Child Psychology*, 1–10. <https://doi.org/10.1007/s10802-017-0279-4>
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VITA

EDUCATION

- 2013 M.S., Psychology
Masaryk University, Brno, Czech Republic
- 2010 B.A., Psychology, Minor: Journalism
Masaryk University, Brno, Czech Republic
- 2010 B.A., English Language and Literature
Masaryk University, Brno, Czech Republic

RESEARCH EXPERIENCE

- University of Kentucky**, Lexington, KY
Research Assistant, August 2013 – present
Masaryk University, Brno, Czech Republic
Research Assistant, January 2013 – June 2013
Masaryk University, Brno, Czech Republic
Research Assistant, June 2010 – June 2011

HONORS & AWARDS

- August 2017 – *Selected for EADP/EARA/SRA Summer School 2017*
Selected as one of eight US participants to attend an intensive workshop in Utrecht, the Netherlands
- May 2015 – *Faculty Fellows at Presentation U, University of Kentucky - \$2,500*
Competitive program offering funding and providing training for selected faculty in implementation of innovative teaching methods (3 semesters)
- May 2015 – *School of Human Environmental Sciences Recognition of Accomplishment*
Recognition for receiving the SRA Emerging Scholar Grant
- May 2015 – *School of Human Environmental Sciences Graduate Student of Distinction*
Recognition for research, teaching, and service activities
- July 2014 – *Society for Research on Adolescence Emerging Scholar Small Grant – \$3,500*
A grant awarded to a winning project on analyzing Department of Education data on racial disparities in US schools
- May 2014 – *Lyman T. Johnson Diversity Fellowship, University of Kentucky*
Competitive one-year fellowship for fostering diversity among graduate students awarded by the Graduate School, University of Kentucky, Lexington, KY, USA

SELECTED PEER REVIEWED PUBLICATIONS

- Vazsonyi, A. T., & **Ksinan, A. J.** (2017). Understanding deviance through the dual systems model: Converging evidence for criminology and developmental sciences. *Personality and Individual Differences, 111*, 58-64. doi: 10.1016/j.paid.2017.
- Ksinan, A. J.** & Vazsonyi, A.T. (2016). Longitudinal associations between parental monitoring discrepancy and delinquency: An application of the Latent Congruence Model. *Journal of Youth and Adolescence, 45*, 2369-2386. doi: 10.1007/s10964-016-0512-4
- Vazsonyi, A.T., Ksinan Jiskrova, G., Kelley, E., & **Ksinan, A. J.** (2016). Online and offline bullying perpetration in a rural development context: The impact by social media use. *Journal of Rural Social Sciences, 31*, 86-106.
- Ksinan, A. J.** & Vazsonyi, A. T. (2016). Narcissism, Internet, and social relations: A study of two tales. *Personality and Individual Differences, 94*, 118-123. doi: 10.1016/j.paid.2016.01.016
- Vazsonyi, A. T., Ksinan Jiskrova, G., & **Ksinan, A.**, & Blatný, M. (2016). An empirical test of self-control theory in Roma adolescents. *Journal of Criminal Justice, 44*, 66-76. doi:10.1016/j.jcrimjus.2015.12.004
- Vazsonyi, A. T., **Ksinan, A.**, Mikuska, J., & Jiskrova, A. (2015) The Big Five and adolescent adjustment: An empirical test across six cultures. *Personality and Individual Differences, 83*, 234-244. doi: 10.1016/j.paid.2015.03.049

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