GENE-ENVIRONMENT INTERACTIONS AND CRIMINOLOGICAL THEORY: TOWARD

A MORE SYSTEMATIC UNION

by

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(Under the Direction of Thomas L. McNulty)

ABSTRACT

This dissertation focuses on integrating several popular theories of crime into a biosocial

framework that accounts for recent research in the gene-environment interaction (GxE) literature.

The three theories I specifically focus on are self-control theory, general strain theory (GST), and

social learning theory. Regression analyses conducted using data from the National Longitudinal

Study of Adolescent Health (Add Health) demonstrate the utility of combining these

criminological theories with a GxE modeling approach. These analyses reveal several important

findings. First, in the analysis based on self-control theory, MAOA and DAT1 genotype

moderate the effect of the parent-child relationship on both low self-control and criminal

behavior. Specifically, those in the sample who carry so-called "plasticity alleles" for both

MAOA and DAT1 are more vulnerable the negative effects of parenting as it relates to self-

control and criminal behavior than are those in the sample who do not carry plasticity alleles for

either of these genes, demonstrating a significant a GxE. Secondly, the analyses based on GST

reveal that those in the sample that are homozygous for the s-allele of 5-HTTLPR (s/s) are more vulnerable to the negative effects of the attempted or actual suicide of friends and family during adolescence in regards to their levels of depressive symptoms and criminal behavior than are those in the sample that carry other allelic variations for 5-HTTLPR (s/l, l/s, and l/l). Finally, the analysis focusing on social learning theory shows that the effect of affiliations with delinquent peers on one's own criminal behavior is greater among those individuals who are homozygous for the 10R allele of DAT1 (10R/10R) than among those who carry no 10R DAT1 alleles. These results represent a contribution to the evolving field of biosocial criminology, and call for more theorizing and research of this type. Suggested directions for future research stemming from this project are also discussed.

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DEDICATION

This is for Kristine and Graham.

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CHAPTER 1

INTRODUCTION AND LITERATURE REVIEW

The prevailing approaches to explaining deviant and criminal behavior in the sociological and criminological literature have focused almost exclusively on the social environment, with little attention given to the potential role of genetics in crime causation. This is mostly due to the strong emphasis on the social environment in both of these fields, but it is also partly due to the controversial history of biological explanations for crime and deviance and their legacy of racism and eugenics (Akers and Sellers 2009). Recent advancements in technology, however, have allowed geneticists to map the human genome, and in the years since we have learned much about how certain genes are associated with a number of behaviors and psychological conditions of interest to sociologists and criminologists. These behaviors and psychological conditions include, but are not limited to, aggression, depression, risky sexual behavior, and substance abuse (Walsh and Beaver 2009). At the same time, we have learned that much, if not most, of what makes these genes express themselves is found in the social and physical environment (for a review, see Belsky and Pluess 2009). It has become a generally accepted fact in many scientific disciplines that neither genetics nor the social and physical environment is solely responsible for variations in behavior. Arguments concerning nature versus nurture have largely been replaced with an emphasis on nature *via* nurture.

Unfortunately, traditional sociological and criminological theorizing has been slow to incorporate these findings concerning genetics into more comprehensive, biosocial theories of crime and deviance (for exceptions, see Beaver et al. 2011; Guo et al. 2008; and Simons et al.

2011). This is despite (or in spite of) the well-documented evidence that has accrued over the last decade as to the salience of gene-environment interactions (GxE), which are the observable characteristics or traits of individuals that result from the interaction of genetic propensities with the social and physical environment. With this dissertation I will attempt to bridge the gap between traditional theories of crime and deviance and gene-environment interaction studies. I will accomplish this by testing in separate analyses biosocial models of criminal behavior that combine three time-tested and highly popular theories of crime and delinquency, self-control, general strain, and social learning theory, with a GxE approach to statistical modeling.

In the following sections I will do several things. First, I will briefly discuss how genes affect behavior in general, and then I will discuss how they affect behavior in combination with the environment (GxE). Second, I will discuss how three particular genes (MAOA, 5-HTTLPR, and DAT1) that I will focus on in my analyses affect behavior directly and in combination with the environment. Finally, I will briefly describe the three criminological theories I will be testing, and why I think they are well-suited to integration into a biosocial framework, namely due to how well they fit prior research in the GxE literature as explanations of the relationship between genetics, the social environment, and criminal and deviant behaviors.

GENES, ENVIRONMENT, AND BEHAVIOR

We have recently learned a lot about how genes help shape behavior. In this section I discuss how genes affect our behavior directly, in combination with the environment (GxE), and I discuss the three specific genes that will be the focus of the analyses in this dissertation. I will first offer a very brief discussion of how genes directly affect human behavior.

Direct Genetic Effects on Behavior

The completion of the mapping of the human genome, formally called the Human Genome Project, was a significant breakthrough in the history of science. This project has provided data for researchers that have allowed them to study the potential genetic origins of behavioral disorders, mental illness, and terminal diseases, among others. Indeed, in a relatively short time span, molecular genetics research has discovered specific genes linked to many disorders, including anorexia and bulimia, alcoholism, ADHD, and delinquency.

Genes can directly affect a phenotype, which is an observable characteristic or trait of an organism, in three main ways. First, a single gene can be responsible for the development of a single disease, personality trait, or some other observable characteristic. More than 1,200 diseases are known to be caused by a single gene (Wilson 1998). These one-to-one correspondences between a specific gene and a phenotype are known by the acronym OGOD (one gene, one disorder). When more than one gene affects the development of a characteristic or trait, that characteristic or trait is said to be polygenic. Polygenic effects are the second main way that genes can directly shape a phenotype. For one example, attention deficit hyperactivity disorder (ADHD) has been linked to numerous genes, such as DRD4 and DAT1, suggesting that ADHD is polygenic (Barr et al. 2000; Gill et al. 1997). Lastly, genes can directly affect phenotypes through what are called pleiotropic effects. In this case, a single gene can have multiple effects that span various phenotypes.

Of more concern to sociologists of crime, however, is the literature concerning how one's genotype moderates how they perceive and react to their environment. This gene-environment interaction (GxE) literature is next up for discussion.

Gene-Environment Interaction (GxE)

Genetic forces influence any number of diseases, personality traits, and behavioral patterns. Most phenotypes, however, are not the result of just one gene, or even just the result of only genetic influence. There is good reason to believe that most phenotypic variation is due to a complex web of genetic and environmental influences acting independently and interactively (Plomin, Owen, and McGuffin 1994). The most innovative scientific research of the last ten years has left behind the nature versus nurture debate to focus on probing the interplay between genetics and the physical and social environment (Belsky and Pluess 2009; Caspi et al. 2002, Caspi et al. 2003, Moffitt 2005). This focus on gene-environment interplay has slowly started to gain more attention from sociologists and criminologists, as the added value of these types of biosocial models for explaining various social behaviors has started to become clear in research done in other scientific fields.

This line of research has been predominantly focused on one particular type of gene-environment interplay, gene X environment interactions. A gene X environment interaction, or GxE, can be defined as a gene helping cause the development of a phenotype only when the person is confronted with certain environmental conditions (Moffitt 2005; Walsh 2002). To put it another way, the effect of a particular variant of a gene is contingent on specific environmental stimuli, and vice versa. Without the environmental stimulus, the genetic effect remains mute.

GxEs are an important determinant of behavior that sociologists and criminologists are slowly becoming more aware of and open to integrating into models that emphasize traditional sociological and criminological theories and variables. Taking into account the way that gene-environment interplay effects behavior is important for social scientists because it can potentially allow us to explain why similar experiences across individuals result in dramatically different

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behavioral outcomes. Similar environments and experiences may be experienced very differently depending on a person's age, genetic makeup, and other qualities that vary across individuals (Turkheimer and Waldron 2000). Different behavioral outcomes across individuals experiencing similar environmental events may simply reflect the fact that different individuals have different genotypes, and these different genotypes differentially shape reactions to similar environments and events.

How exactly does one's genetic makeup cause them to react differently to their environment than a person in a similar environment would? Belsky and Pluess (2009) have observed that the genes most commonly observed in the GxE literature are related to the dopaminergic and serotonergic systems, with the dopaminergic system implicated in reward sensitivity and the serotonergic system implicated in sensitivity to punishment and displeasure (Simons et al. 2011). This leads them to posit that some individuals are more responsive to their environment because of their different thresholds for experiencing pleasure or displeasure, meaning that because of their genetic makeup these individuals are more readily shaped by environmental rewards and punishments than are others.

Next, I will discuss three genes in particular, MAOA, 5-HTTLPR, and DAT1, that will be the focus of the genetic portion of this dissertation. For each gene I will discuss its general functionality, its direct effects on behavior, and the existing evidence pointing toward an interaction between the gene and the environment. I will begin by discussing MAOA.

Monoamine Oxidase A (MAOA)

Monoamine oxidase A (MAOA) is one of the most studied enzymes in the human body, and perhaps one of the most important. MAOA is responsible for the breakdown of several different neurotransmitters, including dopamine, serotonin, and norepinephrine, all of which

have been related in past research to a variety of antisocial behaviors (Caspi et al. 2002; Rowe 2002). Levels of these neurotransmitters in the brain depend on MAOA activity levels. When MAOA is overactive, the levels of these neurotransmitters can decrease tremendously, while under-active MAOA activity can cause neurotransmitter levels to rise rapidly. Given how important MAOA is in the regulation of neurotransmitters, it is not surprising that the monoamine oxidase A promoter gene, which controls the production of MAOA, has been widely studied. Research has thus far identified this gene as a strong candidate in the etiology of antisocial behaviors (Caspi et al. 2002; Edwards et al. 2010; Fergusson 2011; Foley et al. 2004; Kim-Cohen et al. 2006).

While the main function of MAOA is to break down and discard neurotransmitters, it also plays an important role in the regulation of brain activity. A class of antidepressant medications, called MAOA inhibitors, are often prescribed for depression and related mood disorders. These drugs work to reduce symptoms of depression by blunting MAOA activity levels, thus causing an increase in levels of neurotransmitters whose presence reduce depressive symptoms (Catalano 1999). Due to the control that MAOA exerts over neurotransmitter levels and the use of antidepressants that decrease MAOA activity, geneticists have believed for some time that MAOA may be involved in other important phenotypes, including violent and aggressive behaviors (Shih, Chen, and Ridd 1999). Research into the genetic basis of criminality looking specifically at MAOA has recently begun to focus on a polymorphism in the promoter region of the gene.

The monoamine oxidase A gene has been mapped to the X chromosome and contains a 30 base-pair variable number of tandem repeats upstream in the 5' regulatory segment of the gene (Sabol, Hu, and Hamer 1998). This functional polymorphism affects the gene's

transcriptional efficiency with different alleles corresponding to different levels of MAOA enzyme activity (Ito et al. 2003; Sabol, Hu, and Hamer 1998). The 2R (repeat) and 3R alleles have been shown to be the least efficient at transcription, while the 3.5R, 4R, and 5R alleles are the most efficient (Deckert et al. 1999). The 2R and 3R alleles are usually grouped together and described as "low-activity" while the 3.5R, 4R, and 5R alleles are usually grouped together and described as "high-activity" (Caspi et al. 2002). The low-activity alleles (2R and 3R) are the ones typically associated with antisocial behavior (Caspi et al. 2002), and the 3R allele (along with the 4R allele) is the most common MAOA variant in the human population (Deckert et al. 1999; Sabol, Hu, and Hamer 1998).

The location of MAOA on the X chromosome has meant that most of the research looking at the effects of this gene has focused on males (Beaver et al. 2010; Caspi et al. 2002). Since MAOA is located on the X chromosome, male have only one copy (or one allele), while females have two copies (two alleles). That females have an extra copy of the MAOA gene means that it affects males and females very differently. If a male has a defective MAOA allele then they are unable to manufacture a functional MAOA enzyme, but a female will not have this problem so long as at least one of the copies of the gene she carries is not defective. MAOA is thus considered a potentially problematic gene for males, but not generally for females (Caspi et al. 2002; Simons et al. 2011).

The MAOA gene has been one of the more widely studied genes in the GxE literature. A seminal article in the GxE literature by Caspi and his colleagues (Caspi et al. 2002) focused on MAOA genotype and childhood experiences of abuse and neglect. Caspi et al. (2002) hypothesized that a relationship between MAOA and child abuse might exist when noting that the literature on antisocial behavior has tied both the low-activity MAOA allele and early

experiences of abuse and neglect to this developmental outcome. They hypothesized that the effects of maltreatment on antisocial behaviors may be moderated by MAOA genotype, and this is precisely what they found in their research carried out on a New Zealand birth cohort followed into early adulthood. The individuals in this all male sample that carried the low-activity version of the MAOA gene were shown to be the most violence prone when they had been subjected to childhood maltreatment. For those individuals who carried the high-activity version of the MAOA gene a much smaller effect of childhood maltreatment on later violence emerged. In one of a number of studies that attempted to replicate Caspi et al. (2002), Kim-Cohen and colleagues (Kim-Cohen et al. 2006) found that boys aged 7 with the low-activity MAOA allele were rated by both mothers and teachers as having more mental health problems, specifically symptoms of attention deficit/hyperactivity disorder (ADHD), if they had been victims of abuse than similar boys with the high-activity allele.

In another study, Foley et al. (2004) found that boys with the low-activity version of MAOA were more likely than boys carrying the high-activity version to be diagnosed with conduct disorder if exposed to high levels of childhood adversity in a large longitudinal study of adolescent twins aged 8 to 17 years. Nilsson et al. (2006) produced similar results in a cross sectional investigation of adolescent boys looking at maltreatment and a composite measure of criminal behavior that included vandalism, violence, and stealing. Edwards et al. (2010) found that carrying the low-activity MAOA allele moderated the effects of physical discipline on teacher, maternal, and self-report measures of delinquent behavior in a sample of white males. Also using a sample of white males, Beaver et al. (2010) found that MAOA genotype moderated the effect of neuropsychological functioning (as measured by verbal ability) on levels of both self-control and self-reported delinquency.

To conclude, the monoamine oxidase A promoter gene is an important and widely studied gene that is responsible for breaking down neurotransmitters, most notably dopamine and serotonin. It has been one of the most widely studied genes in the GxE literature, and many different studies with very different kinds of samples and research designs have shown that the low-activity variant of the MAOA gene is a risk factor for various antisocial behaviors and neurological deficits when it is paired with highly negative environmental stimuli. In the next section I will discuss a key gene in the serotonergic system, the serotonin transporter gene.

The Serotonin-Transporter-Linked Polymorphic Region (5HTT-LPR)

Perhaps the most studied gene in the growing GxE literature is the serotonin transporter gene (5HTT). This gene is located at 17q11.1-17q2 on chromosome 17 (Gelernter, Pakstis, and Kidd 1995; Heils et al. 1996). Research has focused on the serotonin-transporter-linked polymorphic region (5-HTTLPR), a degenerate repeat polymorphic region in SLC6A4 (Belsky and Pluess 2009). While many variants of 5-HTTLPR exist (Nakamura et al. 2000), most research has focused on two variants, the short variant (i.e., the s-allele) and the long variant (i.e., the l-allele). 5-HTTLPR is interesting in that the s- and l-alleles have different functional properties, which has interested geneticists. The main function of the serotonin transporter gene is to synthesize the serotonin transporter protein. This protein is responsible for ceasing serotonergic activity in the brain by removing excess serotonin from the synaptic cleft and returning it to presynaptic neurons. This process is known as reuptake, and it is important for maintaining appropriate levels of serotonin. Evidence has thus far suggested that the s-allele, compared to the l-allele, has lower transcriptional activity and is linked to reduced serotonin receptor binding in the brain (David et al. 2005; Heils et al. 1996). Due to this, the s-allele has

been linked both directly and indirectly to various behavioral and mood disorders, most notably depression.

The research on pharmacological drugs has pointed to 5-HTTLPR as being a candidate gene for behavioral and mood disorders, as well as other psychiatric problems (Niehoff 1999; Rowe 2002). Low serotonin levels contribute to a number of disorders, including depression, so a whole category of drugs have been developed to increase serotonin activity in the brain by specifically targeting the 5-HTT gene. These medications are known as selective serotonin reuptake inhibitors (SSRIs), and they function by altering the reuptake process by blocking the serotonin transporter protein from removing serotonin from the synaptic cleft, thus increasing serotonin levels (Niehoff 1999). By increasing serotonin levels in the brain, SSRIs can effectively alleviate or erase altogether the symptoms associated with depression and other psychological disorders. The existence of these medications and their implication of 5-HTTLPR in a range of disorders has suggested the possibility that the s-allele of this gene may be etiologically related to antisocial behavior (Neihoff 1999; Rowe 2002).

A wealth of studies in the GxE literature have identified 5-HTTLPR, and specifically the s-allele, as being involved in the development of both psychological and behavioral disorders. In another ground breaking GxE study, Caspi et al. (2003) were the first to show the moderating effects of 5-HTTLPR on life stressors. Those carrying two s-alleles were the most vulnerable to the effects of life stressors while those carrying two l-alleles were the least vulnerable when the outcomes of interest were depressive symptoms, major depressive episodes, and suicidal ideation and attempts at age 26 years. Numerous other studies have found similar results looking at 5-HTTLPR and depression. These include Eley et al.'s (2004) study of adolescent girls who were and weren't exposed to risky family environments, Taylor et al.'s (2006) study of young adults

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who had and hadn't been exposed to early adversity (operationalized as a problematic child-rearing history), and Brummett et al.'s (2008) research of middle-aged and aging adults who had and hadn't serve as the primary caregiver of a relative with Alzheimer's disease.

The moderating effect of 5-HTTLPR on environmental influences is not limited to depression and its symptoms. This effect has also emerged in studies of anxiety (Gunthert et al. 2007; Stein, Schork, and Gelernter 2008) and ADHD (Retz et al. 2008). Of the most interest, however, to sociologists concerned with crime and deviance are the recent findings concerning 5-HTTLPR and criminal and deviant behaviors. Retz et al. (2004) found a direct effect of 5-HTTLPR on violent behavior, observing that 5% of the variance in violence history was explained by the presence of the s-allele alone in a clinical sample of white males. In a sample of Swedish adolescents Nilsson et al. (2005) found that the s-allele of 5-HTTLPR interacted with poor family relations to increase alcohol consumption; this association did not exist for carriers of the l-allele. In another sample of Swedish adolescents, Aslund et al. (2012) found that low family SES interacted with 5-HTTLPR in a manner similar to stressful life events to predict increased involvement in juvenile delinquency. Simons et al. (2011) showed in a sample of African Americans that 5-HTTLPR genotype (along with DRD4 and MAOA genotype) moderated the effects of a composite measure of the hostility of one's environment, such that those carrying the s-allele were more likely to adopt antisocial attitudes and engage in aggressive behavior when faced with environmental adversity than those homozygous for the l-allele.

To conclude, the serotonin-transporter-linked polymorphic region (5-HTTLPR) of the serotonin transporter gene (5HTT) has been widely studied in the GxE literature. This gene is very important for researchers interested in genetic effects on behavior because of its responsibility for managing serotonin levels in the brain. The earliest research on the importance

of 5HTT-LPR linked the s-allele of this gene both directly and indirectly, in conjunction with stressful environments, to risks for depression and other psychological disorders. More recent research in the GxE literature has begun to link 5-HTTLPR with serious deviant and antisocial behaviors, and it is thus receiving more attention from sociologists and criminologists. Next, I will discuss a key gene in the dopaminergic system, the dopamine transporter gene.

The Dopamine Transporter Gene (DAT1)

The dopamine transporter gene (DAT1) has been mapped to chromosome 5 at location 5p15.3 and has a 40 base pair variable number of tandem repeats (VNTRs) (Vandenbergh et al. 1992). The DAT1 gene codes for the dopamine transporter protein, which helps remove dopamine from the synaptic cleft in a process called reuptake. The most common polymorphisms of this gene are the 9R (repeat) and 10R alleles, with the 10R allele being associated with a dopamine transporter that is excessively efficient in the reuptake process (Doucette-Stamm et al. 1995; Swanson et al. 2000). Manipulation of DAT1 activity can alter levels of dopamine found in the body, with dopamine levels being directly related to one's ability to stay focused and to feelings of happiness, so the DAT1 gene is potentially very important in the etiology of psychopathology (Niehoff 1999; Rowe 2002).

One line of research, that of the pharmacological methods to deal with ADHD, has pointed to the possibility that the DAT1 gene may play an important role in the etiology of deviant and antisocial behaviors. Many of the prescription drugs that have proven widely successful at suppressing the symptoms of ADHD and other psychopathological problems specifically target the DAT1 gene (Loo et al. 2002). These drugs work by depressing the dopamine transporter protein's ability to clear dopamine from the synapse, effectively interfering with the reuptake process (Seeman and Madras 1998). Another important fact about the

dopamine transporter that makes it an important gene to focus on in the literature concerning deviant and antisocial behaviors is that illegal drugs, such as cocaine and amphetamines, get their psychotropic effects by interfering with the reuptake process and effectively rendering the dopamine transporter ineffective (Kang, Palmatier, and Kidd 1999; Ritz et al. 1987).

While the empirical research directly tying DAT1 to phenotypes such as ADHD has been inconclusive at best (Maher et al. 2002), a large number of studies have now given evidence to the importance of DAT1 in moderating the environment's effects on behavior and psychopathology. Stevens et al. (2009) found that DAT1 moderated the effect of institutional deprivation on ADHD symptoms in a sample of children in Romanian orphanages, with those carrying 10R alleles showing more ADHD symptoms at 6, 11, and 15 years of age. Guo, Tong, and Cai (2008) found that the 10R allele of DAT1 had direct effects on the number of sexual partners that white males reported having in the Add Health sample. Guo et al. (2008) found in the same study that the proportion of students in one's school who were having sex by age 16 exacerbated this relationship. Numerous other studies have produced similar results concerning DAT1 and antisocial outcomes (Beaver, Wright, and Walsh 2008; Guo et al. 2007; Guo, Roettger, and Shih 2007).

Also among the findings in the GxE literature concerning DAT1 that should be of particular interest to sociologists concerned with crime and delinquency are the number of studies that have identified DAT1 and the combination of DAT1 and risky environments as risk factors for exposure to delinquent peers and the formation of delinquent peer groups. Using a direct measure of peer delinquency, Cleveland, Wiebe, and Rowe (2005) found that as much as 64% of the variance in delinquent peer affiliations was explained by genetic factors, including DAT1. In an important finding concerning GxE effects on delinquent peer group formation,

Beaver, Wright, and Delisi (2008) found that the 10R allele of DAT1 interacted with high-risk family environments to predict associating with substance-using peers. Expanding on this finding, Yun, Cheong, and Walsh (2011) found similar results looking at the 10R allele of DAT1, risky family environments, and delinquent peer associations while utilizing a direct measure of peer delinquency.

To conclude, the dopamine transporter gene (DAT1) has been prominent in the GxE literature. The DAT1 gene is an important candidate for study in the literature on criminal and deviant behaviors because of its importance in the maintenance of appropriate dopamine levels in the brain. Research has shown that the 10R allele of DAT1 is too efficient at clearing dopamine from the synaptic cleft, so this version of the gene, both directly and in interaction with the environment, is important for researchers to take into account who are interested in the etiology of low self-control, ADHD, delinquency, and substance abuse.

Now that I have reviewed some basic information on genes and how they affect behavior directly and in combination with the environment, as well as reviewed the three particular genes that will be the focus of the three analyses presented in this dissertation, I turn my focus to criminological theory. Specifically, I will give brief reviews of the three theories being tested in this dissertation (self-control, general strain, and social learning theories), and I will argue for why including them into a larger biosocial framework that introduces the gene-environment interaction literature is appropriate and logical given prior GxE research and theorizing.

CRIMINOLOGICAL THEORY AND BEHAVIORAL GENETICS: HOW THEY FIT

Theories of crime and delinquency have come a long way since the turn of the twentieth century. Sociologists of crime and criminologists have left behind theories of crime that focused solely on explaining how criminals were biologically inferior, less intelligent, insane, or even

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possessed to develop more nuanced theories that focus on the social environment and its effects

on individual criminal and deviant behavior. While the last hundred years or so have seen the

development of a large number of theories of crime that have since been supported through

empirical research and have added greatly to our knowledge and understanding of crime in

society, I would argue that we are reaching a point of saturation. Some of the more classic and

supported theories of crime have been getting tested and retested ad nauseum for some time. It's

time to freshen things up and bring some originality back into these theories of crime. This is

where the GxE literature comes in, by sociologists of crime and criminologists considering the

wealth of knowledge that has been generated in the last ten years concerning how our biological

makeup shapes our behavior. As I will argue shortly, some of our best theories of crime seem

particularly suited to this sort of combination, given their particular focus and the wealth of

findings concerning certain genes and how they interact with particular environmental stimuli to

effect behavior. In this section I will discuss three important theories of crime and how genetics

fit in to each one by making connections among seemingly disparate studies that suggest these

theories are appropriate and logical explanations of the genetics-environment-behavior

relationship. I will begin by discussing self-control theory.

Self-Control Theory

In a relatively short time since the publication of Gottfredson and Hirschi's *A General

Theory of Crime* (1990) their self-control theory has attracted much attention in the criminology

literature (Cohn and Farrington 1999; Pratt and Cullen 2000). This theory's popularity (or

infamy, perhaps) is due in no small part to its author's rather bold assertion that all other

criminological theories are wrong and that all individual variation in criminal activity can be

explained by one variable: self-control. While this assertion has not been supported and other

theories of crime continue to develop and flourish, the research on self-control theory has shown it to be an important theory of crime and delinquency (Pratt and Cullen 2000). I will briefly run through this theory's central premises and its support in the empirical literature before turning to a discussion of why self-control theory is a prime candidate for integration into a biosocial framework that seeks to account for crime and deviance.

A central assumption behind Gottfredson and Hirschi's (1990) self-control theory is that opportunities for crime are ubiquitous and that individuals engage in criminal activity because it is gratifying, and no other explanation is needed. Crime provides quick access to pleasure and things of value (Gottfredson and Hirschi 1990). Gottfredson and Hirschi (1990) also argue that engaging in crime is easy, requiring very little planning and no special training. Along with criminal activity, "analogous" behaviors (drinking heavily, having unprotected sex, etc.), which can be considered deviant when committed by certain people and under certain conditions, also provide quick and easy gratification. According to Gottfredson and Hirschi (1990), those who are likely to engage in crime are also likely to engage in these analogous behaviors. Inherent in this belief is an assumption of versatility by Gottfredson and Hirschi, they believe all offenders will be general offenders rather than specialists.

By ignoring the central nature of crime, that it's fun, Gottfredson and Hirschi argue that other theories of crime offer convoluted and unnecessary explanations for why individuals commit crime. Again, these authors argue very simply that people commit crime because it's gratifying. This marks a similarity between self-control theory and Hirschi's (1969) social bonding theory, in that both seek to explain why people choose not to engage in this very gratifying behavior. The answer, according to self-control theory, is that most people have high self-control. Those who have high self-control will have a strong tendency to not engage in

crime throughout their life course, even when presented with the opportunity. In contrast, those individuals who are low in self-control will, when the opportunity presents itself, engage in crime (Gottfredson and Hirschi 1990).

Gottfredson and Hirschi have provided a controversial definition of self-control. They define self-control as "the differential tendency of people to avoid criminal acts whatever the circumstances in which they find themselves" (Gottfredson and Hirschi 1990:87). Therefore, Gottfredson and Hirschi don't define self-control separately from the propensity to engage in crime. "High self-control" and "low self-control" simply act as labels that stand in for either low or high propensities toward criminality (Akers and Sellers 2009). Gottfredson and Hirschi do, however, note a number of traits of the person low in self-control. The individual low in self-control is impulsive, unable to defer gratification, lacking in diligence, thrill-seeking, physical (as opposed to mental), self-centered, and prone to accidents (Gottfredson and Hirschi 1990).

According to Gottfredson and Hirschi (1990), parenting is the source of self-control. Ineffective child-rearing results in children with low self-control, while effective child-rearing produces children with high self-control. According to Gottfredson and Hirschi (1990) parents who produce children with high self-control monitor their children's behavior, recognize deviant behavior when it occurs, and punish that behavior. As long as parents consistently monitor and discipline their children, Gottfredson and Hirschi say that they will produce offspring who are capable of delaying gratification, who are sensitive to the needs of others (empathetic), who are willing to accept restraint, and are unlikely to force to attain gratification from others. Some scholars have been critical of Gottfredson and Hirschi's assertions of what constitutes "effective" parenting, highlighting the need for nurturance, warmth, and love in the parent-child relationship

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to produce prosocial individuals (Burt, Simons, and Simons 2006). Now that I have described self-control theory, I will discuss how it fits with the existing GxE literature.

Self-Control Theory and Gene-Environment Interactions

Self-control theory fits well into a larger biosocial framework when one takes into account the at-first-glance disparate findings in the GxE literature concerning genes involved in processing dopamine in the brain, self-control and criminal and delinquent behavior. While self-control as defined and measured by self-control theory has not often been addressed in the GxE literature, this literature has focused much research on another important construct dealing with attentional deficits in individuals, that being attention deficit-hyperactivity disorder (ADHD).

As they are often measured, ADHD and Gottfredson and Hirschi's concept of low self-control are very similar constructs (see Unnever and Cornell 2003). Both ADHD and low self-control have as central issues the inability to focus and impulsivity. Much of the research concerning ADHD and genetics can therefore be considered as overlapping with the literature on self-control. To date, many studies, most focusing on genes involved in the handling of dopamine, have shown significant direct genetic effects on ADHD symptomatology. The onset of ADHD is thought to have a strong genetic component (Bobb et al. 2005), while environmental factors that are considered key to sociologists of crime, such as family, peer, and school contexts, have been shown to interact with genetic risks to increase the severity of ADHD symptoms (Coolidge, Thede, and Young 2000; Ficks and Waldman 2009).

In one study, Kim-Cohen et al. (2006) found that among young boys who had been the victim of abuse, individuals with the low-activity version of the MAOA gene were rated by teachers and mothers as having more symptoms of ADHD than similar boys with the high-activity version of the gene. Stevens et al. (2009) found that the DAT1 gene moderated the

effects of experiencing severe institutional deprivation in an orphanage setting on ADHD symptoms in early and middle adolescence. Similarly, Laucht et al. (2005) found that the 10R allele of the DAT1 gene moderated the effects of high "psychosocial adversity" on ADHD symptomatology assessed at age 15 years in a high-risk sample of both males and females. In a more recent study looking at a measure of "self-regulation" that strongly parallels self-control, Belsky and Beaver (2011) found that several genes modified the effect of ineffectual and detached parenting on levels of self-regulation in a national probability sample. Those who experienced the poorest parenting scored the lowest on a scale of self-regulation if they carried more "plasticity" alleles ("risk" alleles in diathesis-stress terminology), including the 2R/3R alleles of MAOA and the 10R allele of DAT1.

These findings in the GxE literature concerning genes involved in the handling of dopamine and self-regulating behaviors strongly parallel those looking in gene-environment interplay and criminal and antisocial behaviors. In a groundbreaking study that followed a New Zealand cohort from adolescence into young adulthood, Caspi et al. (2002) found that males with the low-activity version of the MAOA gene that had been subjected to childhood maltreatment were much more violence prone than a matched sample of males with the high-activity MAOA gene that had also been subject to childhood maltreatment. A number of studies that sought to replicate the findings of Caspi et al. (2002) found similar results tying the low-activity version of MAOA to aggression when paired with childhood maltreatment and adversity (Ducci et al. 2008; Foley et al. 2004; Kim-Cohen et al. 2006; Nilsson et al. 2006; Widom and Brzustowicz 2006). In a seminal study in the sociological literature on GxE and crime and delinquency, Guo et al. (2008) found that genes involved with the processing of dopamine moderated the effects of family, school, and friendship processes on both general and violent delinquency.

Taken together, the GxE literature on dopamine, "self-control", as measured in various ways, and criminal and antisocial behaviors provides a strong rationale for integrating the parenting-self-control-crime model of self-control theory into a biosocial framework that takes into account genetic risks for low self-control and crime and deviance. Having shown the basis for this rationale, I will now turn to a discussion of general strain theory (GST) and why the existing GxE literature suggests it is a good fit for a broader biosocial framework.

General Strain Theory

General strain theory (GST) was proposed by Robert Agnew (1992) as an individual level, social psychological explanation of crime and delinquency. The theory is an expansion of traditional strain theories, which primarily focus on an individual's inability to achieve economic and class-based status goals and the resulting stress this causes (see Cloward & Ohlin 1960; Cohen 1955; Merton 1938). Agnew added to the threatened or actual experience of not achieving highly valued goals two other types of strain likely to lead to deviant coping: the loss of valued persons or objects and the presentation of noxious stimuli (i.e., childhood abuse, ineffectual parenting, etc.). The focus on the loss of valued persons or property and the presentation of noxious stimulus as important strains comes from the literature on perceptions of justice and exposure to stress (Mirowsky & Ross 2003; Pearlin 1989). Research in the GST literature suggests that strains that are high in magnitude (severe), seen as unjust (undeserved, unfair), and linked with low social control may be particularly criminogenic (Agnew 2001).

According to Agnew, experiences of strain are important because they lead to the development of negative emotions, such as anger, depression, fear, and anxiety. Individuals have many different ways to cope with these negative emotions, and the likelihood that they cope through deviant methods is influenced by the kinds of coping mechanisms available to them and

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numerous variables drawn from other theories of crime (social control, social learning, and self-control theory). For example, the individual who associates with many delinquent peers may be more likely to engage in deviant coping, while an individual who is firmly attached to their family and school may be less likely to do so. Similarly, respondents with low self-control may be less able to effectively cope with significant strains and more inclined to respond in a delinquent manner.

In GST, individuals are essentially pressured into criminal and delinquent acts by the negative affective states, such as anger and depression, that result from negative relationships and experiences (Agnew 1992). The negative affect created by negative relationships and experiences requires corrective action. Without other avenues to vent or due to a person's level of self-control, etc. (see above), they may deal with strain and negative affect through the use of illegitimate channels to achieve goals, attack or escape from the source of their adversity, or manage their negative affect through other deviant means, such as the use of illicit drugs (Agnew 1992). Almost 20 years of research have produced large amounts of empirical support for GST and have shown it to be an important theory of crime and deviance (Agnew 2002; Kaufman 2009; Piquero and Sealock 2000). Now that I have described GST, I will discuss how it fits into the existing GxE literature, particularly the GxE literature on serotonin, depression, and criminal and deviant behaviors.

General Strain Theory and Gene-Environment Interactions

General strain theory is a good candidate for explaining a number of findings in the GxE literature. Specifically, there is a large literature on what the combination 5-HTTLPR and various "stressful life events" mean for levels of depression in individuals over the life-course. More recently, studies have also connected 5-HTTLPR and various environmental factors to

delinquent and aggressive behaviors. GST seems like a logical theoretical fit for explaining

these previous findings in the GxE literature concerning 5-HTTLPR, "strains", depression, and

delinquent and aggressive behaviors. I will begin by discussing the rather large literature on

serotonin, stress, and depression.

Again breaking new ground, Caspi et al. (2003) were the first to show empirically the

existence of a relationship between 5-HTTLP, stress, and depression. In a prospective,

longitudinal study of a representative birth cohort, Caspi et al. (2003) found that the number of

copies of the s-allele (0-2) of 5-HTTLPR that a person carried moderated the effect of stressful

life events on depressive symptoms, major depressive episodes, and suicidal ideation and

attempts at age 26 years. The more s-alleles a person carried, the more likely they were to

respond with psychological problems when confronted with stress. Importantly for GST and its

utility in explaining these findings, the kinds of life events counted as stressors in this study are

all events that would be counted as strains in the GST literature, including employment,

financial, housing, health, and relationship-related stressors (Caspi et al. 2003).

In attempting to replicated Caspi et al., numerous other studies have added evidence to

the utility of GST for explaining findings concerning 5-HTTLPR, strain, and depression utilizing

various variables that constitute strain in the GST literature. Eley et al. (2004) produced similar

findings to Caspi et al. while measuring stress as family social adversity, recent serious illness,

bereavement, relationship breakdowns, unemployment, and financial crisis. Taylor et al. (2006)

produced similar results concerning 5-HTTLPR and depressive symptomatology while looking

at strains that included early family adversity, such as feeling unloved and uncared for, being put

down, sworn at, and insulted by caregivers, witnessing or experiencing physical violence in the

home, and living with a substance user, and current stress. Similarly, Brummett et al. (2008)

made the 5-HTTLPR-depression connection where the strain in question was caring for a relative with Alzheimer's disease.

Taken together, these and other studies firmly establish the link between strain and depression as moderated by the serotonin-transporter-linked polymorphic region. This is, however, only one half of the GST equation. More recently, a few studies have also connected 5-HTTLPR to aggressive and delinquent behaviors through its moderation of the effects of stressful environments, though this thread of research is still fairly underdeveloped. Aslund et al. (2012) found in a sample of Swedish adolescents that 5-HTTLPR moderated the effect of family socioeconomic status on a scale of juvenile delinquency that included by property and violent offending items. Also in a sample of Swedish adolescents, Nilsson et al. (2005) found that poor family relations interacted with the s-allele of 5-HTTLPR to produce higher levels of alcohol consumption and more frequent intoxication. In the most promising finding of these recent studies looking at 5-HTTLPR genotype and aggressive and delinquent behaviors, Simons et al. (2011) showed in a sample of African Americans that the s-allele of 5-HTTLPR (along with DRD4 and MAOA genotype) moderated the effects of a composite measure of the hostility of one's environment on antisocial attitudes and aggression.

The past research in the GxE literature on 5-HTTLPR, what could be called "strains", depression, and aggression and delinquency point toward GST as a logical theoretical model to explain these findings. Integrating a biosocial framework into GST could make for a more thorough explanation of why strains matter for negative affect and deviant behaviors. Strains matter on their own, but also in combination with how we react in terms of neurological functioning to strain, which is where serotonin and 5-HTTLPR come in. Next, I will briefly discuss the last criminological theory that will be tested in this dissertation, social learning

theory, and provide a discussion for why it fits well into the existing GxE literature, particularly the literature on the dopamine transporter gene (DAT1) and affiliations with delinquent peers.

Social Learning Theory

Despite many revisions over the last fifty or more years, what remains at the heart of the broad category of theories known as learning theories is Sutherland's (1947) theory of differential association. Sutherland argued that criminal behavior is learned behavior, and this learning occurs through a process of symbolic interaction with others, most notably intimates or primary group members (Akers and Sellers 2009). Delinquency and crime are a result of attitude transference in social learning theory, whereby one takes on the deviant definitions to which others, most importantly intimates, expose them (Warr 2002). Not surprisingly then, a key criminogenic variable in social learning theory is affiliating with delinquent peers. Having delinquent friends matters because they expose an individual frequently, for long durations, and with great intensity (importance) to deviant definitions. When individuals take on these deviant definitions and attitudes as their own, they are more likely to break the law than someone who has been consistently exposed to definitions unfavorable to deviance.

Burgess and Akers (1966) extended this idea, proposing a "differential association-reinforcement" theory that combined the work of Sutherland with the principles of operant conditioning. Their theory argues that individual offending is not only influenced simply by affiliating with delinquent peers and the behavioral models and antisocial attitudes these relationships provide, but also by the reinforcing rewards as opposed to punishments that criminal offending provides within these kinds of peer groups. When someone affiliates frequently or almost exclusively with delinquent peers, their own delinquent behavior is likely to be frequently met with esteem and approval from their peers, thus resulting in a learning history

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(balance of rewards versus punishments) favorable to repeating delinquent and deviant behaviors in the future. In contrast, the person who does not affiliate with delinquent individuals is likely to have their own delinquent behavior met with disapproval and punishment at the hands of others, thus resulting in a learning history that is unfavorable to repeating delinquent behavior in the future.

This theory, later developed further by Akers and labeled a theory of "social structure and social learning" (SSSL) (1985, 1998; Akers and Sellers 2009), remains the foundation of social learning theory in criminology, and affiliations with delinquent peers remains a key variable in the social learning theory literature. Now that I have briefly described social learning theory and the importance of affiliations with delinquent peers in this literature, I will turn to a discussion of why this theory works well in combination with the genetic literature on the functioning of dopamine in the brain and the existing GxE literature on the dopamine transporter gene (DAT1) and affiliations with delinquent peers.

Delinquent Peers, Dopamine, and Gene-Environment Interactions

As with other genes involved in the dopaminergic system (MAOA, DRD2, DRD4), DAT1 has been consistently tied to sensation-seeking and antisocial behaviors (Guo et al. 2007; Guo et al. 2008; Stevens et al. 2009). Of interest for thinking about DAT1 as it relates to social learning theories of crime and delinquency are a few recent studies that have identified DAT1 and the combination of DAT1 with risky environments as risk factors for exposure to delinquent peers. These studies have revealed both direct and indirect effects of DAT1 on the likelihood that young people will engage with delinquent peers (Beaver et al. 2008; Cleveland et al. 2005; Yun et al. 2011). The next logical step would seem to be connecting prior research on DAT1, delinquent peers, and offending and test whether DAT1 moderates the effects of delinquent peer

affiliation on one's own offending. DAT1 could be related to selection processes that result in delinquent peer association while also simultaneously moderating the peers-offending relationship. Recent theorizing in the GxE literature suggests this could be the case.

As previously mentioned, Belsky and colleagues (Belsky 1997, 2005; Belsky and Pluess 2009; Belsky et al. 2009) have argued that certain forms of genes like DAT1 make individuals more susceptible to environmental influence because dopamine is directly related to both pleasure and reward sensitivity. Due to genetic makeup, some people may simply be more receptive to the kinds of reinforcement and reward they receive from delinquent peers, meaning they may offend more when they affiliate with delinquent peers than does someone without a genetic makeup that puts them at risk. In the terms of social learning theory, some individuals may have a genetic makeup that causes them to more readily accept the deviant attitudes and definitions presented to them by their delinquent peers, and will more readily seek out their approval and recognition by engaging in delinquent behavior. In other words, genetic risks may put some people in a better position to not only seek out and affiliate with delinquents, but to also more readily accord with their delinquent friends' views and attitudes relating to delinquent behavior, thus, they themselves offend more often. Having now discussed in detail genes, their interplay with the environment, and why several theories of criminology fit well as explanations of previous findings in the GxE literature looking at criminal and deviant behaviors, I will briefly conclude this dissertation introduction.

CONCLUSION

Past theoretical and empirical work that has sought to explain criminal and deviant behaviors has too often taken a fragmented approach, with scholars in criminology, sociology, and numerous biological fields not communicating effectively with one another. The social

sciences have stuck mostly to explanations of deviance in terms of social factors, while genetic explanations of deviance have consisted mainly of arguments concerning hereditary influences. When viewed separately, these perspectives give us an incomplete view of criminal and deviant behavior.

More recent work, cited in this introduction, has started to dissolve the boundary between environmental and genetic explanations of criminal and deviant behaviors. Indeed, some of the most promising approaches to explaining crime are coming from a blend of environmental and genetic theorizing (see Simons et al. 2011). This dissertation adds to this expanding biosocial literature by testing three classic and highly supported theories of crime and delinquency in a GxE framework wherein genetics effect deviant phenotypes by moderating known criminogenic environmental influences on behavior. Specifically, I seek to determine how two genes involved in the dopaminergic system (MAOA and DAT1) moderate the parenting-self-control-offending relationship, how 5-HTTLPR moderates the strain-depression-offending relationship, and how DAT1 moderates the delinquent peers-offending relationship.

CHAPTER 2

GENES, PARENTING, SELF-CONTROL, AND CRIMINAL BEHAVIOR

In the past 20 years self-control theory (or, the general theory of crime) has become one of the most debated and tested theories of crime and deviance. This work all sprang from the publication of Gottfredson and Hirschi's *A General Theory of Crime* (1990). Due in part to this book's success these two researchers are among two of the most cited criminologists, and *A General Theory of Crime* is one of the most cited books in criminology (Cohn and Farrington 1999). Their success is due in no small part to the large body of research that has accumulated supporting their central claim that low self-control is strongly related to criminal and deviant behavior (for example, Pratt and Cullen 2000).

Importantly, however, numerous studies have found that the central variable in self-control theory that Gottfredson and Hirschi argue is the source of levels of self-control, parenting, is not the only factor relating to the development of self-control (Burt, Simons, and Simons 2006; Teasdale and Silver 2009; Wright et al. 2008). This raises a question of what other factors may play a role in determining levels of self-control and through it effect levels of criminal and deviant behavior. The current study examines the extent to which genetic factors may moderate the effect parenting has on levels of self-control and offending. Specifically, this study explores the way in which certain variants (alleles) of the monoamine oxidase A gene (MAOA) and the dopamine transporter gene (DAT1) interact with parenting to decrease levels of self-control and increase offending in a sample of males. Recent studies and reviews have reported that both of these genes are related to neuropsychological deficits and criminal

offending among this population (Caspi et al. 2002; Guo, Roettger, and Shih 2007; Kim-Cohen et al. 2006; Schilling, Walsh, and Yun 2011).

The current study takes criminological theorizing in a new direction. First, I integrate a highly supported theory of crime and deviance into a biosocial framework whereby psychological and behavioral outcomes are the result of an interaction between the social environment and genetics. The combination of self-control theory and gene-environment interactions (GxE) is a natural extension of recent findings in a variety of fields that show that certain genes, in combination with certain environmental triggers, are related to neuropsychological deficits like attention-deficit hyperactivity disorder (Schilling et al. 2011) and low levels of self-control (Belsky and Beaver 2011) on the one hand and aggression and criminal offending (Caspi et al. 2002; Guo et al. 2007) on the other.

This study also examines whether these gene-environment interactions operate in a fashion proposed by the cumulative plasticity hypothesis, as proposed within the differential susceptibility perspective (Belsky and Pluess 2009; Belsky et al. 2009; Belsky, Bakermans-Kranenburg, and van Ijzendoorn 2007). Most of the past genetically informed studies of neuropsychological deficits and criminal behavior have assumed a diathesis-stress paradigm where certain genetic alleles increase one's vulnerability to environmental adversity. This perspective focuses on how particular genetic variants amplify the likelihood that exposure to some negative social environment or condition, such as poor parenting, will result in problematic behavior (e.g., Caspi et al. 2002). In contrast, the differential susceptibility perspective focuses on how certain individuals, due to particular genetic makeups, are simply more susceptible to environmental influence than others. Simply put, some people are programmed by their genes to be more sensitive to environmental context, both for better and for worse (Belsky et al. 2007).

As part of this radically different view of genetic effects, it is expected that carrying more genetic "risk" should mean more reactivity to the environment. This expectation of "cumulative plasticity" is absent in the diathesis-stress paradigm.

The current study tests several implications of this biosocial model that combines self-control theory with the differential susceptibility perspective. First, I test the extent to which individuals with certain variants of the genes MAOA and DAT1 show higher or lower ratings of self-control than the comparison group based on the parenting to which they are exposed. Second, I test whether this gene-environment interaction shapes levels of criminal offending. Finally, in line with predictions based on self-control theory, I test whether levels of self-control mediate the relationship between this gene-environment interaction and criminal offending. There is evidence based on recent research in the gene-environment interaction literature that the simultaneous focus on both psychological and behavioral outcomes is appropriate, as genes are related to behavioral outcomes such as criminal offending because they influence brain processes related to attention and learning (Schilling et al. 2011; Simons et al. 2011). I test my predictions utilizing data from the National Longitudinal Study of Adolescent Health (Add Health). In the following sections, I will explicate my theoretical model more fully and describe the data set and methods in more detail.

PARENTING, SELF-CONTROL, AND CRIMINAL OFFENDING

Self-control theory has a very simple explanation for why certain individuals commit crimes fairly frequently and others almost never commit crime, even throughout their entire life-course: Some individuals are low in self-control, and those low in self-control will be inclined to commit crimes when presented with the opportunity. The definition of self-control within Gottfredson and Hirschi's theory is controversial because they never define it separately from the

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propensity to commit crime, but they do lay out a number of traits of the person with low self-control. The person low in self-control is impulsive, unable to defer gratification, lacking in diligence, a thrill-seeker, physical (as opposed to mental), and self-centered (Gottfredson and Hirschi 1990).

Self-control theory posits that the source of self-control is parenting. Effective child-rearing results in children high in self-control, while ineffective child-rearing results in children with low self-control. In self-control theory effective and adequate parenting involves monitoring children's behavior, recognizing antisocial behavior, and correcting this behavior. Parents who engage in these parenting practices will produce children who are capable of delaying gratification, are sensitive to the needs of others, are willing to accept restraint, and are unlikely to use force or violence to attain gratification from others (Gottfredson and Hirschi 1990). Some scholars, however, have been critical of self-control theory's low bar for parenting, noting that monitoring and discipline are most effective when paired with warmth and nurturance (Burt et al. 2006).

The overall evidence supporting the importance of self-control in crime causation that has accrued in the criminological literature in the past 20 years has been overwhelming. The bulk of the empirical evidence supports the central concepts of the theory, namely that parenting is a key source of self-control and that self-control is a strong predictor of both crime and analogous behaviors (Akers and Sellers 2009; Perrone et al. 2004). In a widely cited article, Pratt & Cullen (2000) conducted a meta-analysis of 21 empirical studies of self-control theory and found that self-control had consistent and strong effects on crime and analogous behaviors. More recent empirical tests of self-control theory have also added to the theory's support base (Boutwell and Beaver 2010; Felson and Staff 2006; Hay 2001; Hay and Forrest 2006; Nofziger 2008).

Some tests of self-control theory, however, have found that Gottfredson and Hirschi's theorizing about the relationship between parenting and self-control falls short. Burt et al. (2006) found that low self-control only partially attenuated the negative effect that poor parenting has on delinquency in a sample of African Americans. Simons et al. (2007) found that parenting is not the only factor related to low self-control utilizing the same sample, as family SES was also significantly related to levels of self-control. In another study, Wright et al. (2008) found negligible effects of parenting on levels of self-control.

In the current study I argue that one missing piece of explaining the effect of parenting on both levels of self-control and offending could be genetics. Could it be that some individuals, because of their genetic makeup, are simply more sensitive to the effects that parenting has on both self-control and offending? I will now turn to a discussion of the issue of moderation of environmental effects on neuropsychological functioning and behavior by genes.

DIFFERENTIAL SUSCEPTIBILITY TO CONTEXT AND CUMULATIVE GENETIC PLASTICITY

Much of the research looking at the genetic basis of neuropsychological functioning and antisocial behavior has focused on variations in genes involved in the regulation of neurotransmitters, such as serotonin and dopamine. Two such genes that have received much attention in this literature are the monoamine oxidase A gene (MAOA) and the dopamine transporter gene (DAT1).

The MAOA gene is located on the X chromosome. It encodes the MAOA enzyme, which metabolizes neurotransmitters, including norepinephrine, serotonin, and dopamine and therefore plays a key role in regulating behavior (Belsky and Pluess 2009). The MAOA gene has been infamously referred to as the "Warrior gene" due to its relationship with aggressive and violent behavior (Beaver et al. 2010). This relationship between MAOA and antisocial behavior

has only held in research on males. Since the MAOA gene is found on the X chromosome, males only have a single copy. Females by contrast have two copies, so even if one copy of the gene is "defective", their other copy usually compensates (Beaver et al. 2010; Simons et al. 2011).

Importantly though, most studies looking at the link between MAOA and antisocial behavior have found that it is only when individuals carrying certain variations of MAOA meet with environmental stressors that its negative effects appear. In the most famous study looking specifically at gene-environment interactions and MAOA, Caspi et al. (2002) found that it was young males in a sample of New Zealanders with the low-activity version of MAOA (the 2R and 3R alleles) that were most effected by childhood maltreatment in regards to their later antisocial behavior and aggression. Males in the sample with the high-activity version of MAOA who had also been the victims of childhood maltreatment displayed substantially less antisocial behavior later in life. In another study looking specifically at childhood maltreatment and MAOA, Kim-Cohen et al. (2006) found that 7 years old boys with the low-activity variant of MAOA who had been abused were rated by their mothers and teachers as having more attentional deficits than their abused peers with the high-activity version of MAOA.

While these findings can and have been interpreted in terms of diathesis-stress, some scholars have recently noticed that in these two studies those who are most "vulnerable" to the adverse effects of maltreatment actually tend to do better when not exposed to maltreatment than their peers. This seems to suggest more susceptibility or "plasticity", rather than simply more vulnerability to adversity. A number of other studies have also shown this for-better-and-for-worse relationship between MAOA and environmental adversity. In a large longitudinal study of adolescent twin boys, Foley et al. (2004) found that boys with the low-activity variant of MAOA

were more likely than their high-activity carrying peers to be diagnosed with conduct disorder when exposed to high levels of childhood adversity but were also less likely to be diagnosed with conduct disorder when adversity was absent. Nilsson et al. (2006) produced similar results with MAOA in a cross-sectional study, finding that maltreatment and living arrangement experiences were related to criminal behavior in this for-better-and-worse fashion. Numerous other studies have produced similar findings (Ducci et al. 2008; Widom and Brzustowicz 2006).

A number of other studies in the gene-environment interaction literature have focused on the dopamine transporter gene (DAT1). DAT1 largely determines the magnitude and duration of synaptic dopamine signaling. It does this by transporting dopamine from the synaptic cleft back into the presynaptic knob for repackaging and reuse after it is finished exciting downstream neurons (Schilling et al. 2011). A particular variation of DAT1 (the 10R allele) is more efficient in this reuptake process than other variations, which means there is less dopamine in the synaptic cleft available for activation (Miller-Butterworth 2008). This is problematic because dopamine activates pleasure centers in the brain, so it being too rapidly cleared from the synaptic cleft leads the individual to seek out pleasures that raise dopamine levels, whether legal or illegal.

Numerous studies have looked at the relationship between DAT1, the social environment, and sensation-seeking and antisocial behaviors. Guo, Tong, and Cai (2008) found that the 10R allele of DAT1 had direct effects on the number of sexual partners that white males reported having in the Add Health sample. Guo et al. (2008) found in the same study that the proportion of students in one's school who were having sex by age 16 exacerbated this relationship. Importantly though for arguments based on differential susceptibility, they found that marriage and frequent church attendance dramatically reduced the effect of the 10R allele on number of sex partners in this sample. Stevens et al. (2009) found that DAT1 moderated the effect of

institutional deprivation on ADHD symptoms in a sample of children in Romanian orphanages, with those carrying 10R alleles showing more ADHD symptoms at 6, 11, and 15 years of age. Beaver and Belsky (2012) found that among other genes, DAT1 was associated with the intergenerational transmission of parenting practices. Specifically, carriers of the 10R allele and other plasticity alleles experienced the highest levels of parenting stress when they were themselves exposed to negative maternal parenting, but also the lowest levels of parental stress when exposed to positive maternal parenting in adolescence (Beaver and Belsky 2012). Numerous other studies have produced similar results concerning DAT1 and antisocial outcomes (Beaver, Wright, and Walsh 2008; Guo et al. 2007; Guo, Roettger, and Shih 2007).

In summation, past studies have shown that particular variations of MAOA and DAT1 moderate the effects of the social environment on various aggressive, sensation-seeking, and generally antisocial behaviors, as well as self-control. While these findings are usually interpreted in a diathesis-stress fashion, where certain forms of a genes are defined as "risk" alleles that increase the probability that adverse circumstances will lead to problematic behavior, Belsky and colleagues (Belsky et al. 2007; Belsky and Pluess 2009) have opted for a different interpretation, that of differential susceptibility. They suggest that these so-called "risk" alleles influence the extent to which individuals are responsive to their social environment, for both good and bad.

How do genes cause some individuals to be more sensitive to their environment? Belsky and Pluess (2009) observed that the genes in question, such as MAOA and DAT1, are related to the dopaminergic and serotonergic systems, with the dopaminergic system implicated in reward sensitivity and the serotonergic system implicated in sensitivity to punishment and displeasure (Simons et al. 2012). This leads them to posit that some individuals are more responsive to their

environment because of their different thresholds for experiencing pleasure or displeasure, meaning that because of their genetic makeup these individuals are more readily shaped by environmental rewards and punishments than are others.

Belsky and Pluess (2009) have further speculated that the more of these plasticity alleles an individual carries, the more susceptibility to their environment they will evince. This is known as the cumulative plasticity hypothesis. So, for example, if someone carries plasticity alleles for both MAOA and DAT1, they should be more readily shaped by their environment than someone who carries a plasticity allele for either MAOA or DAT1 alone. This is in addition to being much more readily shaped by their environment than someone who doesn't carry a plasticity allele for either MAOA or DAT1, holding the presence of other plasticity alleles constant. Several recent studies have provided support for this hypothesis (Beaver and Belsky 2012; Belsky and Beaver 2011; Simons et al. 2011; Simons et al. 2012), and this hypothesis will be a central focus of this study.

THE CURRENT STUDY

The current study expands on past gene-environment interaction studies of criminal behavior in two important ways. First, I integrate a gene-environment interaction approach into a highly supported and time-tested criminological theory, self-control theory. All three of the key elements present in self-control theory, parenting, self-regulation, and criminal offending have shown up in research in the gene-environment interaction literature and have been tied specifically to MAOA and DAT1. Therefore, the integration of these two literatures and the testing of a combined model is a natural extension of past research and theorizing.

My focus on self-control theory is important for a couple of reasons. First, I go beyond most prior gene-environment interaction models by testing whether these interactions are linked

to a key neuropsychological deficit in the criminological literature, low self-control, which is linked with criminal offending. I then examine the extent to which the effect of the interaction of parenting and genetics on criminal offending is mediated by low self-control. Past research has established that low self-control mediates much of the effect of parenting on criminal offending. I expect that in my elaborated model that includes genetic effects; low self-control will mediate a significant portion of the interaction of parenting and genotype on criminal offending. Thus, low self-control should act as a mediated moderator.

The second significant expansion on past gene-environment interaction studies in the current study is in testing Belsky and Pluess' (2009) propositions regarding cumulative genetic plasticity. With some recent exceptions (see Beaver and Belsky 2012; Belsky and Beaver 2011; Simons et al. 2011; Simons et al. 2012), few studies in the gene-environment interaction literature have focused on more than one gene at a time in a given analysis. In the current study, I focus on the combined effects of MAOA and DAT1. Coming from the differential susceptibility perspective, the cumulative plasticity hypothesis would predict that carriers of plasticity alleles for both these genes would be expected to show greater susceptibility to their environment than carriers of one or no plasticity alleles. The current study will test this idea.

Prior research in the GxE literature leads me to expect that MAOA and DAT1 genotype will condition the theoretical model of parenting-self-control-crime identified by self-control theory. Specifically, I expect that those individuals who carry the 2R or 3R allele of MAOA, are homozygous for the 10R allele of DAT1, or both will have both lower levels of self-control and higher levels of criminal offending than individuals who carry neither of these plasticity alleles when they experience a poor relationship with their primary caregiver. Furthermore, I expect that the greatest contrast in the parenting-self-control and parenting-crime relationships will be

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between those who carry plasticity alleles for neither MAOA nor DAT1 and those who carry plasticity alleles for both. In addition, I expect that the parenting by MAOA/DAT1 genotype effect on offending behavior will be mediated by low self-control, per the expectations of self-control theory. The parenting by MAOA/DAT1 genotype effect on offending will thus operate as a mediated moderator model that combines self-control theory with previous findings in the GxE literature, with self-control acting as the mediating variable.

I draw on these expectations as well as self-control theory and the cumulative plasticity hypothesis within the differential susceptibility perspective to derive specific hypotheses about the relationship between parenting, genetics, self-control, and criminal offending. Drawing on self-control theory, *hypothesis 1* predicts that parenting will significantly shape levels of self-control. Drawing on the differential susceptibility perspective's expectations of cumulative plasticity, *hypothesis 2* predicts that genotype will moderate the effect of parenting on self-control in such a way that those with 2 plasticity alleles will be the most susceptible to the effects of parenting in regards to their levels of self-control. *Hypotheses 3-4* make identical predictions to *hypotheses 1-2*, but criminal offending is substituted as the dependent variable of interest. Lastly, drawing on self-control theory, *hypothesis 5* predicts that low self-control will significantly mediate the effect that the interaction between parenting and genotype has on criminal offending. Next, I will describe the data set, measures, and methods used to test these hypotheses.

DATA AND METHODS

Data

For the current study I draw on data from the National Longitudinal Study of Adolescent Health (Add Health). Add Health is a nationally representative sample of American adolescents

who were first recruited during the 1994-1995 school year while they were in grades 7-12 (Harris et al. 2003; Udry 1998). Add Health obtained a nationally representative sample of adolescents by utilizing a multistage stratified sampling process to select 80 high schools and 52 middle and junior high schools for inclusion in the study. More than 90,000 students completed in-school self-report surveys, and of this group a subsample was randomly chosen for the Wave I in-home component of Add Health. In total, 20,745 adolescents and 17,700 of their primary caregivers participated in the Wave I in-home component (Harris et al. 2003). Wave II data collection occurred approximately 1 to 2 years after Wave I data collection, Wave III data was collected during 2001-2002 when respondents were between 18-26 years old, and Wave IV data was collected during 2007-2008 when respondents were between 24-32 years old.

During Wave IV in-home interviews Add Health collected a number of types of biological data. Among the data collected, Add Health took saliva swabs from all Wave IV interviewees for DNA analysis. In conjunction with the Institute for Behavioral Genetics (IBG) in Boulder, CO, Add Health genotyped Wave IV interviewees for a set of genetic markers of interest to biosocial researchers. That Add Health is a large and nationally representative data set that contains variables measuring both the social environment and genetics makes it highly desirable for the current study. The current study includes male Add Health respondents who were interviewed at Waves I, II, and IV who did not have any missing genetic data. After dealing with missing data, the current study includes the analysis of information gathered from 3,610 male respondents in the Add Health data set.

Measures

Criminal Behavior

The dependent variable consists of 9 items drawn from Wave II that asked respondents about various criminal activities they engaged in during the prior year. These items are a mixture of violent and property offending. Measures of violence included questions asking how often respondents used or threatened to use a weapon on someone to get something from them, pulled or actually used a knife or gun on someone, used a weapon during a fight, or hurt someone so badly in a fight that they needed medical attention. Property offending measures included asking respondents how often they painted graffiti, stole cars, burglarized buildings, and stole items worth more than $50. This scale is closely related to scales that other researchers have developed for use in the Add Health data set in past studies (Guo et al. 2008; Hagan and Foster 2003; Haynie 2001, 2003). I summed these 9 items into a global measure of involvement in criminal activity in the past year (alpha = .74). Due to extensive skew, I logged this measure. Logging the measure resulted in a scale with greatly reduced skew and a relatively normal distribution. Higher scores on this scale of criminal behavior indicate more offending.

Poor Parent-Child Relationship

The measure of parenting in the current study consists of 5 items drawn from Wave I. These items focus on the respondent's relationship with their mother, and include asking how close they feel to their mother, how much they think she cares about them, whether she is warm and loving most of the time, whether they are satisfied with how they and their mother communicate, and their overall satisfaction with their relationship with their mother. I summed these five items to create a measure of poor parent-child relationship (alpha = .81). I transformed

this scale by standardizing it to facilitate easy interpretation of the GxE interaction terms. Higher scores on this scale indicate less maternal warmth and more maternal disengagement.

This scale of parenting is limited because it does not directly measure parental involvement, monitoring or disciplinary preferences, all of which are key concepts in self-control theory that relate to an adolescent's level of self-control (Gottfredson and Hirschi 1990). These types of items are unfortunately fairly limited in the Add Health data set, often have very few response categories, and do not correlate very highly with each other or other parenting measures in the Add Health study.[1] There is reason to believe, however, that the absence of these items will not unnecessarily bias this measure of parenting. Parental warmth, attachment, monitoring, and disciplinary style are all highly correlated (Simons and Burt 2011; Simons et al. 2007), and the parents of low self-control individuals usually evince a combination of a lack of warmth, monitoring, and consistent discipline (Burt et al. 2006). Prior research has shown that this scale of poor parent-child relationship has predictive validity in regards to levels of self-control (Belsky and Beaver 2011).

Low Self-Control

The appropriate method for measuring self-control has been the source of much debate in the criminological literature (Beaver et al. 2009; Longshore 1998; Longshore and Turned 1998). Grasmick et al. (1993) developed the most commonly used scale, but this scale or measures very similar to it are not available in Add Health, thus a different scale must be used to measure self-control. In the current study I use a slightly altered version of a scale of low self-control developed by Beaver et al. (2009) for use in the Add Health survey. This low self-control scale contains 21 items from both parent and self-report responses in Wave I interviews. The items in

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[1] As an example, the dichotomous nature of measures making up a "maternal involvement" scale, as well as this scale's suspect alpha, can be noted in two recent studies (Beaver and Belsky 2012; Belsky and Beaver 2011).

this scale measure a respondent's temper, their self-centeredness, their attention span, and their use of rational decision making, among others. A composite measure of self-control was created by summing all items (alpha = .71). Higher scores on this measure indicate lower self-control. Prior research has shown that this scale has predictive validity (Belsky and Beaver 2011; Beaver et al. 2009). All the items included in this scale are presented in Appendix A.

Genes

Four genetic polymorphisms were genotyped and included in the Wave IV Add Health genetic sample. In the current study I focus on two of these: MAOA and DAT1. The literature on MAOA has revealed that two low-activity versions of this gene (2R and 3R) are associated with negative behavioral and mental health outcomes among males (Belsky and Pluess 2009; Caspi et al. 2002; Kim-Cohen et al. 2006). Following past research, I code MAOA to reflect the non-presence (0) or presence (1) of either the 2R or 3R allele. Based on this coding, 59.2% of respondents in this sample were not carriers of either the 2R or 3R allele, while 40.2% of respondents were carriers of either the 2R or 3R allele.

DAT1 has a 40-base pair (bp) variable number of tandem repeats that can be repeated 3-11 times (Beaver et al. 2008). Past research has shown that males who are homozygous for the 10R allele of DAT1 (10R/10R) are significantly more susceptible to a number of behavioral and psychological problems (Beaver et al. 2008; Guo et al. 2007; Schilling et al. 2011). Accordingly, I coded DAT1 to reflect the non-presence (0) or presence (1) of two 10R alleles. Based on this coding, 40.9% of respondents in this sample had an allelic combination other than 10R/10R, and 59.1% of respondents were 10R homozygotes.

Finally, I created dummy variables for the number and type of plasticity alleles individuals carry for use in all of the analyses. I defined these dummy variables as follows: two

plasticity alleles = 2R or 3R-allele MAOA and 10R-allele DAT1 homozygotes (25% of the

sample); MAOA only = 2R or 3R-allele MAOA and not 10R-allele DAT1 homozygotes (16%);

DAT1 only = 10R-allele DAT1 homozygotes and no 2R or 3R MAOA allele (34%). The

reference category in all of the analyses includes those who do not carry a 2R or 3R MAOA

allele who are also not homozygous for the 10R allele of DAT1 (25%). The Hardy-Weinberg

equilibrium test showed that this distribution of MAOA and DAT1 did not differ significantly

from that predicted on the basis of simple Mendelian inheritance.

Controls

I also include several general controls in all analyses that have been shown to be

correlated with involvement in crime: Age, dummy variables for Hispanic, non-Hispanic Black,

Native American, Asian, and Other (with non-Hispanic White as the reference category),

parent's education (1=4 year degree or more), and parent receiving public assistance (1=yes).

About 55.8% of the sample is non-Hispanic white, 14.9% Hispanic, 17.8% non-Hispanic black,

with the remainder comprising Native American (2.6%), Asian (7.7%), and members of other

racial/ethnic groups (1.1%). About 27.4% of parents are college graduates, while 7% report

receiving public assistance.

Analytic Strategy

I test hypotheses derived from self-control theory and the differential susceptibility

perspective using Ordinary Least Squares (OLS) regression techniques. The models test whether

parenting significantly shapes levels of self-control, per self-control theory (Hypothesis 1). The

models additionally test whether the interaction of a poor parent-child relationship and a

combination of MAOA and DAT1 significantly affect levels of self-control, and whether this

effect corresponds to predictions made by the differential susceptibility perspective (Hypothesis

2). I further test these predictions with criminal offending substituted as the outcome of interest (Hypotheses 3-4). Lastly, I test whether low self-control significantly mediates the effect that the interaction of a poor parent-child relationship and a combination of MAOA and DAT1 has on levels of criminal offending (Hypothesis 5). I utilize the appropriate weight, cluster, and strata variables in all analyses to account for the complex Add Health survey design. Tests using Variance Inflation Factors (VIFs) showed that multicollinearity was not a problem in any of the equations.

RESULTS

Table 2.1 presents the descriptive statistics for the study variables. As expected, criminal offending levels are fairly low. The average respondent in the sample was about sixteen years old at the time of Wave II data collection. Most respondents scored reasonably well on the low self-control scale. The majority of respondents in this dataset carry at least one plasticity allele between MAOA and DAT1. Table 2.2 further describes the data by presenting means and mean comparisons for the four genetic subgroups (2 plasticity alleles, 1 MAOA plasticity allele, 1 DAT1 plasticity alleles, and no plasticity alleles). Importantly, there are no mean differences between these groups in the key dependent variable criminal behavior, the key independent variable poor parent-child relationship, or the mediating variable low self-control. However, there are several racial/ethnic differences of note. Whites are underrepresented in the 2 plasticity allele group while blacks and Asians are overrepresented in the 2 plasticity allele group. There is clear empirical evidence that DAT1 genotype varies by ancestry and race/ethnicity (Kang, Palmatier, and Kidd 1999), while there is circumstantial evidence that MAOA genotype may vary slightly by race/ethnicity (Balciuniene et al. 2001; Gilad et al. 2002; Sarich and Miele 2004).

Table 2.3 presents the correlation matrix for the study variables. The correlation matrix serves to check for gene-environment correlations (rGE). Gene-environment correlation refers to a nonrandom distribution of environments among different genotypes (Simons et al. 2011). These gene-environment correlations potentially confound GxE effects (Guo, Roettger, and Cai 2008; Guo, Tong, and Cai 2008). Table 2.3 shows that there is no significant correlation between poor parent-child relationship and any of the genetic subtypes in the sample (2 plasticity alleles, an MAOA plasticity allele, a DAT1 plasticity allele, 0 plasticity alleles). This suggests an absence of rGE effects in these data.[2] Furthermore, genotype does not significantly correlate with criminal behavior. As expected, poor parent-child relationship is significantly related to levels of self-control at Wave I and criminal behavior at Wave II. In addition and in line with expectations, self-control is significantly correlated with criminal behavior. Of note, the presence of 0 plasticity alleles significantly and positively correlates with low self-control. This means that individuals with 0 plasticity alleles report slightly lower levels of self-control compared to the other genetic subtypes.

Table 2.4 presents OLS regression models using low self-control as the outcome of interest. Model 1 examines the effects of poor parent-child relationship and genetics on levels of self-control, while controlling for various factors. This model shows that as predicted by self-control theory and in support of hypothesis 1, parenting significantly affects levels of self-control, such that more detached, less warm parenting results in lower levels of self-control, and vice versa. Interestingly, plasticity alleles for both MAOA and DAT1 or just DAT1 alone evince a direct, negative genetic effect on low self-control compared to carriers of 0 plasticity alleles. Among the controls, blacks report slightly higher levels of self-control than do non-Hispanic

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[2] In addition, an independent samples t-test confirmed no mean-difference in poor parent-child relationship between the most and least plastic individuals in the sample.

whites, and individuals whose primary caregiver reported receiving public assistance at W1 report slightly lower levels of self-control.

Model 2 in table 2.4 provides a test of the cumulative plasticity perspective. The differential susceptibility perspective suggests that those who carry plasticity alleles for both MAOA and DAT1 should evince a stronger response to their social environment than those who carry plasticity alleles for neither or only one of the two. To test this possibility, I created interactions between the genetic dummy variables and poor parent-child relationship. The reference category in all analyses is the interaction between poor parent-child relationship and 0 plasticity alleles. As can be seen in model 2, DAT1 has a main, negative effect on low self-control compared to the reference group of those who carry 0 plasticity alleles. This effect is not shared by MAOA or, in this model, the presence of two plasticity alleles. Also, the direct, main effect of poor parent-child relationship remains positive and highly significant in this model. This means that even with the GxE terms in the model, there is a still significant effect of poor parent-child relationship on low self-control for individuals who carry 0 plasticity alleles. Turning to the interaction terms, and in line with hypothesis 2, having a plasticity allele for both MAOA and DAT1 interacts with poor parent-child relationship to significantly predict low self-control in the expected direction, meaning the effect of poor parent-child relationship on low self-control is significantly greater among this group than among the 0 plasticity alleles group. Furthermore, plasticity alleles for only MAOA or DAT1 separately do not have a significant interaction with poor parent-child relationship on levels of self-control. This means that among these genetic subgroups the effect of poor parent-child relationship on low self-control is essentially identical as it is for individuals with 0 plasticity alleles.

With table 2.5 I probe the effect of the interaction of poor parent-child relationship and genotype on low self-control found in model 2 of table 2.4. In this table, the effect of poor parent-child relationship on low self-control is broken down by the number and type of plasticity alleles an individual carries. The first finding to note is that there is a highly significant effect of poor parent-child relationship on low self-control regardless of the number and type of plasticity alleles one carries. This is highly supportive of the parenting-self-control link hypothesized by self-control theory. The effect of poor parent-child relationship, however, does differ some by genotype. Those who carry a plasticity allele for either MAOA or DAT1 alone do demonstrate a larger effect of parenting on low self-control than those without any plasticity alleles, but this difference is not statistically significant (see table 2.4, model 2). The main contrast is between those who carry both of these plasticity alleles and those who carry none. The difference between the two coefficients is statistically significant (see table 2.4, model 2), and the coefficient for those with two plasticity alleles is approximately 58.6% larger than for those who carry no plasticity alleles. Falling in line with predictions in the differential susceptibility perspective about cumulative plasticity, these findings suggests that individuals with both of these plasticity alleles respond more strongly to their environment, specifically parenting, than individuals who carry none or only one of these plasticity alleles.

Table 2.6 presents a similar modeling strategy with criminal behavior substituted as the outcome of interest. The baseline model in model 1 shows that the genes of interest in this study have no main effects on criminal behavior. As expected and in support of hypothesis 3, poor parent-child relationship is significantly and positively predictive of criminal behavior, with less warmth and more disengagement resulting in more offending. Among the controls, Native Americans report significantly more offending than do non-Hispanic whites.

Model 2 in table 2.6 is similar to model 2 in table 2.4, providing a test of cumulative genetic plasticity. The genetic dummy variables and interaction terms in this model are identical to those in table 4. As model 2 shows, the genetic dummy variables again have no main effects on criminal behavior. Similar to the results when looking at low self-control, and as predicted by hypothesis 4, having a plasticity allele for both MAOA and DAT1 interacts with poor parent-child relationship to significantly predict criminal behavior. The effect of poor parent-child relationship on criminal behavior is thus significantly stronger among individuals with 2 plasticity alleles than among those with 0 plasticity alleles. Much like with low self-control, plasticity alleles for only MAOA or DAT1 separately do not have a significant interactive effect with poor parent-child relationship on criminal behavior. Again, these results support the contention in the differential susceptibility perspective that individuals with more plasticity alleles are more reactive to their environment. A final finding to note in this model is that the direct effect of poor parent-child relationship on criminal behavior is no longer statistically significant. This means that the poor-parent child relationship effect is statistically insignificant among the omitted reference category, individuals with 0 plasticity alleles.

As a final test of expectations coming from self-control theory, low self-control is introduced in model 3 as a potential mediator of the interaction between poor parent-child relationship and genotype. As can be seen in model 3, the effect of the interaction between 2 plasticity alleles and poor parent-child relationship on criminal behavior is fully mediated by this construct, with the coefficient reduced by approximately 17.5% and to statistical insignificance. Low self-control has thus fully mediated the poor parent-child relationship-by-gene effect on criminal behavior, supporting hypothesis 5 and thus supporting the theoretical model identified by self-control theory.

Like with table 2.5 and low self-control, in table 2.7 I probe the effect of the interaction of poor parent-child relationship and genotype on criminal behavior that is found in model 2 of table 2.6. These results contrast greatly to the earlier results for low self-control. As demonstrated in table 2.7, the only group that displays a statistically significant effect of poor parent-child relationship on criminal behavior is those who carry plasticity alleles for both MAOA and DAT1. So, at least in the current sample, the only group among whom the effect of poor parent-child relationship on criminal behavior is statistically significant is those who carry both of these plasticity alleles. This again provides evidence as to the importance of cumulative plasticity when looking at environmental effects on behavior and neuropsychological functioning.

DISCUSSION

With this paper I sought to test whether genetic factors, specifically MAOA and DAT1 genotype, moderate the relationship between parenting, self-control and criminal behavior, as specified by Gottfredson and Hirschi's (1990) self-control theory. Additionally, I tested predictions concerning cumulative plasticity coming from the differential susceptibility perspective (Belsky and Pluess 2009) that focus on the importance of the number of so-called "plasticity" alleles a person carries and what this means for GxE effects. To explore this possibility I utilized data from the Add Health study (Udry 2003) that measured how adolescents are being parented (represented here by the relationship between the respondent and their mother), self-control, criminal offending, MAOA and DAT1 genotype, and numerous control variables. These data serve to examine whether MAOA and DAT1 genotype moderate the effect of parenting on levels of both self-control and criminal offending, whether the latter relationship

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is mediated by self-control, and whether contrasting individuals by the number and type of so-called plasticity alleles they carry effects these relationships.

The results of OLS regression models reveal significant GxE effects. Males who carry the 2R or 3R alleles of MAOA and are homozygous for the 10R allele of DAT1 are more likely to have lower levels of self-control and engage in more criminal behaviors when they experience a poor parent-child relationship than are individuals who do not carry either of these plasticity alleles. Another important finding is that in this study the effect of a poor parent-child relationship on criminal behavior is only statistically significant among the genetic subgroup of individuals who carry both of the plasticity alleles identified in this study. In addition, and in support of self-control theory, the GxE effect produced by the combination of poor parent-child relationship and MAOA and DAT1 genotype on criminal behavior is fully mediated by the scale of low self-control. Importantly for establishing a GxE rather than an rGE, the correlation matrix shows that the number and type of plasticity alleles one carries does not significantly correlate with poor parent-child relationship or criminal behavior. Before turning to the implications of this study, the limitations within should be noted.

While the current study makes important contributions to the GxE and criminological literatures, some limitations should be noted. First, the measure of parenting utilized in this study is not an optimal measure for testing propositions based on self-control theory. Self-control theory focuses on monitoring and discipline with respect to how self-control is shaped, while the measure of parenting in the current study focuses on the quality of the parent-child relationship. While the family literature has shown that loving parenting and watchful parenting tend to go hand in hand (Simons and Burt 2011; Simons et al. 2007), a fuller measure of parenting that includes items tapping parental involvement and monitoring would be best. A

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second measurement related issue is that the measures tapped to represent self-control in the Add Health data are not commonly used measures, and may miss some important elements of Gottfredson and Hirschi's (1990) concept of self-control, such as preferences for risk-taking. The above two issues would be best solved by testing the model put forth in this paper while utilizing a different data set that contains more validated measures of parenting and self-control while also including genetic information. The final key limitation is that while this study is based on a nationally representative data set, only males are included in these models, meaning that these results are not generalizable to all adolescents. This study is notable, however, because the sample size (N = 3,610) is much larger than has typically been available in GxE research.

These results make sense of and bring together previous findings that separately considered the relationship between genotype and attentional deficits and antisocial behaviors. These results point to the possibility that much of the reason that MAOA and DAT1 matter for criminal offending is because of their relationship with attentional deficits like low self-control. Past research might have found MAOA and DAT1 are so consequential for deviant and criminal behaviors precisely because they are so important, in combination with environment, for shaping neuropsychological attributes like levels of self-control.

Given the important place accorded self-control within the criminological literature (Akers and Sellers 2009; Pratt and Cullen 2000), and the results of both this study and others in the GxE literature (Belsky and Beaver 2011), future research would do well to further probe the genetic basis of neuropsychological deficits that approximate low self-control. In addition, this research should continue to identify the environmental factors, like parenting, that shape levels of self-control and test whether these effects are moderated by genotype. One possible candidate

that has emerged in recent research is the community context in which individuals live. The community context can play an important role in determining individual levels of self-control because neighborhood disadvantage usually correlates with parents and their neighbors having smaller amounts of social capital to pull from to properly socialize youth (Anderson 1999; Teasdale and Silver 2009). A neighborhood's relative affluence or disadvantage creates an environment in which the socialization efforts of parents will be more or less successful. It's possible that levels of concentrated disadvantage and collective efficacy in neighborhoods may affect individual levels of self-control, and much like the effect of parenting on self-control this neighborhood effect may be moderated by genotype. These potential neighborhood effects, as well as other environmental effects, are worth probing in the future to measure how they shape levels of self-control and offending, and whether genotype plays a role in this relationship.

In conclusion, this study provides evidence that a poor parent-child relationship interacts with MAOA and DAT1 genotype to increase risks for both developing low self-control and engaging in criminal behavior. These findings support the utility of self-control theory as a theoretical model to explain previous findings concerning genetics, neuropsychological deficits approximating low self-control, and antisocial behaviors. This study is additionally important theoretically in showing the utility in combining traditional criminological theories with a biosocial modeling approach. This kind of theory building and modeling strategy is important because it helps further develop ideas centered on the concept that the environment and biology are always interacting to shape how we experience and react to our world. Moving forward, criminology as a discipline should focus on and consider central this kind of theory building and modeling.

CHAPTER 3

5-HTTLPR, STRAIN, DEPRESSION, AND CRIMINAL BEHAVIOR

Robert Agnew's (1992; 2006) general strain theory (GST) has become one of the more

popular theories of crime in both the sociological and criminological literature over the past two

decades. The popularity of the theory is due in no small part to the large amount of research that

has accumulated establishing support for the theory as an explanation for criminal and delinquent

behavior (Agnew 2006; Botchkovar, Tittle, and Antonaccio 2009; Carson et al. 2009; Hay and

Evans 2006; Jang 2007; Kaufman 2009). Agnew's (1992) theory argues that negative stimuli or

"strains" affect individuals by producing negative emotions, most notably anger and depression,

which can then in turn result in criminal or delinquent behavior.

An important idea proposed by Agnew that has been somewhat less explored, however, is

the proposition that the likelihood of strain resulting in negative emotions and delinquent

behavior is conditioned by other important variables in the criminological literature.

Considering the strong emphasis on the social environment in this literature, it is not surprising

that the candidates that potentially condition the strain-negative emotions-crime relationship that

have thus far been explored tend to be sociological variables, such as relationships with parents,

associations with delinquent peers, and religiosity (Agnew 1999; Broidy and Agnew 1997; Moon

et al. 2009; Thaxton and Agnew 2004). This approach is limited, however, in that it ignores

important advancements in genetics research in the last decade that have opened our eyes as to

the interplay between biology and the social environment in effecting behavior. The current

study examines the extent to which genetic factors may moderate the effect that strain has on

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levels of negative emotions, specifically depressive symptoms, and criminal offending. Specifically, this study explores the way in which a particular variant (allele) of the serotonin-transporter-linked polymorphic region (5-HTTLPR) interacts with strain to increase levels of depressive symptoms and criminal behavior in a sample of males. Recent studies and reviews have reported that 5-HTTLPR is related to neuropsychological issues, most especially depression, and offending when paired with stressful life events (Belsky and Beaver 2011; Belsky and Pluess 2009; Caspi et al. 2003; Vaughn et al. 2009).

The current study takes criminological theorizing in a new direction. With this study I integrate an important theory of crime and deviance into a biosocial framework whereby psychological and behavioral outcomes are the result of an interaction between the social environment and genetics. The combination of GST and a gene-environment interaction (GxE) approach focusing on 5-HTTLPR is a natural extension of recent findings in a variety of fields that show that 5-HTTLPR is related to neuropsychological deficits like depression (Caspi et al. 2003) and attention-deficit hyperactivity disorder (Schilling et al. 2011) on the one hand and aggression and criminal offending (Simons et al. 2011; Simons et al. 2012; Vaughn et al. 2009) on the other.

The current study tests several implications of this biosocial model that combines GST with a GxE approach. First, I test the extent to which individuals with a particular variant of 5-HTTLPR show higher or lower ratings of depressive symptoms than the comparison group based on exposure to strain. Second, I test whether this gene-environment interaction shapes the prevalence of criminal offending. Finally, in line with predictions based on GST, I test whether levels of depressive symptoms mediate the relationship between this gene-environment interaction and criminal offending. There is evidence based on recent research in the GxE

literature that the simultaneous focus on both psychological and behavioral outcomes is warranted, as genes are related to behaviors like criminal offending because they influence brain processes related to attention and learning (Schilling et al. 2011; Simons et al. 2011). I test my predictions using data from the National Longitudinal Study of Adolescent Health (Add Health). In the following sections, I will explicate my theoretical model more fully and describe the data set and methods in more detail.

STRAIN, NEGATIVE EMOTIONS, AND CRIMINAL OFFENDING

GST was proposed by Robert Agnew (1992) as an individual level, social psychological explanation of crime and delinquency. The theory is an expansion of traditional anomie/strain theories, which primarily focus on an individual's ability to achieve culturally valued economic and class-based status goals, and the stress caused when individuals don't reach these goals (Cloward & Ohlin, 1960; Cohen, 1955; Merton, 1938). Agnew added to these threatened or actual experiences of not achieving highly valued goals two other types of strain likely to lead to deviant and criminal coping: the loss of valued persons or objects and the presentation of noxious (negative) stimuli. This specific focus on the loss of valued persons or property and the presentation of noxious stimuli as potentially important strains comes from the literature on stress (Mirowsky and Ross 2003; Pearlin 1989). Further theorizing and research in the GST literature has suggested that strains that are high in magnitude, seen as unjust or undeserved, occur in settings with low social control, and that incentivize crime are more likely to lead to deviant and criminal coping responses (Agnew 2001).

A further expansion of traditional anomie/strain theories found in Agnew's GST is the emphasis on negative emotions as a mediating mechanism between strain and deviant coping. According to Agnew, strains are important because they lead to the development of negative

emotions, such as anger, depression, fear, shame, and anxiety. These negative emotions need to be dealt with, which can lead to deviant coping on the part of individuals. While individuals have many different ways to cope with negative emotions, the likelihood that they will cope with these emotions through deviant methods is conditioned by any number of factors. The most commonly identified factors in the GST literature are sociological variables, such as religiosity, affiliations with delinquent peers, and self-control (Agnew 2006).

In the current study I operationalized strain as having experienced the suicide or suicide attempt of a friend or family member in the past 12 months. Suicides and suicide attempts are often unexpected, traumatic, and violent (Schofield and Ratnarajah 2007). Suicidal behavior by friends and family thus captures both the threatened or actual loss of valued persons and the presentation of noxious stimuli. Past research has demonstrated the validity of friends and family suicidal behavior as a measure of strain. For example, the literature on the aftermath of suicide has shown that experiencing the death by suicide of a parent or close peer during adolescence can have various consequences for psychological functioning, most significant among them being higher risks for post-traumatic stress disorder and depression (Brent et al. 1995; Burton et al. 1994; Schofield and Ratnarajah 2007; Shepherd and Barraclough 1976; Simone 2008; Wilcox et al. 2010). In addition, studies have shown that experiencing the suicide of a loved one in childhood or adolescence, most especially a parent, can have long term consequences for antisocial behavior, including increased aggression and substance use (Cerel 2000; Pfeffer et al. 1997; Wilcox et al. 2010).

While the nature of the link between the suicidal behavior of parents and friends and negative emotional states like depression seems fairly obvious, the link between suicide by others and antisocial behavior is less so. The reason suicidal behavior by friends and family is

consequential for offending has to do with its timing. Experiencing this type of strain during childhood or adolescence makes it consequential for antisocial behavior. Children and adolescents who experience the actual or attempted suicide of a friend or family member may internalize the experience if others surrounding the bereaved don't specifically engage the individual in order to guide them through the bereavement process (Schofield and Ratnarajah 2007). In addition, experiencing the suicide or suicide attempt of a loved one puts strains on one's personal relationships and can lead to interpersonal conflict (Cerel 2000). Overall, the literature suggests that antisocial behavior is one part of an overarching psychopathology that can develop after experiencing suicidal behavior by friends and family, most especially if bereavement care is not readily available (Cerel 2000; Schofield and Ratnarajah 2007; Wilcox et al. 2010). Suicidal behavior by friends and family should thus be considered a strain worthy of study in the GST literature that can affect both negative emotions and antisocial behavior.

Almost twenty years of research have provided much empirical support for Agnew's theory, and have established it as one of the more popular theories of crime and delinquency (Agnew 2002; Langton and Piquero 2007; Mazerolle, Piquero, and Capowich 2003; Piquero and Sealock 2004). However, a key proposition in GST, that the strain-negative emotions-crime relationship might be conditioned by outside factors, has been infrequently tested. In addition, when this proposition has been tested the focus has been on other elements of the social environment (Jang and Johnson 2003, 2005), and there has thus far been little effort to integrate genetics as a potential conditioning factor into the strain-negative emotions-crime relationship. For Agnew and others, the variables most likely to condition the strain-negative emotions-crime relationship are drawn from other prominent theories of crime (social control, social learning, and self-control theory, in particular). For example, the individual who associates with many

delinquent peers may be more likely to engage in deviant coping, while an individual who is firmly attached to their family and school may be less likely to do so. Similarly, respondents with low self-control may be less able to effectively cope with significant strains and more inclined to respond in an antisocial manner.

In the current study I argue that genetics is a factor that could condition the traditional GST model. Could it be that some individuals, because of their genetic makeup, are more or less likely to react to strain by developing negative emotions and engaging in deviant coping responses to these emotions? I will now turn to a discussion of the issue of moderation of environmental effects (in this case, strains) on psychological functioning and behavior by genes.

GENE-ENVIRONMENT INTERACTIONS: 5HTTLPR, STRAIN, DEPRESSION, AND ANTISOCIAL BEHAVIOR

Research exploring the genetic basis of neuropsychological functioning and antisocial behavior has tended to focus on variations in genes responsible for regulating neurotransmitters, such as serotonin and dopamine. One gene that has received extensive attention in this literature is the serotonin-transporter-linked polymorphic region (5-HTTLPR).

5-HTTLPR is a degenerate repeat polymorphic region in SLC6A4, which is the gene that codes for the transportation of serotonin. This makes 5-HTTLPR an important gene in studies of depression and antisocial behavior, because serotonin is thought to be linked to feelings of happiness and well-being (Belsky and Pluess 2009). A polymorphism in the promoter region of 5-HTTLPR means there are two variants, the short (s) and long (l) alleles. The s allele has been associated with reduced expression of the serotonin transporter molecule, and those with two copies of the s allele (s/s) have been shown by prior research to be the most vulnerable to displaying psychological and behavioral issues when having to deal with adversity (Belsky and Pluess 2009).

Recent research has shown that 5-HTTLPR moderates the effects of environmental stressors on both depression and antisocial behaviors. In a groundbreaking study, Caspi et al. (2003) showed that 5-HTTLPR moderated the effects of stressful life events in early adulthood on depressive symptoms and the probability of suicidal ideation and attempts and episodes of major depression at 26 years of age. Those homozygous for the s-allele (s/s) proved the most adversely affected, while l-allele (l/l) homozygotes showed greater resilience, with little to no effect of stressful life events on depression among this group. Taylor et al. (2006) reported similar results in a sample of young adults, finding the same pattern when observing the effect of problematic child-rearing history and recent negative life events on depressive symptomatology. A similar pattern involving 5-HTTLPR and depression emerged in Eley et al.'s (2004) study on adolescent girls who were and were not exposed to risky family environments and in Brummett et al.'s (2008) investigation of middle-aged and aging adults who had and hadn't served as the primary caregiver of a relative with Alzheimer's disease.

While research on 5-HTTLPR has tended to focus on depression, several investigators have recently examined its association with aggressive and antisocial behaviors. Similar to the research on depression, this line of studies has found that 5-HTTLPR moderates the effect of environmental stressors on aggressive and antisocial behaviors. For example, recent studies have shown that carriers of the s-allele are more at risk for aggression and violent criminal behavior when presented with an adverse environment (Reif et al. 2007; Retz et al. 2008; Verona et al. 2006). Most recently, Simons et al. (2011, 2012) showed that 5-HTTLPR (along with other genes) interacted with a composite measure of favorable and adverse environmental factors to effect anger, orientations toward hostility, and aggression.

In summation, recent research has shown that carriers of the s-allele of 5-HTTLPR, but most especially those homozygous for the s-allele, are at greater risk for depression, anxiety disorders, and antisocial behavior when presented with environmental stressors. But how do genes cause some individuals to be at risk when presented with negative stimuli like poor parenting and victimization? Recent theorizing by Belsky and his colleagues (Belsky 1997, 2005; Belsky and Pluess 2009; Belsky et al. 2009) have argued that this is because certain forms of a gene make individuals more susceptible to environmental influence, for both good and bad. Belsky and Pluess (2009) observed that the genes most often studied in the GxE literature are involved in the serotonergic and dopaminergic systems. 5-HTTLPR is directly linked to serotonin activity in the brain, and the serotonergic system is implicated in sensitivity to punishment and displeasure. This means that certain individuals are more responsive to their environment because of their different thresholds for experiencing pleasure and displeasure. Essentially, some people are, because of their genetic makeups, more readily shaped by environmental rewards and punishments than others. In the terms of GST, this means that some individuals may be more influenced by criminogenic strains than others, and their behavior may be more affected by these kinds of experiences. Holding constant the constraints placed on individuals to not respond to strain with antisocial behaviors, the person homozygous for the s-allele of 5-HTTLPR may be more likely than the person who is not to experience depression, and thus perhaps engage in more coping behaviors, whether legal or illegal, when they do experience strain.

THE CURRENT STUDY

The current study extends both criminological theory and GxE studies in two important ways. First, I integrate a GxE approach in an important theory of crime and delinquency, general

strain theory. All three of the key elements present in GST, strain, negative emotions, and criminal offending have been present in the GxE literature, and they have all been specifically tied to 5-HTTLPR. This makes the integration of these two literatures a logical step and the testing of a combined model is a natural extension of prior theorizing and research that benefits both the criminological literature and the GxE literature.

Second, my specific focus on GST is important because it allows me to tie together previous findings concerning 5-HTTLPR, stressful events, depression, and antisocial behaviors. While prior research has looked at the effects that the combination of 5-HTTLPR and stressful events has on depression and antisocial behavior separately, this study will be the first to my knowledge to look at both outcomes in a combined model. Utilizing GST makes this combined mediated moderator model a sensible way to integrate past findings concerning 5-HTTLPR into one unified study. I can examine the effects that the combination of 5-HTTLPR and strain have on depression and criminal behavior, and then determine how much of the effect on criminal behavior is mediated by depression, per the expectations of GST.

Based on prior research in the GxE literature I expect that 5-HTTLPR genotype will condition the theoretical model of strain-negative emotions-crime that is identified in GST. Specifically, I expect that those who are homozygous for the s-allele will experience both more depression and engage in more offending after experiencing the death or near death of a friend or family member to suicide than will individuals with some other allelic combination of 5-HTTLPR (l/l, s/l, l/s). In addition, I expect that the friends and family suicidal behavior by 5-HTTLPR effect on offending behavior will be mediated by depressive symptoms, per the expectations of GST. The friends and family suicidal behavior by 5-HTTLPR effect on

offending will thus act as a mediated moderator model that combines GST with previous findings in the GxE literature, with depressive symptoms acting as the mediating variable.

I draw on these expectations as well as GST and prior GxE theorizing to derive specific hypotheses about the relationship between 5-HTTLPR, strains, depression, and criminal offending. *Hypotheses 1 & 2* predict that suicidal behavior on the part of friends and family will have a significant and positive effect on both depressive symptoms and criminal offending, respectively. Drawing on past research on 5-HTTLPR, *hypothesis 3* predicts that 5-HTTLPR will moderate the effects of friends and family suicidal behavior on depressive symptoms such that those who are homozygous for the s-allele will experience more depressive symptoms when presented with adversity than non-s-allele homozygotes. *Hypothesis 4* makes an identical prediction to *hypothesis 3*, with the substitution of criminal offending as the outcome of interest. Lastly, drawing on GST, *hypothesis 5* predicts that depressive symptoms will significantly mediate the effect that the interaction between 5-HTTLPR and friends and family suicidal behavior has on criminal offending. Next, I will describe the data set, measures, and methods used to test these hypotheses.

DATA AND METHODS

Data

For the current study I draw on data from the National Longitudinal Study of Adolescent Health (Add Health). Add Health is a nationally representative sample of American adolescents who were first recruited during the 1994-1995 school year while they were in grades 7-12 (Harris et al. 2003; Udry 1998). Add Health obtained a nationally representative sample of adolescents by utilizing a multistage stratified sampling process to select 80 high schools and 52 middle and junior high schools for inclusion in the study. More than 90,000 students completed

in-school self-report surveys, and of this group a subsample was randomly chosen for the Wave I in-home component of Add Health. In total, 20,745 adolescents and 17,700 of their primary caregivers participated in the Wave I in-home component (Harris et al. 2003). Wave II data collection occurred approximately 1 to 2 years after Wave I data collection, Wave III data was collected during 2001-2002 when respondents were between 18-26 years old, and Wave IV data was collected during 2007-2008 when respondents were between 24-32 years old.

During Wave IV in-home interviews Add Health collected a number of types of biological data. Among the data collected, Add Health took saliva swabs from all Wave IV interviewees for DNA analysis. In conjunction with the Institute for Behavioral Genetics (IBG) in Boulder, CO, Add Health genotyped Wave IV interviewees for a set of genetic markers of interest to biosocial researchers. That Add Health is a large and nationally representative data set that contains variables measuring both the social environment and genetics makes it highly desirable for the current study. The current study includes male Add Health respondents who were interviewed at Waves I, II, and IV who did not have any missing genetic data. After dealing with missing data, the current study includes the analysis of information gathered from 3,581 male respondents in the Add Health data set.

Measures

Criminal Behavior

The dependent variable consists of 9 items drawn from Wave II that asked respondents about various criminal activities they have engaged in during the prior year. These items are a mixture of both violent and property offending. Measures of violence included questions asking how often respondents used or threatened to use a weapon on someone to get something from them, pulled or actually used a knife or gun on someone, used a weapon during a fight, or hurt

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someone so badly in a fight that they needed medical attention. Property offending measures

included asking respondents how often they painted graffiti, stole cars, burglarized buildings,

and stole items worth more than $50. Inspection of the distributions for each item revealed

extensive skew, with relatively few respondents reporting frequent involvement. I thus recoded

the 9 items into binary measures of whether the respondent reported engaging in these behaviors

in the past year (1=yes), and summed them into a measure of criminal behavior that emphasizes

the prevalence of offending (alpha = .70). This type of scaling has been recommended by

researchers in the past (Hindelang, Hirschi, and Weis 1981; Osgood, McMorris, and Potenza

2002), and this particular scale is closely related to others that researchers have developed for use

in the Add Health data set in past studies (Guo et al. 2008; Hagan and Foster 2003; Haynie 2001,

2003; Kaufman 2009).

GST Measures

I include one measure of serious strain from Wave I: suicidal behavior by friends and

family. In the terms of GST, suicidal behavior by friends and family captures both the

threatened loss of valued persons and the presentation of noxious stimuli. Previous research on

GST indicates that these kinds of serious strain are more likely to lead to crime and deviance

(Agnew 2006). Suicidal behavior by friends and family combines four items that asked

respondents if any friends or family members tried to kill themselves (or succeeded in killing

themselves) during the past 12 months. While the original questions asked separately if

respondents had friends or family who had attempted or completed suicide, very few respondents

had a friend or family member that had attempted or completed suicide, so all four measures are

combined into one measure. This results in a 0/1 item reflecting whether a respondent had

neither a friend or family member attempt or complete suicide (0) or either a friend or family

member or both a friend and family member attempt or complete suicide (1) in the past 12 months.

As discussed above, GST predicts that strain effects on crime may partly operate through negative emotions, including depressive symptoms. To assess this possibility I measure depressive symptoms with 19 items from the CES-D depression scale, administered during wave I interviews (range 0 to 57; alpha = .83). This scale includes items that ask about feelings of loneliness, fear, sadness, fearfulness, and depression in the week before being interviewed, with possible responses ranging from 0 (never or rarely) to 3 (most of the time or all the time). The use of depressive symptoms in the current study is consistent with recent theorizing and research on GST (Brezina 1996; Broidy 2001; Jang and Johnson 2003; Kaufman 2009), as well as prior research on the combination of 5-HTTLPR and stressful life events (Carver et al. 2011; Caspi et al. 2003; Goldman et al. 2010).

5-HTTLPR

Four genetic polymorphisms were genotyped and included in the Wave IV Add Health genetic sample. Since the current study is focused on the relationship between strain, depression, and offending, I focus on one of these genes: 5-HTTLPR. The genotype at 5-HTTLPR located on chromosome 17q11.1-q12 has a functional polymorphism in the variable repeat sequence in the promoter region (Bradley et al. 2005; Simons et al. 2011). Prior research on 5-HTTLPR has focused on three variants of the gene: those homozygous for the long allele (l/l), those carrying one short allele (s/l, l/s), and those homozygous for the short allele (s/s).

Those homozygous for the short allele have been shown by past research to be the most reactive to environmental stressors (Caspi et al. 2003; Manuck et al. 2004). Accordingly, I coded the 5-HTTLPR variable to reflect the non-presence (0) or presence (1) of two s-alleles.

Based on this coding the distribution for 5-HTTLPR was such that 71.7% of respondents were not s-allele homozygotes (l/l, s/l, l/s), and 28.3% were homozygous for the s-allele. The Hardy-Weinberg equilibrium test showed that the distribution of 5-HTTLPR did not differ significantly from that predicted on the basis of simple Mendelian inheritance.

Controls

I also include several general controls in all analyses that have been shown to be predictive of involvement in crime: Age, dummy variables for Hispanic, non-Hispanic Black, Native American, Asian, and Other (with non-Hispanic White as the reference category), parent's education (1=4 year degree or more), and parent receiving public assistance (1=yes). About 55.8% of the sample is non-Hispanic white, 14.9% Hispanic, 17.8% non-Hispanic black, with the remainder comprising Native American (2.6%), Asian (7.7%), and members of other racial/ethnic groups (1.1%). About 27.4% of parents are college graduates, while 7% report receiving public assistance.

Analytic Strategy

I test hypotheses derived from GST and the differential susceptibility perspective using Ordinary Least Squares (OLS) and Negative Binomial (NB) regression techniques where appropriate. The models test whether the interaction of suicidal behavior by friends and family and 5-HTTLPR significantly affect depressive symptoms, whether this interaction has direct effects on self-reported criminal behavior at Wave II, and whether this relationship is mediated by scores on the depressive symptoms scale. I utilize NB regression techniques when looking at the key dependent variable of interest because it is a highly skewed count measure, thus violating the assumption of normality required for OLS regression (Gardner, Mulvey, and Shaw 1995). I utilize the appropriate weight, cluster, and strata variables in all analyses to account for the

complex Add Health survey design. Tests using Variance Inflation Factors (VIFs) showed that multicollinearity was not a problem in any of the equations.

RESULTS

Table 3.1 presents the descriptive statistics for the study variables for the entire sample. As expected, criminal offending levels are fairly low. The average respondent reported committing less than one of the nine acts in the criminal behavior index. Very few respondents reported that a friend, family member, or both had either attempted or completed suicide at Wave I (approximately 14.3% of the sample). Additionally, the average respondent reported fairly low levels of depressive symptoms at Wave I. The average respondent in the sample was about sixteen years old at the time of Wave II data collection. Table 3.2 further describes the data by presenting means and mean comparisons for the two genetic subgroups. Importantly, there are no mean differences between s-allele homozygotes and individuals with other allelic combinations for 5-HTTLPR in the key dependent and independent variables of criminal behavior and friends and family suicidal behavior, respectively. However, several mean differences can be noted, namely depressive symptoms and several racial/ethnic differences. S-allele homozygotes report slightly higher mean levels of depressive symptoms than do individuals who carry some other allelic combination (l/l, s/l, and l/s). Among the racial/ethnic differences, there are significantly fewer non-Hispanic whites and blacks in the s/s subgroup, while there are significantly more Hispanics, Asians, and individuals who identify as "other" in the s/s subgroup. Prior research has also shown racial and ethnic differences in the distribution of 5-HTTLPR (Kunugi et al. 1997).

Table 3.3 presents the correlation matrix for the study variables, along with means and standard errors. The correlation matrix serves to check for gene-environment correlations (rGE).

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Gene-environment correlation refers to a nonrandom distribution of environments among different genotypes (Simons et al. 2011). These gene-environment correlations can potentially confound GxE effects (Guo, Roettger, and Cai 2008; Guo, Tong, and Cai 2008). Table 3.3 shows that there is not a significant correlation between suicidal behavior by friends and family and 5-HTTLPR genotype. This suggests an absence of rGE effects in these data.[3] As expected, suicidal behavior by friends and family is significantly and positively related to depressive symptoms at Wave I and criminal behavior at Wave II. Of note as well, 5-HTTLPR genotype does not significantly correlate with depressive symptoms or criminal behavior. Also, as expected, the depressive symptoms scale is significantly and positively correlated with criminal behavior.

To assess the possibility that suicidal behavior by friends and family and 5-HTTLPR genotype interact to effect depression, table 3.4 presents OLS regression models with Wave I depressive symptoms regressed on friends and family suicidal behavior, 5-HTTLPR genotype, and controls. Model 1 examines the direct effects of friends and family suicidal behavior and 5-HTTLPR genotype on depressive symptoms, while controlling for various other variables. This model shows that as predicted by GST suicidal behavior by friends and family has a highly significant and positive effect on depressive symptoms, supporting hypothesis 1. Similar to most past research on genes and depression, 5-HTTLPR genotype does not exert a direct effect on depressive symptoms.

Model 2 incorporates the interaction between friends and family suicidal behavior and 5-HTTLPR genotype into the model. The interaction term is significant and positive, suggesting that compared to individuals who not homozygous for the s-allele of 5-HTTLPR, those who are

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[3] In addition, an independent samples t-test confirmed no mean-difference in suicidal behavior by friends and family between those who were and were not homozygous for the 5-HTTLPR s-allele.

experience more depressive symptoms when confronted with a strain like suicidal behavior by

friends and family, supporting hypothesis 3. The direct effect of friends and family suicidal

behavior remains highly significant in this model. So while experiencing the death or near death

of a friend or family member or both due to suicide adversely effects everyone in this sample, the

effect is almost twice as big for those who are homozygous for the s-allele (from model 2 in table

3.4, the coefficient for friends and family suicidal behavior plus the interaction term coefficient).

Among the controls, there are significant contrasts among different racial groups in both models,

with Hispanics, blacks, and Asians reporting higher levels of depressive symptoms than non-

Hispanic whites. In addition, individuals whose primary caregiver reported having a college

degree report lower levels of depression, individuals whose primary caregiver reported being on

public assistance at W1 report more depression, and older respondents report more depression.

Turning to criminal behavior, table 3.5 presents NB regression models with Wave II

criminal behavior regressed on friends and family suicidal behavior, 5-HTTLPR genotype, and

controls. The baseline model in model 1 shows that suicidal behavior by friends and family has

a significant and positive effect on criminal behavior, supporting hypothesis 2. Like with

depression, 5-HTTLPR genotype does not affect criminal behavior. Amongst the controls, there

is one racial difference in offending, with Native Americans reporting significantly more

offending behavior than non-Hispanic whites. Additionally, the children of parents with a

college degree report less criminal behavior. Model 2 introduces the GxE term again, and much

like with depression this interaction of friends and family suicidal behavior and 5-HTTLPR

genotype has a significant and positive effect on criminal offending, supporting hypothesis 4.

The direct effect of friends and family suicidal behavior also remains positive and highly

significant. So much like with depression, experiencing the death or near death of a friend or

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family member or both due to suicide increases subsequent criminal behavior among everyone in this sample, but this effect is almost twice as large for those who are homozygous for the s-allele (from model 2 in table 3.5, the coefficient for friends and family suicidal behavior plus the interaction term coefficient).

As a test of the full strain-negative emotions-offending model proposed by GST, the depressive symptoms scale is introduced in model 3 as a potential mediator of the GxE effect on criminal behavior. As predicted and supporting hypothesis 5, the depressive symptoms scale fully mediates the GxE term. The GxE term is no longer significant at the .05 level, and the coefficient has been reduced by approximately 15.2%. By contrast, the direct effect of friends and family suicidal behavior remains highly significant, while the coefficient has been reduced by approximately 12.8%. In these analyses at least, it appears that the biosocial GST model tested provides more support for the mediating model found in the theory than the traditional GST model that does not account for genetic effects. While the strain-offending relationship has not been fully mediated by depressive symptoms, the strain-by-5-HTTLPR GxE effect on offending has been fully mediated by this construct.

DISCUSSION

In this paper I sought to test whether genetic factors, specifically 5-HTTLPR genotype, moderate the relationship between strain, negative emotions, and criminal behavior as specified by general strain theory (GST). To explore this possibility I utilized data from the Add Health study (Udry 2003) that measured adolescents' experience of strain, here represented by whether a friend or family member of the respondent had attempted or completed suicide in the last year, depressive symptoms, criminal offending, 5-HTTLPR genotype, and numerous control variables. These data serve to examine whether 5-HTTLPR genotype moderates the effect of strain on both

depressive symptoms and criminal offending, and whether the latter relationship is mediated by depressive symptoms. The results of both OLS and Negative Binomial regression models reveal a significant GxE, where male adolescents who are faced with the actual or near loss of valued persons to suicide are more likely to experience depressive symptoms and act out in antisocial ways if they are homozygous for the s-allele of 5-HTTLPR versus some other allelic combination (l/l, s/l, l/s). In addition the GxE effect on criminal behavior is fully mediated by the scale of depressive symptoms. Importantly for establishing a GxE rather than an rGE, the correlation matrix shows that 5-HTTLPR genotype does not significantly correlate with suicidal behavior by friends and family, depressive symptoms, or criminal behavior. Before turning to the implications of this study, the limitations within should be noted.

While the current study makes important contributions to the GxE and criminological literatures, some limitations should be noted. First, the measure of strain in these analyses is limited to the attempted or completed suicide of a respondent's friends and family. While this is a very serious strain that past research has tied to both experiences of depression and antisocial behavior (Brent et al. 1995; Burton et al. 1994; Cerel 2000; Kaufman 2009; Pfeffer et al. 1997; Schofield and Ratnarajah 2007; Shepherd and Barraclough 1976; Simone 2008; Wilcox et al. 2010), other candidate strains should be explored as well. For right now, the support provided for the integrated biosocial GST model tested in this study should considered tentative until further research explores more strains and how their effects on negative emotions and offending are condition by 5-HTTLPR. Second, while the measure of depressive symptoms includes items tapping emotions like fear, anxiety, and guilt; these emotions may be worth exploring on their own. As previously mentioned, other neuropsychological deficits, like ADHD, and other negative emotions, such as anger, could use a full examination in future GST models that

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account for genetic effects. The final key limitation is that while this study is based on a nationally representative data set, only males are included in these models, meaning that these results are not generalizable to all adolescents.[4] This study is notable, however, because the sample size (N = 3,581) is much larger than has typically been available in GxE research.

These results tie together nicely previous findings that separately considered the relationship between 5-HTTLPR and depression and 5-HTTLPR and antisocial behaviors. Indeed, these results suggest that much of the recently established relationship between 5-HTTLPR and antisocial behaviors may work through depression or other negative emotions. 5-HTTLPR may matter for violent and delinquent behaviors precisely because it is so strongly related to depression and other neuropsychological deficits. In the terms of GST, the s-allele of 5-HTTLPR is a conditioning factor that makes negative emotions and antisocial behaviors a more likely outcome of experiencing strain.

Future studies would do well to consider the potential role of other neuropsychological deficits in mediating GxE effects consisting of the interaction of 5-HTTLPR with various strains on criminal behavior. One likely candidate is attention deficit/hyperactivity disorder (ADHD). The exact cause of ADHD is unknown, but it is believed to have a strong genetic component (Bobb et al. 2005). 5-HTTLPR is one of the genes that figures prominently in the literature on gene-environment interactions, ADHD, and antisocial behavior, and this literature has focused on the interaction of strains, such as child abuse, with genotype and resulting levels of ADHD symptomatology (Ficks and Waldman 2009; Kim-Cohen et al. 2006). ADHD, as well as other neuropsychological deficits and various negative emotions, could also serve to connect the interaction between strain and 5-HTTLPR to antisocial and criminal behaviors.

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[4] Separate models not presented were run for females, with these models not showing any significant GxE effects. Results available upon request.

In conclusion, this study provides evidence that experiencing the death or near death of a friend or family member interacts with 5-HTTLPR genotype to increase risks for experiencing depressive symptoms and engaging in criminal behavior among males. These findings tentatively support the utility of GST as an explanation of the previously disparate findings concerning the combination of stressful life events and 5-HTTLPR genotype and their effect on depression, other neuropsychological deficits and antisocial behaviors. Future research would do well to further probe the kinds of strains that interact with 5-HTTLPR genotype to effect neuropsychological functioning and behavior, while also exploring other neuropsychological deficits that could potentially be affected by the combination of strain and 5-HTTLPR genotype. Theoretically, this study is important in showing the utility in combining traditional criminological theories with a biosocial modeling approach. This kind of theory building and modeling strategy is important because it helps further develop ideas centered on the concept that the environment and biology are always interacting to shape how we experience and react to our world. Moving forward, criminology as a discipline should focus on and consider central this kind of theory building and modeling.

CHAPTER 4

DAT1, DELINQUENT PEERS, AND CRIMINAL BEHAVIOR

One of the strongest and most consistent correlates of crime and delinquency in the criminological literature is affiliating with delinquent peers (Akers 1998; Akers and Sellers 2009; Haynie 2001, 2002; Pratt and Cullen 2000). Various measures of delinquent peers have been found to predict a wide variety of criminal, drug-using, and generally antisocial behaviors (Akers and Sellers 2009; Warr 1998). Indeed, the delinquent peers-delinquency link is so well established and has been so consistently reproduced that it is easily one of the most taken-for-granted empirical realities in the criminological literature (Akers and Sellers 2009; Warr 2002).

Importantly though, there have not been consistent attempts to identify the factors that might condition the effects that delinquent peer affiliation has one's own involvement in criminal and delinquent behaviors. Not surprisingly, most of the research in the criminological literature that has probed potential factors that condition the delinquent peers-delinquency relationship has focused on important sociological variables, like race (Haynie and Payne 2006), network structures (Haynie 2001, 2002) and intimate relationships (Seffrin et al. 2009). What has thus far gone understudied is the potential role that biological factors play in effecting the delinquent peers-delinquency connection. The current study examines the extent to which genetic factors may moderate the effect that delinquent peer affiliations have on one's own involvement in delinquency. Specifically, this study explores the way in which particular variants of the dopamine transporter gene (DAT1) interact with associations with substance-using peers to affect one's own level of delinquent behavior in a sample of males. Recent research has shown

that DAT1 is related to criminal offending and general antisocial behavior, often by moderating the effects of key criminological variables (Beaver and Belsky 2012; Beaver, Wright, and Walsh 2008; Guo et al. 2007; Guo, Roettger, and Shih 2007; Guo, Tong, and Cai 2008; Stevens et al. 2009).

The current study expands on past criminological theorizing. Affiliation with delinquent peers is a key variable in social learning theory, one of the more highly supported theories of crime and delinquency (Akers 1998; Akers and Sellers 2009; Pratt and Cullen 2000). I integrate this theory into a biosocial framework whereby antisocial outcomes are the result of an interaction between one's social environment (here, affiliating with substance-using peers) and genetics. This combining of social learning theory and the gene-environment interactions (GxE) literature is a natural extension of recent work in a number of fields that have demonstrated that certain genes, in combination with certain environmental factors, are related to outcomes like aggression and criminal offending (Caspi et al. 2002; Guo, Roettger, and Cai 2008). I test my integrated model using data from the National Longitudinal Study of Adolescent Health (Add Health). In the following sections, I will review the literature on delinquent peers and social learning theory, and describe how genetics fit into this literature.

DELINQUENT PEERS AND OFFENDING

Sutherland's (1947) theory of differential association asserts that affiliations with delinquent peers affect one's own offending propensity by encouraging deviant attitudes and providing delinquent models of behavior. In this theory delinquency is the result of an attitude transference, whereby one adopts and absorbs the attitudes of their peers (Warr 2002). Burgess and Akers (1966) extended this idea, proposing a "differential association-reinforcement" theory that combined the work of Sutherland with the principles of operant conditioning. This theory

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argues that individual offending is not only influenced simply by affiliating with delinquent peers and the behavioral models and antisocial attitudes these relationships provide, but also by the reinforcing rewards as opposed to punishments that criminal offending provides within these kinds of peer groups. This theory, later developed further by Akers (1985, 1998; Akers and Sellers 2009), remains the foundation of social learning theory in criminology, and affiliations with delinquent peers remains a key variable in the social learning theory literature.

Empirical research has produced overwhelming support for a direct effect of delinquent peers on one's own criminal and delinquent behaviors (Akers and Sellers 2009; Pratt and Cullen 2000; Warr 1998, 2002). However, only a small number of studies have to date attempted to identify the factors that mediate, moderate, or otherwise condition the delinquent peers-delinquency relationship. Thus far, the research that has been done in an attempt to identify these conditioning factors has focused solely on sociological variables. For example, Haynie (2001; 2002) has conducted research that examined the relative placement of individuals and their delinquent peers in network structures, and the effects of this placement on one's own delinquency. In another study, Haynie and Payne (2006) identified that the way race structures friendship networks contributes to the gap in offending between different racial groups, essentially identifying differences in exposure to delinquent peers as an important contributor to the racial gap in offending.

However, recent research in the gene-environment interaction (GxE) literature suggests a potential explanatory factor in explaining the delinquent peers-delinquency relationship lies in a biosocial framework centered on both the social environment and genetics. Recent scientific breakthroughs have made it clear that genetics plays more of a role in behavior than social scientists had previously believed. In the following section, I will review the recent research on

the dopamine transporter gene (DAT1) and antisocial behaviors and the potential role that delinquent peers plays in this relationship.

<center>DAT1, DELINQUENT PEERS, AND OFFENDING</center>

Research probing the genetic basis for antisocial behaviors has centered on genes involved in the regulation of neurotransmitters, such as dopamine. One such gene involved in the transportation and maintenance of dopamine that has received increasingly greater attention in this literature is the dopamine transporter gene, DAT1. DAT1 is largely responsible for the magnitude and duration of synaptic dopamine signaling in the brain. This is accomplished by transporting dopamine from the synaptic cleft back into the presynaptic knob for repackaging and reuse after it has finished exciting downstream neurons (Schilling et al. 2011). A number of allelic variations exist for DAT1, with the two most common being the 9-repeat and 10-repeat alleles. The 10R allele is the most efficient in this reuptake process compared to other alleles. What this means is that there is less dopamine in the synaptic cleft available for activation (Miller-Butterworth 2008). This is problematic because dopamine activates pleasure centers in the brain, so when it is cleared too rapidly from the synaptic cleft the individual is led to seek out pleasures to increase dopamine levels, whether legal or illegal. In addition, dopamine levels in the brain are related to reward-seeking behavior.

Numerous studies have looked at the relationship between DAT1, the social environment, and sensation-seeking and antisocial behaviors. Stevens et al. (2009) found that DAT1 moderated the effect of institutional deprivation on ADHD symptoms in a sample of children in Romanian orphanages, with those carrying 10R alleles showing more ADHD symptoms at 6, 11, and 15 years of age. Guo, Tong, and Cai (2008) found that the 10R allele of DAT1 had direct effects on the number of sexual partners that white males reported having in the Add Health

<center>77</center>

sample. Guo et al. (2008) found in the same study that the proportion of students in one's school who were having sex by age 16 exacerbated this relationship. Various other studies have produced similar results concerning DAT1 and antisocial outcomes (Beaver, Wright, and Walsh 2008; Guo et al. 2007; Guo, Roettger, and Shih 2007).

Interestingly, there have also been a number of studies that have identified DAT1 and the combination of DAT1 and risky environments as risk factors for exposure to delinquent peers and the formation of delinquent peer groups. While much of the research into how individuals meet delinquent peers and come to form delinquent peer groups has focused on the direct and indirect roles of family (Hirschi 1969) and proximity to delinquent peers (Warr 2002), more recent research has focused on the possibility that genes may underlie propensities for associating with antisocial peers. Using a direct measure of peer delinquency, Cleveland, Wiebe, and Rowe (2005) found that as much as 64% of the variance in delinquent peer affiliations was explained by genetic factors, including DAT1. In an important finding concerning GxE effects on delinquent peer group formation, Beaver, Wright, and Delisi (2008) found that the 10R allele of DAT1 interacted with high-risk family environments to predict associating with substance-using peers. Expanding on this finding, Yun, Cheong, and Walsh (2011) found similar results looking at the 10R allele of DAT1, risky family environments, and delinquent peer associations while utilizing a direct measure of peer delinquency.

Taking into account prior findings concerning DAT1, antisocial behaviors, and self-selection into delinquent peer groups a logical step to take is to test whether the effect of delinquent peer affiliations on one's own offending behavior is moderated by DAT1. This is a gene that may be important for directing youth towards affiliating with delinquent peers, but does it then also moderate the effects that these affiliations have on one's own offending? Could

DAT1 be related to social selection processes determining whether one engages with delinquent peers, while also being involved in the socialization processes whereby delinquent peers influence one's subsequent delinquent behavior? There is reason to believe that this could be the case, based on recent theorizing in the GxE literature.

Belsky and his colleagues (Belsky 1997, 2005; Belsky and Pluess 2009; Belsky et al. 2009) have argued that certain forms of genes like DAT1 make individuals more susceptible to environmental influence, for both good and bad. Belsky and Pluess (2009) observed that the genes most often studied in the GxE literature are involved in the dopaminergic system. DAT1 is directly linked to dopamine activity in the brain, and the dopaminergic system is a powerful regulator of reward-seeking behavior. Therefore, some people may simply be more receptive to the kinds of learning they receive from their delinquent peers and may be more readily reinforced in their own delinquent behavior by their delinquent peers. In short, delinquent peers may be a more powerful influence and learning tool for some individuals because of their genetics.

THE CURRENT STUDY

The current study extends both criminological theory and GxE studies. I integrate a time-tested measure in the criminological literature, affiliations with delinquent peers, into a biosocial, GxE approach to explaining criminal and delinquent behaviors. The delinquent peers-offending relationship is one of the most commonly cited in the criminology literature, DAT1 has been tied to criminal and general antisocial behaviors, and some recent research has suggested that the DAT1 gene is implicated in selection into delinquent peer groups. A further logical step is to test whether the DAT1 gene moderates the effects that delinquent peer affiliations have on one's own

offending patterns. This study will benefit theorizing in both the criminology and GxE

literatures.

Based on prior research I expect that the effect of delinquent peers on one's own

offending behavior should be conditioned by DAT1 genotype. Specifically, I expect that the

10R allele should condition the effect of delinquent peers in such a way that affiliating with

delinquent peers should be more consequential for offending among those individuals who carry

at least one 10R allele. In addition I expect that the number of 10R DAT1 alleles one carries, 0,

1, or 2, should be important for the delinquent peers-offending relationship. Specifically, I

expect that the sharpest contrast in the effect of delinquent peers on one's own offending should

be between those who carry 0 and 2 10R DAT1 alleles. I draw on these expectations and

previous research on DAT1, delinquent peers and offending to derive specific hypotheses

concerning the relationship between these variables. *Hypothesis 1* predicts that the interaction

between affiliating with substance-using peers and the 10R allele of DAT1 will have a

significant and positive effect on the prevalence of individual offending, even while controlling

for theoretically important control variables. In line with prior research on DAT1 and delinquent

peers, *hypothesis 2* predicts that the biggest difference in offending will be when comparing

individuals with two 10R DAT1 alleles to individuals with zero 10R DAT1 alleles. In the

following section, I will describe the data set, measures, and methods used to test these

hypotheses.

DATA AND METHODS

Data

For the current study I draw on data from the National Longitudinal Study of Adolescent

Health (Add Health). Add Health is a nationally representative sample of American adolescents

who were first recruited during the 1994-1995 school year while they were in grades 7-12 (Harris et al. 2003; Udry 1998). Add Health obtained a nationally representative sample of adolescents by utilizing a multistage stratified sampling process to select 80 high schools and 52 middle and junior high schools for inclusion in the study. More than 90,000 students completed in-school self-report surveys, and of this group a subsample was randomly chosen for the Wave I in-home component of Add Health. In total, 20,745 adolescents and 17,700 of their primary caregivers participated in the Wave I in-home component (Harris et al. 2003). Wave II data collection occurred approximately 1 to 2 years after Wave I data collection, Wave III data was collected during 2001-2002 when respondents were between 18-26 years old, and Wave IV data was collected during 2007-2008 when respondents were between 24-32 years old.

During Wave IV in-home interviews Add Health collected a number of types of biological data. Among the data collected, Add Health took saliva swabs from all Wave IV interviewees for DNA analysis. In conjunction with the Institute for Behavioral Genetics (IBG) in Boulder, CO, Add Health genotyped Wave IV interviewees for a set of genetic markers of interest to biosocial researchers. That Add Health is a large and nationally representative data set that contains variables measuring both the social environment and genetics makes it highly desirable for the current study. The current study includes male Add Health respondents who were interviewed at Waves I, II, and IV who did not have any missing genetic data. After dealing with other missing data, the current study includes the analysis of information gathered from 3,557 male respondents in the Add Health data set.

Measures

Criminal Behavior

The dependent variable consists of 17 items drawn from Wave II that asked respondents about various criminal activities they engaged in during the prior year. These items are a mixture of violent and property offending, and two items also asked about drug selling and gang activity. Measures of violence included questions asking how often respondents used or threatened to use a weapon on someone to get something from them, took part in a group fight, used a weapon during a fight, pulled or actually used a knife or gun on someone, or hurt someone so badly in a fight that they needed medical attention. Property offending measures included asking respondents how often they painted graffiti, damaged property, stole cars, shoplifted, burglarized buildings, and stole items worth more and less than $50. Inspection of the distributions for each item revealed extensive skew, with relatively few respondents reporting frequent involvement. I thus recoded the 17 items into binary measures of whether the respondent reported engaging in these behaviors in the past year (1=yes), and summed them into a measure of criminal behavior that emphasizes the prevalence of offending (alpha = .82). This type of scaling has been recommended by researchers in the past (Hindelang, Hirschi, and Weis 1981; Osgood, McMorris, and Potenza 2002), and this particular scale is closely related to others that researchers have developed for use in the Add Health data set in past studies (Guo et al. 2008; Hagan and Foster 2003; Haynie 2001, 2003).

Affiliations with Delinquent Peers

Affiliation with delinquent peers is represented in the current study by a three item index measuring a respondent's association with substance-using friends. Specifically, Add Health respondents were asked at Wave I how many of their three closest friends smoke at least one

cigarette per day, drink alcohol at least once a month, and smoke marijuana at least once a month. I summed these three items to create a measure of association with delinquent peers (alpha = .74). I transformed this scale by standardizing it to facilitate easy interpretation of the GxE interaction terms. Higher scores on this measure indicate more involvement and contact with substance-using friends.

This measure of peer delinquency focuses only on substance use, while an optimal measure would have included items tapping a spectrum of delinquent activities among one's friends. While this measure is limited in this way, substance use does correlate highly with more general delinquent behavior (Kaufman 2009), and prior research has established the predictive validity of this particular measure in the Add Health study (Beaver et al. 2008).

DAT1

While four genetic polymorphisms were genotyped and included in the Wave IV Add Health genetic sample, I focus in this paper on one of these: the DAT1 gene. DAT1 has a 40-base pair (bp) variable number of tandem repeats that can be repeated 3-11 times (Beaver et al. 2008). Past research has shown that carriers of the 10R allele of DAT1 are significantly more susceptible to a number of behavioral and psychological problems (Beaver et al. 2008; Guo et al. 2007; Schilling et al. 2011).

I coded the DAT1 variable to reflect the presence of zero, one, or two 10R alleles (0-2). The distribution for DAT1 was 6.2% for zero, 34.7% for one, and 59.1% for two 10R alleles. The Hardy-Weinberg equilibrium test showed that the distribution of DAT1 did not differ significantly from that predicted on the basis of simple Mendelian inheritance.

Controls

I include several general controls in all analyses that have been shown to be predictive of involvement in crime: Age, dummy variables for Hispanic, non-Hispanic Black, Native American, Asian, and Other (with non-Hispanic White as the reference category), parent's education (1=4 year degree or more), and parent receiving public assistance (1=yes). About 55.8% of the sample is non-Hispanic white, 14.9% Hispanic, 17.8% non-Hispanic black, with the remainder comprising Native American (2.6%), Asian (7.7%), and members of other racial/ethnic groups (1.1%). About 27.4% of parents are college graduates, while 7% report receiving public assistance.

As a check for spuriousness, I additionally control for low self-control and substance use. While the criminological literature has shown that both low self-control and affiliations with delinquent peers are significantly related to offending, some scholars have argued that low self-control predates both delinquent peer group formation and offending and thus low self-control leads some individuals to both self-select into delinquent peer groups and offend more frequently (Chapple 2005; Gottfredson and Hirschi 1990; Meldrum, Young, and Weerman 2005). If this were the case, then the delinquent peers-offending relationship would be rendered spurious. I include a 21-item low self-control scale in all models to test this possibility. This Wave I scale includes items from both the respondent and their parent measuring their temper, trustworthiness, self-centeredness, attention span, and use of rational decision making processes, among others (alpha = .71).

As an additional check for spuriousness, I control for the respondent's self-reported use of cigarettes, alcohol, and marijuana at Wave I. I include items that asked respondents if they have ever smoked cigarettes regularly, defined as having smoked at least 1 cigarette a day for a

month (1=yes), how many days did they drink alcohol in the past year, and how many times in the past month did they use marijuana.

Analytic Strategy

I test hypotheses derived from social learning theory and the differential susceptibility perspective using Negative Binomial (NB) regression techniques. These models test whether the interaction of delinquent peers and DAT1 significantly affect self-reported criminal behavior in the presence of theoretically important controls (Hypothesis 1), and whether it is those with two 10R DAT1 alleles versus those with none that are the most effected by exposure to delinquent peers (Hypothesis 2). I utilize NB regression based on the fact that the dependent variable is a highly skewed count measure that violates the assumption of normality required for OLS regression (Gardner, Mulvey, and Shaw 1995). I utilize the appropriate weight, cluster, and strata variables in all analyses to account for the complex Add Health survey design. Tests using Variance Inflation Factors (VIFs) showed that multicollinearity was not a problem in any of the equations.

RESULTS

Table 4.1 presents the descriptive statistics for the study variables for the full sample. As expected, criminal offending levels are fairly low. The average respondent reported committing less than two of the seventeen acts in the criminal behavior index. The average respondent in the sample was about sixteen years old at the time of Wave II data collection. Among the more important controls, the average respondent reported fairly low levels of substance use and scored reasonably well on the low self-control scale. Table 4.2 further describes the data by presenting means and mean comparisons for the three genetic subgroups (0 10R alleles, 1 10R allele, and 2 10R alleles). Importantly, there are no mean differences between these groups in the key

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dependent and independent variables of criminal behavior and affiliations with delinquent peers, respectively. However, several mean differences can be noted, namely low self-control and two racial/ethnic differences. There are significant differences in self-control among the different genetic subgroups, with 10R homozygotes reporting the highest levels of self-control and those who carry zero 10R alleles reporting the lowest levels of self-control. Among the racial/ethnic differences, whites are underrepresented in the 10R/10R subgroup compared to the other two groups, while Asians are overrepresented in the 10R/10R subgroup compared to the other two groups. Prior genetics research has established that the type and frequency of DAT1 alleles that an individual carries is affected by a combination of ancestry and racial/ethnic background (Kang, Palmatier, and Kidd 1999).

Table 4.3 presents the correlation matrix for the study variables. The correlation matrix serves to check for gene-environment correlations (rGE). Gene-environment correlation refers to a nonrandom distribution of environments among different genotypes (Simons et al. 2011). Gene-environment correlations potentially confound GxE effects (Guo, Roettger, and Cai 2008; Guo, Tong, and Cai 2008). Importantly, table 4.3 shows there is not a significant correlation between affiliating with delinquent peers and DAT1 genotype (0, 1, or 2 10R alleles). This suggests an absence of rGE effects in this analysis.[5] As expected, affiliation with delinquent peers is significantly and positively related to criminal behavior at Wave II. Also of note, DAT1 genotype is not correlated with criminal behavior.

Table 4.4 presents NB regression models with criminal behavior at Wave II regressed on affiliation with delinquent peers, DAT1 genotype, and controls. Model 1 examines the effects of affiliations with delinquent peers and DAT1 genotype on criminal behavior, while controlling for

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[5] In addition, independent samples t-tests confirmed no mean-differences in affiliations with delinquent peers between the different categories of the DAT1 variable.

various variables that correlate with offending. As predicted by social learning theory and in line with past studies, affiliation with delinquent peers has a highly significant, positive effect on later criminal behavior. Similar to much past research on genes and antisocial behaviors, DAT1 genotype does not exert a direct effect on criminal behavior. Compared to the reference group of 0 10R DAT1 alleles, neither 1 nor 2 10R DAT1 alleles have a direct effect on offending. Amongst the controls, Native Americans report more offending than the reference group (non-Hispanic white), and older respondents report less offending.

Model 2 in table 4.4 introduces the GxE interaction terms while also testing whether there is a contrast in the effect of affiliations with delinquent peers on later criminal behavior between those carrying 0 or 1 and 0 or 2 10R alleles of DAT1. To test this possibility, I created interactions between the genetic dummy variables and affiliations with delinquent peers. The reference category in these models is the interaction between affiliations and zero 10R DAT1 alleles. First, this model again shows that neither carrying one or two 10R DAT1 alleles results in a direct genetic effect on criminal behavior in comparison to the reference group. Turning to the interaction terms, and in support of hypothesis 2, carrying two 10R DAT1 alleles interacts with affiliations with delinquent peers to significantly and positively shape later criminal behavior. This is in contrast to the interaction term with one 10R allele, which is not significant. That the interaction with two 10R DAT1 alleles is significant means that the effect of affiliations with delinquent peers on offending is significantly greater for carriers of two 10R DAT1 alleles than the effect for the omitted reference category, zero 10R DAT1 alleles. The coefficient for affiliations with delinquent peers is the effect for the omitted reference category, zero 10R alleles, which remains positive and highly significant. So while the effect of affiliations with delinquent peers is significant and positive for all genetic subgroups in model 2, the effect is

60% greater for carriers of two 10R alleles compared to carriers of zero 10R alleles. These results compare well to past research showing that carrying more 10R alleles means individuals are more vulnerable to negative environmental influences, such as affiliating with substance-using peers.

Models 3-5 in table 4.4 introduce the theoretically important controls of low self-control and substance use in a stepwise fashion as a check for spuriousness. First, in model 3, low self-control is introduced. As is expected, low self-control has a positive and highly significant effect on criminal behavior. Looking at the interaction terms, the same ones are significant that were in model 2, although the affiliations with delinquent peers coefficient, the effect for those with zero 10R DAT1 alleles, has been reduced in both size and significance.

In model 4, low self-control is removed and W1 substance use measures are introduced. The introduction of the substance use measures changes the results from previous models dramatically. First, smoking and alcohol use have highly significant and positive effects on criminal behavior. This is not the case for marijuana use, which has a statistically insignificant effect on offending. Among the interaction terms, the GxE coefficient for two 10R alleles remains significant and positive. The big change is among the lower order terms, where the affiliation with delinquent peers coefficient is no longer significant. This means that in this model the effect of affiliations with delinquent peers on criminal behavior is not statistically significant for those who carry zero 10R DAT1 alleles. Thus, the delinquent peers-offending relationship appears to have been rendered spurious by controlling for the respondent's use of substances in this model. By contrast, the effect for individuals with two 10R DAT1 alleles remains positive and significantly greater than the effect for the reference group. Thus, the GxE effect of delinquent peers-by-DAT1 has remained in this model despite controlling for individual

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substance use, supporting hypothesis 1. Model 5 controls for both low self-control and substance use, and the results mirror that of model 4. Of note in this final model, several racial differences in offending have appeared, as Hispanics, blacks, and Native Americans have significantly higher levels of offending than non-Hispanic whites when controls are in place for both low self-control and substance use.

Pulling from model 5 in table 4.4, table 4.5 probes the effect of the interaction between affiliations with delinquent peers and DAT1 genotype on criminal behavior while controlling for the baseline controls and low self-control and substance use. In this table, the effect of affiliations with delinquent peers on criminal behavior is broken down by the number of 10R DAT1 alleles an individual carries. Of note, the group among whom the effect of affiliations is greatest is those who carry two 10R DAT1 alleles. Most interesting, however, is that it is only among these individuals that there is a statistically significant effect of affiliations on criminal behavior. So, at least in the current sample, the classic crime generating effect of affiliating with delinquent peers only holds among those males carrying two 10R DAT1 alleles when controls are in place for low self-control and substance use.

<div align="center">DISCUSSION</div>

In this paper I sought to test whether genetic factors, specifically DAT1 genotype, moderate the widely observed delinquent peers-offending relationship during adolescence. To answer this question I used data from the Add Health study (Udry 2003) that measured adolescents' peer substance use, criminal offending, DAT1 genotype, and numerous other theoretically important variables. These data serve to examine whether DAT1 genotype moderates the effect of delinquent peer association on criminal offending net of controls for respondent's self-control and substance use. Importantly for establishing a GxE rather than an

rGE, the correlation matrix shows that DAT1 genotype does not significantly correlate with affiliating with delinquent peers. The results of the Negative Binomial regression models reveal a significant GxE, where an affiliation with delinquent peers interacts with the 10R allele of DAT1 to influence offending. Specifically, and in support of hypothesis 2, the greatest contrast is between those who possess 0 and 2 10R DAT1 alleles, in line with past research that has shown that not only does the mere presence or non-presence of the 10R allele matter for antisocial phenotypes, but also the number of 10R alleles one carries. Finally, controlling for low self-control and substance use reveals a spurious relationship between affiliations and offending among those who carry zero 10R DAT1 alleles and shows that, at least in this sample, the only group that displays a statistically significant effect of affiliation with delinquent peers on criminal behavior is those males who carry two 10R DAT1 alleles. This finding supports hypothesis 1. Before turning to the implications of this study, the limitations within should be noted.

The current study draws attention to the linkages between genotype and the social environment. Only data sets that are genetically informative, such as the one utilized in the current analysis, can help to provide information about how genetics and the environment interrelate. This is not to suggest that the current study is free of limitations, however, as I would note two key shortcomings of these analyses. First, the measure of delinquent peers available in Add Health (Udry 2003) taps only the substance-using habits of a respondent's friends. A fuller and more appropriate construct would also include measures of more serious delinquent acts, such as violent and property offending, committed by a respondent's friends. Future research would benefit from utilizing a data set with more measures of antisocial peers that index more diverse and serious forms of offending. The second key limitation is that while this study is

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based on a nationally representative data set, only males are included in these models, meaning that these results are not generalizable to all adolescents.[6] This study is notable, however, because the sample size (N = 3,557) is much larger than has typically been available in GxE research.

These results lead to an important question to be answered in future research, namely what, if any, are the mechanisms by which the interaction of affiliations with delinquent peers and genotype affect criminal offending? Social learning theory would suggest that one mechanism that connects peers and offending would be the endorsement of delinquent attitudes that is often the result of delinquent peer association. In the context of the current study this seems like a distinct possibility, given ongoing research in the GxE literature. A good example of this possibility comes from a recent study by Simons et al. (2011), who in a sample of African Americans showed that genotype moderated the effect of a composite measure of positive and negative environment on aggression. Much of this effect was mediated by a composite measure of "hostile orientation" that included measures like one's belief in the importance of appearing tough and their view of others as untrustworthy and potentially threatening. So it could be that DAT1 genotype enhances the criminogenic effects of affiliating with delinquent peers in part because carriers of the 10R allele more readily accept and endorse the attitudes and justifications for deviant behavior that they receive from their delinquent peers. According to social learning theory, this would then in turn result in more criminal offending on the part of that individual.

Theoretically, this study is important in showing the utility in combining traditional criminological theories with a biosocial modeling approach. This kind of theory building and modeling strategy is important because it helps further develop ideas centered on the concept that

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[6] Separate models not presented were run for females, with these models not showing any significant GxE effects. Results available upon request.

the environment and biology are always interacting to shape how we experience and react to our world. Moving forward, criminology as a discipline should focus on and consider central this kind of theory construction.

CHAPTER 5

CONCLUSION

Criminology has traditionally been dominated by social explanations of crime and delinquency (Gottfredson and Hirschi 1990; Walsh 2002; Walsh and Beaver 2009). Theories of crime that are purely sociologically informed, for example, highlight the importance of parents, peers, schools, neighborhoods, and other social institutions in the etiology of crime and delinquency (Anderson 1999; Gottfredson and Hirschi 1990; Sampson and Groves 1989; Warr 2002). Until very recently, criminology has shied away from explanations of antisocial behavior that are influenced by the literature on genetics and phenotypes (Walsh and Beaver 2009). As a result, a limited number of empirical studies have examined the contributions of genetics to antisocial phenotypes, and even fewer have done this while integrating genetics research with existing criminological theory (for notable exceptions see Beaver et al. 2011; Simons et al. 2011). The current dissertation took a step in the direction of integrating further gene-environment interaction (GxE) research and criminological theory by specifically testing three highly popular theories of crime and delinquency (self-control theory, general strain theory, social learning theory) in a biosocial framework where environmental processes are conditioned by genotype. Analysis of the Add Health data revealed that genotype moderated the effects of three different environmental stimuli on criminal behavior.

This conclusion is meant to provide a summary of the major findings garnered from chapters 2-4 in this dissertation. To that end, this conclusion is divided into three distinct parts. First, I will begin by summarizing the key findings from each of the three empirical chapters.

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Next, I will discuss the important limitations of this dissertation and directions for future

research. Finally, I will finish by discussing the implications of these findings for criminology.

SUMMARY OF FINDINGS

The empirical element of this dissertation consisted of three separate analyses. Each of

these analyses involved arguing for the integration of a classic theory of crime into a broader

biosocial framework, and each of these theories was subsequently tested. First, I tested a model

provided by self-control theory whereby parenting effects both self-control and criminal

behavior, with self-control mediating the parenting-crime relationship. Coming from the GxE

literature I sought to show how the effects of parenting are moderated by both MAOA and DAT1

genotype. Next, I tested a model coming from GST wherein strain effects both depression and

criminal behavior, with depression mediating the strain-crime relationship. Expanding on

research in the GxE literature I sought to show how the effects of strain are moderated by 5-

HTTLPR genotype. Lastly, I tested a model utilizing a key variable from social learning theory,

affiliations with delinquent peers, wherein affiliations with delinquent peers affect one's own

criminal behavior, while additionally testing whether the delinquent peers effect is moderated by

DAT1 genotype. I will discuss the major findings from each of these analyses in turn, beginning

with the self-control chapter, chapter 2.

Genes, Parenting, Self-Control, and Criminal Behavior

Chapter 2 was concerned with whether genotype conditions the relationship between

parenting, self-control, and criminal behavior that is identified by self-control theory

(Gottfredson and Hirschi 1990). Specifically, I tested whether the 2R and 3R alleles of MAOA

and the 10R allele of DAT1 moderated, both separately and together, the effect of parenting on

levels of self-control and self-reported criminal behavior. Furthermore, in line with self-control

theory, I tested whether self-control mediated the parenting by genotype effect on criminal behavior. Results confirmed the utility and need to account for genetics when considering environmental effects such as parenting and how they shape offending, as well as the utility of integrating a GxE modeling approach into self-control theory.

Ordinary least squares (OLS) regression models showed that there was a significant effect of the interaction of poor parent-child relationship with MAOA and DAT1 genotype on levels of self-control. Specifically, as the parent-child relationship becomes worse, levels of self-control decrease, as expected by self-control theory. What my models additionally show is that this negative effect of poor parent-child relationship on levels of self-control is enhanced among those males in the sample who carry the so-called "plasticity" alleles of the MAOA and DAT1 genes. Additionally, and in support of expectations from the cumulative plasticity hypothesis, it is those who carry plasticity alleles for both MAOA and DAT1 compared to those with no plasticity alleles who are most vulnerable to the effects of a poor parent-child relationship. The slopes for the effect of poor parent-child relationship on levels of self-control do not significantly differ between those with either just the plasticity allele for MAOA or DAT1 and those with no plasticity alleles. One finding in these models that was not expected was that genotype, specifically the 10R allele of DAT1, had a direct, significant effect on levels of self-control, with those who possess 2 10R alleles having higher levels of self-control than the reference category of those who do not carry plasticity alleles for either MAOA or DAT1.

OLS regression models where self-reported criminal behavior was the outcome of interest largely replicated results from models looking at low self-control. There was a significant GxE, and the significant contrast was between those with 2 and 0 plasticity alleles. Of particular interest in these models was the finding that the effect of poor parent-child

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relationship on criminal behavior was significant only among those individuals in the sample who carry plasticity alleles for both MAOA and DAT1. To test the full mediating model specified by self-control theory where parenting shapes levels of self-control which then shapes criminal behavior I additionally ran models where I introduced the self-control scale as a potential mediator of the parenting-by-genotype effect on crime. Results showed that the scale of self-control fully mediates the parenting-by-genotype effect on crime, reducing the coefficient among the two plasticity allele group to insignificance. These results support self-control theory and the utility of integrating a GxE modeling approach into this theory of crime and deviance. Next, I will discuss the results of chapter 3.

Genes, Strain, Depression, and Criminal Behavior

Chapter 3 was concerned with whether genotype conditions the relationship between strain, negative emotions, and criminal behavior that is identified by general strain theory (Agnew 1992). In this chapter I focused on the s-allele of 5-HTTLPR, as I contrasted individuals with two s-alleles to those with some other allelic combination (l/l, s/l, l/s) of 5-HTTLPR. In these analyses I operationalized strain as the threatened or actual loss of loved ones by combining two measures that asked respondents whether any of their friends or family members had attempted or completed suicide in the previous year. The key GST variable negative affect was represented by a scale of depressive symptoms reported for a period covering the week before being interviewed at Wave I. I tested whether 5-HTTLPR genotype moderated the effect of strain on both depression and criminal behavior, and I further tested the full strain-negative emotions-criminal behavior model specified by GST. Results provide positive support for both the validity of GST, as well as the importance of 5-HTTLPR genotype in explaining why strain affects both negative affect and crime.

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Ordinary least squares (OLS) regression models showed that there was a significant effect of the interaction of friends and family suicidal behavior with 5-HTTLPR genotype on levels of depressive symptoms. Specifically, those males in the sample who were homozygous for the s-allele experienced more depressive symptoms following the attempted or completed suicide of friends and family members than males with any other allelic combination for 5-HTTLPR (l/l, s/l, l/s). Negative binomial (NB) regression models where a count measure of criminal behavior was the outcome of interest produced similar results. The males in the sample carrying two s-alleles of 5-HTTLPR reported more criminal behavior when exposed to the serious strain of friends and family attempting or completing suicide than did males with other variations of 5-HTTLPR. The full model specified by GST was supported in that the introduction of the depressive symptoms scale as a predictor of criminal behavior completely mediated the strain-by-genotype effect on crime. These results tie together nicely the large amounts of research in the GxE literature that have shown that 5-HTTLPR genotype moderates the effects of stressful life events on experiences of depression with the fairly recent literature examining how the environment-offending relationship is moderated by 5-HTTLPR genotype (Caspi et al. 2003; Simons et al. 2011). I will now to a discussion of the results of the final empirical chapter of this dissertation, chapter 4.

Genes, Delinquent Peers, and Criminal Behavior

In chapter 4 I sought to examine how genotype moderates the widely cited relationship between affiliating with delinquent peers and one's own offending behavior. The dopamine transporter gene (DAT1) was the focus of chapter 4, and I contrasted individuals based on the number of 10R alleles of the DAT1 gene that they carried (0-2). In this analysis I operationalized affiliations with delinquent peers by utilizing items that measured a respondent's

three best friends' use of three controlled substances that correlate with criminal behavior in adolescence: tobacco, alcohol, and marijuana. I tested whether DAT1 genotype moderated the effect of affiliating with substance using peers on criminal behavior, and I further tested whether the number of 10R alleles someone carried (0 versus 1 and 0 versus 2) made a difference or if it was merely the presence of the 10R allele or not that mattered for the peers-offending relationship.

Negative binomial (NB) regression models showed that there was a significant effect of the interaction of affiliating with substance using peers with DAT1 genotype on levels of criminal behavior. This GxE effect held despite controls for self-control and the respondent's use of substances. In addition, the final model showed that the number of 10R alleles someone carries is an important factor affecting the delinquent peers-offending relationship. In contrasting those who carried either 1 or 2 10R alleles to those who carry 0, the 0 versus 2 contrast was significant, while the 0 versus 1 contrast was not. A particularly interesting finding in this final model was that in this sample only those who carry two 10R alleles for DAT1 display a statistically significant effect of affiliating with delinquent peers on their own delinquency. These findings supports the notion, argued in other studies in the GxE literature, that the number of 10R alleles one carries is what matters, not merely the presence or non-presence of the 10R allele.

The regression models in chapter 4 expand on previous findings in the GxE literature concerning DAT1, delinquent peers, and offending. Prior research had shown that entry into delinquent peer groups is conditioned by DAT1 genotype (Beaver et al. 2008; Yun et al. 2011), and numerous studies have tied the 10R allele to criminal offending (Guo et al. 2007), but none so far had examined whether DAT1 genotype moderates the peers-offending relationship. These

analyses thus serve to expand our understanding of how DAT1 genotype conditions behavior, and future studies should seek to examine the mechanisms whereby the delinquent peers-by-genotype interaction shapes criminal behavior. Next, I will touch on the theoretical implications of this dissertation, as well the implications for criminology as a discipline and the potential public policy implications of this and other GxE research.

<div align="center">IMPLICATIONS FOR CRIMINOLOGY AND PUBLIC POLICY</div>

The theories tested in this dissertation prove to be a good fit with the biosocial literature. Moving forward, these theories should be expanded even further. In the case of self-control theory, several possible directions can be noted. First, other candidate genes should be explored that could condition the parenting-self-control-crime relationship. Potential candidate genes that are already widely cited in the GxE literature include 5-HTTLPR, DRD2, and DRD4. This implication also goes for GST, with the substitution of MAOA and DAT1 for 5-HTTLPR. Secondly, independent variables other than parenting should be examined. While parenting is the variable most commonly singled out in the self-control literature, other variables could be important for shaping self-control and crime in a manner similar to the parent-child relationship. It should additionally be tested whether these other variables interact with genetics to shape self-control and crime. Lastly, another important neuropsychological deficit in adolescence that bears a striking resemblance to low self-control, ADHD, should be examined in a manner similar to how low self-control was examined in this dissertation. While genetics have been noted as important in the etiology of ADHD and ADHD is important in the etiology of crime and deviance, the nature of what this relationship actually looks like theoretically is still murky.

In the case of GST, it would be key to first explore additional strains that may be moderated by 5-HTTLPR in their effects on negative emotions and deviant behavior. In

addition, negative emotions other than depression should be the focus of further research. In particular, a focus on how genes involved with the processing of dopamine, like MAOA and DAT1, would match up well with a focus on the most commonly cited negative emotion in the GST literature that leads to offending, anger.

Finally, an important next step in further identifying the nature of the delinquent peers by DAT1-delinquency relationship is to identify what mechanism(s) make DAT1 matter in the delinquent peers-delinquency relationship. A potentially important factor is the acceptance of antisocial attitudes and justifications for delinquency that affiliating with delinquent peers is expected to foster. With DAT1 being implemented in learning, reward-seeking behavior, and pleasure-seeking behavior, perhaps some people are biologically geared towards being more open to antisocial learning than others. Thus, it would be important to gauge the effect of the delinquent peers by DAT1 relationship on the endorsement of antisocial attitudes and justifications for antisocial behavior.

The results of this dissertation suggest that several traditional theories of crime are a good fit for integration with the GxE literature on antisocial behaviors. So what then are the implications for criminology as a discipline moving forward? I offer several implications of this work for criminology. First, criminologists and sociologists concerned with crime should begin to lead the charge in developing biosocial explanations of deviant and antisocial behaviors. Too much of the work on genetics and deviance is being done by individuals outside of the fields of criminology and sociology, and too often these researchers fail to consider time-tested theories of crime and important controls that should be included in their models. If a biosocial approach to criminology becomes widespread then we may be able to begin finally explaining some of the old "facts" in criminology for which a complete, sociological explanation has eluded researchers,

such as the age-crime curve, racial and gender gaps in offending, and trajectories of criminal behavior (Walsh 2002). Sociology and criminology cannot continue to largely discount biosocial perspectives concerning antisocial behaviors without risking becoming out-of-touch and dated when compared to other academic disciplines.

On a related note, the disciplines of sociology and criminology as a whole need to shed both their ideological allegiance to sociological explanations of crime, and their aversion to genetic explanations of crime. This is not to say sociological explanations of crime need to be abandoned, but that an interdisciplinary approach needs to be the norm moving forward. This interdisciplinary approach, wherein criminologists and sociologists are communicating their theories and results with researchers from a broad array of academic disciplines, will make for great strides in full explicating the pathways that lead to criminal and delinquent behaviors, as well as the pathways that lead to desistance from these behaviors.

Lastly, if the nature versus nurture debate has not yet been put to rest, then it needs to be put to rest in a public fashion. While many other fields of scientific inquiry have come to the conclusion that it is actually nature by way of nurture, many sociologists and criminologists still vehemently deny the genetic basis of behavior. This is going on while research is showing that most of the variables that are the central concern of criminologists and sociologists have at least some of their etiology in genetics, and more are being discovered every day (Beaver et al. 2006; Cleveland, Wiebe, and Rowe 2005; Walsh 2002). To argue that our most sacred variables are purely sociological goes against vast amounts of scientific evidence coming from other disciplines. Therefore, a biosocial perspective must become the norm to explain how genes and the environment interlock and work together to shape offending patterns.

An area where the implications of this dissertation and other works in the GxE literature still needs to be sorted out are the public policy implications of biosocial models of criminal behavior. The one thing I would say we definitely do not want to do is focus on the biological implications of this and other works. We should not, for example, go around genotyping kids and identifying them as "at risk" because they carry the 2R allele of MAOA or the 10R allele of DAT1. First, these genetic subtypes are actually not that uncommon, so we would be focusing on a large chunk of the population and labeling them as at risk due to their genotype. Second, if in doing this kind of targeting we find that certain subgroups within the population, like certain racial groups, disproportionately carry these genotypes, then it would too easily invite the kind of racism and discrimination that made earlier research and theorizing on the biological nature of offending so disastrous.

I think these results and the results of other GxE research actually point to much simpler policy solutions for crime and deviance. Much like with the traditional sociological and criminological literature, these kinds of studies point out the importance of the social environment for children's development. The only reason genetics matter in these studies is because of the environment. Research in the GxE literature, including this dissertation, consistently demonstrate that adverse social environments of various types increase offending behaviors among everyone, it's just that the effects of the environment are exacerbated among some subgroups in the population. So, simply speaking, the policy implications of research in the GxE literature are the same as the policy implications in the sociological and criminological literatures: a strong, positive parent-child relationship is important, children should be shielded from exposure to violence and stress, education should be emphasized and made rewarding, etc. There is no need to specifically invoke genetics or some other aspect of biology because these

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things are only a factor in so far as the social environment makes them a factor. Next, I will discuss the key limitations of these analyses, as well as the future directions for research that are suggested by this dissertation.

LIMITATIONS AND DIRECTIONS FOR FUTURE RESEARCH

This dissertation provided evidence to the utility of integrating a GxE approach into three highly popular theories of crime; self-control theory, general strain theory, and social learning theory. It has thus served to provide a strong theoretical explanation, mixing theories from criminology and biosocial fields, for why genetics are consequential for neuropsychological deficits like low self-control and depression, as well as criminal and other antisocial behaviors. Before discussing the future directions suggested by this dissertation, however, it is important to mention the limitations of this work. Most of these limitations relate back to the data set utilized in all the analyses, the Add Health study. While the Add Health data set is a rich and full data set and has the added advantage of having a nationally representative sample of adolescents, there are a few drawbacks to mention.

First, the respondents in the Add Health data set are all adolescents and young adults. This truncated age range makes for a couple of key limitations. For one, two key independent variables in these analyses; the parent-child relationship in the self-control analyses and delinquent peers in the social learning analyses, would be better measured at an earlier time. Gottfredson and Hirschi have argued that parenting effects on levels of self-control show up fairly early (1990), while the effect of delinquent peers would also be expected to show up much earlier in adolescence than the timeframe that the Add Health data covers (Akers and Sellers 2009). Both of these issues make time ordering in the models at least somewhat suspect, as one could easily imagine a scenario where a child's poor self-regulation results in a parent

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withdrawing and becoming emotionally distant, or that a child's early delinquent behavior may lead them to seek out delinquent peers with which to associate.

Another limitation of the Add Health data set is that important items traditionally used to measure many key criminological concepts and key criminological variables are either inadequate or absent. In the case of the parenting variable, the Add Health data set lacks commonly used items that measure parental monitoring and disciplinary practices. This results in a measure of parenting being utilized in the self-control analyses that is not entirely adequate for testing propositions coming from the theory in question. Also in the same analysis, the scale of low self-control is not one that has been commonly used, but has been cobbled together by past researchers seeking to make a passable scale of self-control in the Add Health data set. It lacks some key elements inherent in Gottfredson and Hirschi's conception of self-control, namely reported preferences for risky behavior. Missing from the GST analyses is anger, which is the negative emotion that is most commonly focused on in GST research (Akers and Sellers 2009). This absence is due to there being no measures of state-based anger in the Add Health data set, as well as the poor quality of the only item measuring trait-based anger in Add health. This trait-based measure consists of one question that is only found in Wave I. The target respondent's parent was asked whether the target respondent had a "bad temper" and the only answer options were yes and no. Lastly, the measure of delinquent peers is not wholly desirable, because it only asks respondents about their peer's use of controlled substances. A more desirable measure would have also asked about peer delinquency besides the use of substances, and would have focused on acts like fighting, stealing, etc.

A final limitation is that only three genetic polymorphisms are examined in these analyses. These genes only represent a fraction of the possible genes that could condition

traditional models of crime and deviance. More research is needed into the genes that have thus far been undiscovered or understudied that may influence both neuropsychological functioning and antisocial behaviors. Once they have been identified data collectors like Add Health must go back and type respondents for these genes to then test their relationship to the environment, neuropsychological functioning, and antisocial behavior.

Even with these limitations in mind, the analyses in this dissertation provide a good start for integrating previously unconnected findings in the GxE literature with those that have examined traditional theories of crime and deviance. Much of the GxE literature has failed to control for extraneous influences on the genes-behavior relationship, while in these analyses numerous variables that past research has shown to correlate with delinquent and criminal behavior are held constant. That GxE effects remain despite important controls means that there may be even more to the influence of the combination of genetic risk and environmental triggers on neuropsychological functioning and antisocial behavior than was previously thought.

Future research can expand on these findings. In the broadest possible way, more time-tested theories of crime could be selected and given a fresh approach utilizing the findings in the GxE literature. Two very good opportunities for this kind of work are present in social disorganization and life course theories. To date, very little research has been done to examine how neighborhood effects on behavior may be moderated by genotype (for one good example, see Beaver et al. 2011). Might concentrated disadvantage and collective efficacy differentially shape people's offending behaviors, much like parenting, strain, etc., depending on a person's genetic makeup? Life course theory represents another good candidate for integrating research on the genetic causes and correlates of behavior. In particular, so-called "turning points" are an important concept in the criminological literature that has recently emerged. Is it possible that

people are more or less receptive to turning points, such as marriage or getting a good job, based on their genetic makeup? Could the effect of turning points on desistance be moderated by genotype? Social disorganization and life course theories are just two out of many that could be made fresh by infusing appropriate findings from the GxE literature. Now that I have discussed the limitations of this work and made some suggestions for future research, I will turn to a discussion of the implications of this work for the field of criminology.

IN CONCLUSION

Until recently, criminologists have largely rejected biosocial explanations of antisocial behavior and stayed true to the discipline's roots in sociological theorizing (Walsh 2002). While some criminologists have drawn on biosocial explanations of crime in their research, this sort of theorizing is still marginalized within the discipline (Walsh and Beaver 2009). Given advancements made in the last decade, however, it is impossible to ignore biosocial theorizing as a major new force in the academic literature on crime and deviance. What this dissertation demonstrated is that even very traditional theories of crime and deviance can be combined with a GxE modeling approach that is not only appropriate, but expands on and enriches traditional theories in the criminological literature. Without furthering these sorts of biosocial expansions of traditional theories of crime and deviance, the discipline has the potential to stagnate and cease development of our understanding of the etiology of antisocial behavior. With all that is going on in other scientific disciplines of late pertaining to the genetic basis of behavior, the time is ripe for a new program of biosocial theorizing in the study of criminal and deviant behaviors to become the predominant theoretical lens by which researchers approach explaining these behaviors (Walsh and Beaver 2009).

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Table 2.1 Descriptive statistics

Variables	Range	Full Sample (n=3,610) Mean (SE)
Dependent Variable		
Criminal Behavior W2	.69-3.53	2.26 (.00)
Independent Variables		
Poor Parent-Child Relationship	-1.38-5.45	.00 (.01)
MAOA & DAT1	0/1	.25 (.01)
MAOA (2R or 3R)	0/1	.16 (.01)
DAT1 (10R/10R)	0/1	.34 (.01)
None	0/1	.25 (.01)
Mediating Variable		
Low Self-Control	25-84	46.72 (.12)
Controls		
White	0/1	.56 (.01)
Hispanic	0/1	.15 (.01)
Black	0/1	.18 (.01)
Native American	0/1	.03 (.00)
Asian	0/1	.08 (.00)
Other	0/1	.01 (.00)
Age W2	11-21	16.11 (.03)
Parent's Education	0/1	.27 (.01)
Parent Receiving Public Assistance	0/1	.07 (.00)

Note. Because these statistics are weighted and adjusted for survey design, standard errors are produced rather than standard deviations.

Table 2.2 Descriptive statistics and mean comparisons by genotype

Variables	Range	MAOA & DAT1 (n=897) Mean (SE)	MAOA (2R or 3R) (n=576) Mean (SE)	DAT1 (10R/10R) (n=1,235) Mean (SE)	None (n=902) Mean (SE)
Dependent Variable					
Criminal Behavior W2	.69-3.53	2.26 (.01)	2.26 (.01)	2.25 (.00)	2.26 (.01)
Independent Variable					
Poor Parent-Child Relationship	-1.38-5.45	-.01 (.03)	.05 (.04)	.02 (.02)	-.05 (.03)
Mediating Variable					
Low Self-Control	25-84	46.44 (.24)	46.93 (.30)	46.51 (.20)	47.16 (.23)
Controls					
White	0/1	.41 (.02)	.55 (.02)	.60 (.01)	.66 (.02)**
Hispanic	0/1	.15 (.01)	.14 (.01)	.15 (.01)	.15 (.01)
Black	0/1	.25 (.01)	.22 (.02)	.14 (.01)	.12 (.01)**
Native American	0/1	.03 (.01)	.02 (.01)	.03 (.00)	.02 (.00)
Asian	0/1	.14 (.01)	.06 (.01)	.07 (.01)	.03 (.01)**
Other	0/1	.01 (.00)	.01 (.00)	.01 (.00)	.01 (.00)
Age W2	11-21	16.04 (.05)	16.06 (.07)	16.19 (.04)	16.12 (.05)
Parent's Education	0/1	.29 (.01)	.25 (.02)	.28 (.01)	.27 (.01)
Parent Receiving Public Assistance	0/1	.06 (.01)	.08 (.01)	.08 (.01)	.06 (.01)

Note. Because these statistics are weighted and adjusted for survey design, standard errors are produced rather than standard deviations.

* p < .05, ** p < .01 mean one-way ANOVAs denote significant genotype comparisons.

Table 2.3 Correlation matrix for the study variables (N = 3,610)

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
1. Criminal Behavior W2	X															
2. Poor Parent-Child Relationship	.07**	X														
3. MAOA & DAT1	.02	-.01	X													
4. MAOA (2R or 3R)	-.01	.03	-.25**	X												
5. DAT1 (10R/10R)	-.02	.02	-.42**	-.31**	X											
6. None	.00	-.03	-.33**	-.25**	-.42**	X										
7. Low Self-Control	.17**	.36**	-.02	.01	-.02	.04*	X									
8. White	-.05**	.05**	-.17**	-.01	.05**	.11**	.07**	X								
9. Hispanic	.04*	-.03	-.01	-.01	.01	.01	-.02	-.47**	X							
10. Black	.02	-.08**	.11**	.05**	-.07**	-.08**	-.07**	-.52**	-.20**	X						
11. Native American	.04*	.00	.01	-.01	.01	-.02	.03	-.18**	-.07**	-.08**	X					
12. Asian	-.02	.06**	.15**	-.03	-.02	-.10**	-.02	-.33**	-.12**	-.13**	-.05**	X				
13. Other	.02	-.01	.01	-.01	-.01	.01	-.01	-.05**	-.02	-.05**	-.02	-.03	X			
14. Age W2	.01	.19**	-.03	-.02	.03*	.00	.03	-.05**	.07**	-.04**	.00	.05**	.01	X		
15. Parent's Education	-.03	.02	.03	-.03	.01	-.01	-.03	.02	-.16**	.03*	-.01	.14**	.01	-.03	X	
16. Parent Receiving Public Assistance	.05**	.01	-.02	.01	.02	-.02	.02	-.13**	.10**	.11**	.02	-.06**	-.03	-.02	-.14**	X
Mean	.84	-.12	.25	.16	.34	.25	46.72	.56	.15	.18	.03	.08	.01	16.11	.27	.07
Standard Error	.12	.01	.01	.01	.01	.01	.12	.01	.01	.01	.00	.00	.00	.03	.01	.00

* p < .05, ** p < .01

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Table 2.4 Low self-control regressed on poor parent-child relationship,
genotype, and controls

Variables	Model 1 Coef. (SE)	Model 2 Coef. (SE)
Environment and Genetic Variables		
Poor Parent-Child Relationship	3.36 (.24)**	2.89 (.33)**
MAOA & DAT1	-.83 (.41)*	-.72 (.42)
MAOA (2R or 3R)	-.49 (.58)	-.42 (.58)
DAT1 (10R/10R)	-.89 (.40)*	-.86 (.39)*
Dummy Variable Interactions		
PPCR x MAOA & DAT1		1.69 (.83)*
PPCR x MAOA (2R or 3R)		.23 (.50)
PPCR x DAT1 (10R/10R)		.03 (.48)
Controls		
Hispanic	-.84 (.53)	-.84 (.52)
Black	-.87 (.40)*	-.90 (.39)*
Native American	1.93 (1.00)	1.82 (.99)
Asian	-.99 (.62)	-1.01 (.60)
Other	-.85 (.90)	-.92 (.86)
Age W1	-.15 (.11)	-.14 (.11)
Parent's Education	-.38 (.33)	-.38 (.32)
Parent Receiving Public Assistance	1.08 (.53)*	1.17 (.53)*
Constant	50.16 (1.79)**	49.95 (1.7)**
R-Sq.	.14	.15

Notes. Zero plasticity alleles is the refence category for all genetic variables and
GxE terms. Non-Hispanic White is the reference category for all race/ethnic groups.
This table includes unstandardized coefficients (linearized standard errors) from
OLS models.
*p < .05, **p < .01

Table 2.5: Effect of poor parent-child relationship on low self-control by number and type of risk alleles

Risk Alleles	Coeff.
None	2.89**
MAOA only	3.12**
DAT1 only	2.92**
MAOA & DAT1	4.58**

**p < .01

Table 2.6 Criminal behavior W2 regressed on poor parent-child relationship, genotype, low self-control, and controls

Variables	Model 1 Coef. (SE)	Model 2 Coef. (SE)	Model 3 Coef. (SE)
Environment and Genetic Variables			
Poor Parent-Child Relationship	.02 (.01)**	.00 (.01)	-.01 (.01)
MAOA & DAT1	.00 (.01)	.01 (.01)	.01 (.01)
MAOA (2R or 3R)	-.01 (.01)	-.01 (.01)	-.01 (.01)
DAT1 (10R/10R)	-.01 (.01)	-.01 (.01)	.00 (.01)
Dummy Variable Interactions			
PPCR x MAOA & DAT1		.04 (.02)*	.03 (.02)
PPCR x MAOA (2R or 3R)		.01 (.01)	.01 (.01)
PPCR x DAT1 (10R/10R)		.02 (.01)	.00 (.01)
Mediating Variable			
Low self-control			.01 (.00)**
Controls			
Hispanic	.01 (.02)	.01 (.02)	.01 (.01)
Black	.02 (.01)	.02 (.01)	.02 (.01)*
Native American	.05 (.02)*	.05 (.02)*	.04 (.02)
Asian	-.02 (.01)	-.02 (.01)	-.02 (.01)
Other	.02 (.03)	.02 (.03)	.02 (.02)
Age W2	.00 (.00)	.00 (.00)	.00 (.00)
Parent's Education	-.01 (.01)	-.01 (.01)	-.01 (.01)
Parent Receiving Public Assistance	.01 (.01)	.02 (.01)	.01 (.01)
Constant	2.28 (.04)**	2.27 (.04)**	2.07 (.05)**
R-Sq.	.02	.02	.05

Notes. Zero plasticity alleles is the refence category for all genetic variables and GxE terms.
Non-Hispanic White is the reference category for all race/ethnic groups. This table includes
unstandardized coefficients (linearized standard errors) from OLS models.
*p < .05, **p < .01

Table 2.7: Effect of poor parent-child relationship on criminal behavior by number and type of risk alleles

Risk Alleles	Coeff.
None	.00
MAOA only	.02
DAT1 only	.02
MAOA & DAT1	.04*

*p < .05

Table 3.1 Descriptive statistics

	Range	Full Sample (n=3,581)
Variables		Mean (SE)
Dependent Variable		
Criminal Behavior W2	0-9	.64 (.02)
Independent Variables		
Friends and Family Suicidal Behavior	0/1	.14 (.01)
5-HTTLPR Genotype	0/1	.28 (.01)
Mediating Variable		
Depressive Symptoms	0-51	9.94 (.11)
Controls		
White	0/1	.56 (.01)
Hispanic	0/1	.15 (.01)
Black	0/1	.18 (.01)
Native American	0/1	.03 (.00)
Asian	0/1	.08 (.00)
Other	0/1	.01 (.00)
Age W2	11-21	16.11 (.03)
Parent's Education	0/1	.27 (.01)
Parent Receiving Public Assistance	0/1	.07 (.00)

Note. Because these statistics are weighted and adjusted for survey design, standard errors are produced rather than standard deviations.

Table 3.2 Descriptive statistics and mean comparisons by genotype

Variables	Range	l/l, s/l, l/s (n=2,569) Mean (SE)	s/s (n=1,012) Mean (SE)
Dependent Variable			
Criminal Behavior W2	0-9	.64 (.02)	.63 (.04)
Independent Variable			
Friends and Family Suicidal Behavior	0/1	.14 (.01)	.15 (.01)
Mediating Variable			
Depressive Symptoms	0-51	9.79 (.12)	10.27 (.21)*
Controls			
White	0/1	.58 (.01)	.50 (.02)**
Hispanic	0/1	.14 (.01)	.17 (.01)*
Black	0/1	.20 (.01)	.13 (.01)**
Native American	0/1	.03 (.00)	.02 (.00)
Asian	0/1	.04 (.00)	.16 (.01)**
Other	0/1	.01 (.00)	.02 (.00)*
Age W2	11-21	16.09 (.03)	16.17 (.05)
Parent's Education	0/1	.28 (.01)	.27 (.01)
Parent Receiving Public Assistance	0/1	.07 (.00)	.07 (.01)

Note. Because these statistics are weighted and adjusted for survey design, standard errors are produced rather than standard deviations.

* $p < .05$, ** $p < .01$ mean one-way ANOVAs denote significant genotype comparisons.

Table 3.3 Correlation matrix for the study variables (N = 3,581)

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
1. Criminal Behavior W2	X												
2. Friends and Family Suicidal Behavio	.14**	X											
3. 5-HTTLPR Genotype (1 = s/s)	.00	.00	X										
4. Depressive Symptoms	.15**	.15**	.03	X									
5. White	-.03	.03	-.08**	-.13**	X								
6. Hispanic	.04*	.02	.04*	.06**	-.47**	X							
7. Black	-.01	-.04	-.08**	.03	-.52**	-.20**	X						
8. Native American	.05**	.03	-.02	.02	-.18**	-.07**	-.08**	X					
9. Asian	-.03	-.03	.21**	.10**	-.33**	-.12**	-.13**	-.05**	X				
10. Other	.02	.01	.03	.04*	-.12**	-.05**	-.05**	-.02	-.03	X			
11. Age W2	-.02	.04*	.02	.15**	-.05**	.07**	-.04**	.00	.05**	.01	X		
12. Parent's Education	-.02	.03	-.01	-.07**	.02	-.16**	.03*	-.01	.14**	.01	-.03	X	
13. Parent Receiving Public Assistance	.02	-.03	.01	.04**	-.13**	.10**	.11**	.02	-.06**	-.03	-.02	-.14**	X
Mean	.64	.16	.28	9.94	.56	.15	.18	.03	.08	.01	16.11	.27	.07
Standard Error	.02	.01	.01	.11	.01	.01	.01	.00	.00	.00	.03	.01	.00

* $p < .05$, ** $p < .01$

Table 3.4 Depressive symptoms regressed on friends and family suicidal behavior, 5-HTTLPR genotype, and controls

Variables	Model 1 Coef. (SE)	Model 2 Coef. (SE)
Environment and Genetic Variables		
Friends and Family Suicidal Behavior	2.77 (.50)**	2.22 (.52)**
5-HTTLPR (1 = s/s)	.33 (.29)	.01 (.31)
Two-Way Interaction		
Friends and Family Suicidal Behavior x 5-HTTLPR		1.99 (.99)*
Controls		
Hispanic	1.30 (.39)**	1.33 (.39)**
Black	1.41 (.43)**	1.43 (.43)**
Native American	1.74 (1.08)	1.74 (1.08)
Asian	3.25 (.64)**	3.27 (.64)**
Other	2.50 (1.36)	2.48 (1.33)
Parent's Education	-1.18 (.28)**	-1.17 (.27)**
Parent Receiving Public Assistance	1.06 (.51)*	1.05 (.52)*
Age W1	.57 (.09)**	.57 (.09)**
Constant	.34 (1.41)	.44 (1.42)
R-Sq.	.07	.07

Notes. Non-Hispanic White is the reference category for all race/ethnic groups.
This table includes unstandardized coefficients (linearized standard errors) from OLS models.
$*p < .05, **p < .01$

Table 3.5 Criminal behavior W2 regressed on friends and family suicidal behavior, 5-HTTLPR
genotype, depressive symptoms, and controls

Variables	Model 1 Coef. (SE)	Model 2 Coef. (SE)	Model 3 Coef. (SE)
Environment and Genetic Variables			
Friends and Family Suicidal Behavior	.54 (.11)**	.52 (.14)**	.45 (.15)**
5-HTTLPR (1 = s/s)	.02 (.10)	-.07 (.10)	-.08 (.10)
Two-Way Interaction			
Friends and Family Suicidal Behavior x 5-HTTLPR		.42 (.21)*	.36 (.20)
Mediating Variable			
Depressive Symptoms			.04 (.01)**
Controls			
Hispanic	.16 (.17)	.17 (.17)	.12 (.17)
Black	.15 (.13)	.15 (.13)	.12 (.13)
Native American	.52 (.24)*	.51 (.24)*	.40 (.23)
Asian	-.26 (.21)	-.26 (.22)	-.34 (.22)
Other	.27 (.35)	.27 (.35)	.22 (.35)
Parent's Education	-.23 (.11)*	-.23 (.11)*	-.22 (.11)*
Parent Receiving Public Assistance	.13 (.15)	.13 (.15)	.09 (.15)
Age W2	.00 (.03)	.00 (.03)	-.03 (.03)
Constant	-.70 (.52)	-.66 (.52)	-.58 (.52)

Notes. Non-Hispanic White is the reference category for all race/ethnic groups. This table includes
unstandardized coefficients (linearized standard errors) from negative binomial models.
*p < .05, **p < .01

Table 4.1   Descriptive statistics

	Range	Full Sample (n=3,557)
Variables		Mean (SE)
Dependent Variable		
Criminal Behavior W2	0-17	1.78 (.04)
Independent Variables		
Affiliation with Delinquent Peers	-.93-2.71	.00 (.02)
2 10R Alleles	0/1	.59 (.01)
1 10R Allele	0/1	.35 (.01)
0 10R Alleles	0/1	.06 (.00)
Controls		
White	0/1	.56 (.01)
Hispanic	0/1	.15 (.01)
Black	0/1	.18 (.01)
Native American	0/1	.03 (.00)
Asian	0/1	.08 (.00)
Other	0/1	.01 (.00)
Age W2	11-21	16.11 (.03)
Parent's Education	0/1	.27 (.01)
Parent Receiving Public Assistance	0/1	.07 (.00)
Low Self-Control	25-84	46.72 (.12)
Smoking W1	0/1	.17 (.01)
Alcohol Use W1	0-6	.98 (.02)
Marijuana Use W1	0-99	2.29 (.43)

Note. Because these statistics are weighted and adjusted for survey design, standard errors are produced rather than standard deviations.

Table 4.2 Descriptive statistics and mean comparisons by genotype

Variables	Range	0 10R DAT1 alleles (n=222) Mean (SE)	1 10R DAT1 allele (n=1,235) Mean (SE)	2 10R DAT1 alleles (n=2,100) Mean (SE)
Dependent Variable				
Criminal Behavior W2	0-17	1.82 (.16)	1.78 (.07)	1.77 (.06)
Independent Variable				
Affiliation with Delinquent Peers	-.93-2.71	-.02 (.07)	-.02 (.03)	.01 (.02)
Controls				
White	0/1	.61 (.03)	.62 (.01)	.52 (.01)**
Hispanic	0/1	.14 (.02)	.15 (.01)	.15 (.01)
Black	0/1	.18 (.03)	.16 (.01)	.19 (.01)
Native American	0/1	.03 (.01)	.02 (.00)	.03 (.00)
Asian	0/1	.04 (.01)	.04 (.01)	.10 (.01)**
Other	0/1	.01 (.01)	.01 (.00)	.01 (.00)
Age W2	11-21	16.02 (.11)	16.10 (.04)	16.13 (.03)
Parent's Education	0/1	.22 (.03)	.27 (.01)	.28 (.01)
Parent Receiving Public Assistance	0/1	.07 (.02)	.07 (.01)	.07 (.01)
Low Self-Control	25-84	48.27 (.49)	46.82 (.20)	46.43 (.16)**
Smoking W1	0/1	.18 (.03)	.16 (.01)	.17 (.01)
Alcohol Use W1	0-6	.98 (.09)	.94 (.04)	.99 (.03)
Marijuana Use W1	0-99	3.29 (2.40)	1.24 (.18)	2.43 (.56)

Note. Because these statistics are weighted and adjusted for survey design, standard errors are produced rather than standard deviations.

$* p < .05, ** p < .01$ mean one-way ANOVAs denote significant genotype comparisons.

Table 4.3 Correlation matrix for the study variables (N = 3,557)

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.
1. Criminal Behavior W2	X																	
2. Affiliation with Delinquent Peers	.28**	X																
3. 2 10R Alleles	.00	.01	X															
4. 1 10R Allele	.00	-.01	-.88**	X														
5. 0 10R Alleles	.00	.00	-.31**	-.19**	X													
6. White	-.05**	.06**	-.09**	-.09**	.03	X												
7. Hispanic	.06**	.02	.01	.00	-.01	-.47**	X											
8. Black	-.01	-.07**	.03*	.04*	.00	-.52**	-.20**	X										
9. Native American	.06**	.02	.02	-.03	.00	-.18**	-.07**	-.08**	X									
10. Asian	-.04*	-.05**	.11**	-.09**	-.04*	-.33**	-.12**	-.13**	-.05**	X								
11. Other	.03	.03	.00	.00	.01	-.12**	-.05**	-.05**	-.02	-.03	X							
12. Age W2	-.03	.30**	.01	-.01	-.02	-.05**	.07**	-.04*	.00	.05**	.01	X						
13. Parent's Education	-.03	-.06**	.02	-.01	-.03	.02	-.16**	.03*	-.01	.14**	.01	-.03	X					
14. Parent Receiving Public Assistance	.04*	.04*	.01	-.01	.00	-.13**	.10**	.11**	.02	-.06**	-.03	-.02	-.14**	X				
15. Low Self-Control	.26**	.27**	-.04*	.01	.06**	.07**	-.02	-.07**	.03	-.02	-.01	.03	-.03	.02	X			
16. Smoking W1	.19**	.47**	.01	-.01	.01	.13**	-.04*	-.13**	.03*	-.02	-.02	.16**	-.03	-.01	.25**	X		
17. Alcohol Use W1	.24**	.56**	.02	-.02	.00	.08**	.03	-.10**	.00	-.05**	.01	.27**	-.02	-.02	.23**	.36**	X	
18. Marijuana Use W1	.08**	.12**	.02	-.03	.01	-.01	.03	-.01	.00	-.01	.01	.03	.00	.02	.08**	.10**	.09**	X
Mean	1.78	.00	.59	.35	.06	.56	.15	.18	.03	.08	.01	16.11	.27	.07	46.72	.17	.98	2.29
Standard Error	.04	.02	.01	.01	.00	.01	.01	.01	.00	.00	.00	.03	.01	.00	.12	.01	.02	.43

$* p < .05, ** p < .01$

Table 4.4 Criminal behavior W2 regressed on affiliations with delinquent peers, DAT1 genotype, and controls

Variables	Model 1 Coef. (SE)	Model 2 Coef. (SE)	Model 3 Coef. (SE)	Model 4 Coef. (SE)	Model 5 Coef. (SE)
Environment and Genetic Variables					
Affiliations with Delinquent Peers	.42 (.04)**	.29 (.07)**	.18 (.08)*	.14 (.08)	.08 (.08)
2 10R alleles	.03 (.10)	.03 (.10)	.10 (.10)	.03 (.10)	.10 (.10)
1 10R allele	.09 (.11)	.10 (.10)	.15 (.11)	.09 (.10)	.14 (.11)
Dummy Variable Interactions					
Affiliations x 2 10R alleles		.18 (.09)*	.20 (.09)*	.20 (.08)*	.22 (.09)*
Affiliations x 1 10R allele		.08 (.09)	.10 (.09)	.10 (.09)	.10 (.09)
Controls					
Hispanic	.19 (.11)	.20 (.11)	.24 (.10)*	.19 (.10)*	.22 (.09)*
Black	.09 (.09)	.09 (.09)	.14 (.08)	.15 (.08)	.18 (.08)*
Native American	.52 (.16)**	.53 (.16)**	.44 (.13)**	.55 (.16)**	.46 (.14)**
Asian	-.18 (.16)	-.18 (.16)	-.17 (.15)	-.09 (.16)	-.09 (.16)
Other	.25 (.21)	.24 (.22)	.31 (.23)	.34 (.23)	.37 (.24)
Parent's Education	-.08 (.07)	-.08 (.07)	-.11 (.07)	-.11 (.07)	-.13 (.07)
Parent Receiving Public Assistance	.05 (.11)	.04 (.11)	.01 (.11)	.03 (.11)	.00 (.11)
Age W2	-.13 (.02)**	-.13 (.02)**	-.11 (.02)**	-.14 (.02)**	-.12 (.02)**
Low Self-Control			.04 (.00)**		.04 (.00)**
Smoking W1				.26 (.07)**	.16 (.07)*
Alcohol Use W1				.12 (.03)**	.10 (.03)**
Marijuana Use W1				.00 (.00)	.00 (.00)
Constant	2.46 (.40)**	2.46 (.39)**	.27 (.47)	2.49 (.38)**	.47 (.45)

Notes. Zero 10R alleles is the reference category for all the genetic variables and GxE terms. Non-Hispanic White is the reference category for all race/ethnic groups. This table includes unstandardized coefficients (linearized standard errors) from negative binomial models.
*p < .05, **p < .01

Table 4.5: Effect of affiliations with delinquent peers on criminal behavior by number of 10R DAT1 alleles, controlling for low self-control and substance use

# of 10R DAT1 Alleles	*Coeff.*
0	.08
1	.18
2	.30*

*p < .05

APPENDIX A

Items for Scaled Variables

Criminal Behavior Scale Items:
In the past 12 months, how often did you...
1. paint graffiti or signs on someone else's property or in a public place?
2. deliberately damage property that didn't belong to you?
3. take something from a store without paying for it?
4. drive a car without its owner's permission?
5. steal something worth more than $50?
6. go into a house or building to steal something?
7. use or threaten to use a weapon to get something from someone?
8. use a weapon in a fight?
9. sell marijuana or other drugs?
10. steal something worth less than $50?
11. take part in a fight where a group of your friends was against another group?
12. hurt someone badly enough to need bandages or care from a doctor or nurse?
13. get into a serious physical fight?
14. carry a weapon at school?
15. You pulled a knife or gun on someone.
16. You shot or stabbed someone.
17. Have you been initiated into a named gang?

Poor Parent-Child Relationship:
1. How close do you feel to your mother?
2. How much do you think she cares about you?
3. Most of the time, your mother is warm and loving toward you.
4. You are satisfied with the way your mother and you communicate with each other.
5. Overall, you are satisfied with your relationship with your mother.

Low Self-Control:
1. All things considered, how is your child's life going?
2. You get along well with your child.
3. You can trust your child.
4. Does your child have a bad temper?
5. You never argue with anyone.
6. When you get what you want, it's usually because you worked hard for it.
7. You never criticize other people.
8. You usually go out of your way to avoid having to deal with problems in your life.
9. Difficult problems make you very upset.

10. When making decisions, you usually go with your "gut feeling" without thinking too much about the consequences of each alternative.
11. When you have a problem to solve, one of the first things you do is get as many facts about the problem as possible.
12. When attempting to find a solution to a problem, you usually try to think of as many different ways to approach the problem as possible.
13. When making decisions, you generally use a systematic method for judging and comparing alternatives.
14. After carrying out a solution to a problem, you usually try to analyze what went right and what went wrong.
15. You like yourself just the way you are.
16. You feel like you are doing everything just about right.
17. You feel socially accepted.
18. Do you have trouble getting along with your teachers?
19. Do you have trouble paying attention in school?
20. Do you have trouble keeping your mind focused?
21. Do you have trouble getting your homework done?

Depressive Symptoms:
How often was each of the following things true in the past week?
1. You were bothered by things that usually don't bother you.
2. You didn't feel like eating, your appetite was poor.
3. You felt that you could not shake off the blues, even with help from your family and your friends.
4. You felt that you were just as good as other people.
5. You had trouble keeping your mind on what you were doing.
6. You felt depressed.
7. You felt that you were too tired to do things.
8. You felt hopeful about the future.
9. You thought your life had been a failure.
10. You felt fearful.
11. You were happy
12. You talked less than usual.
13. You felt lonely.
14. People were unfriendly to you.
15. You enjoyed life.
16. You felt sad.
17. You felt that people disliked you.
18. It was hard to get started doing things.
19. You felt life was not worth living.